Queen's Economics Department Working Paper No. 1332

Addiction and Network Influence

Michal Ksawery Popiel
Queen's University

Department of Economics
Queen's University
94 University Avenue
Kingston, Ontario, Canada
K7L 3N6

11-2014

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Michał Ksawery Popiel†
Queen's University

November 9, 2014

Abstract

Social networks are an important component in understanding the decision to consume addictive substances. They capture the role of limited access, peer influence, and social acceptance and tolerance. However, despite the empirical evidence of their role, they have been absent from theoretical models. This paper proposes a mechanism through which agents can influence each other in their decision to consume an addictive good. An agent's decision is sensitive to her state of addiction as well as to the composition of her neighbourhood. The model is consistent with the empirical evidence that peer influence can work in both ways: influencing an individual to use and helping them to quit. The structure of the network has important implications on the outcome of agents' decisions as well as the effectiveness of policies aimed at limiting use of addictive substances through deterrence. I provide a network-based explanation of why usage rates can vary across otherwise similar agents and show how in some situations encouraging network ties can lead to lower use while in others it can have the opposite effect. Furthermore, I explore the effect of networks on diffusion of addiction and, using simulations, find that addiction spreads faster in an environment where there are few strong links than in one with many weak links.

JEL Codes: C70, D01, I18.

Keywords: Addiction, dual-self, networks, random utility.

1 Introduction

The National Institute on Drug Abuse defines addiction as "a chronic, often relapsing brain disease that causes compulsive drug seeking and use, despite harmful consequences to the addicted individual and to those around him or her." Although initial use can be voluntary, once consumed, drugs physically alter the function of the brain, making it easy to slip into a pattern of use despite a strong desire to stop. As a result, addiction continues to be a major public concern as policy makers devise plans to stop its devastating consequences without overlooking its inherent complexities.

Although economics has a long history of participating in the policy debate, the focus of theoretical models has been on explaining the actions of addicts who prefer to quit but continue to

*I thank Marie-Louise Vierø, Jim Bergin, as well as participants at the CEA conference in Vancouver, BC and the Midwest Economic Theory Conference in Lawrence, KS for their comments.

†Department of Economics, Queens University, Dunning Hall, Room 345, 94 University Ave, Kingston, ON K7L3N6, Canada. Phone: (613) 533-6000 x75525. E-mail: popielm@econ.queensu.ca.

use, exhibiting behaviour that is in clear violation of the revealed preference paradigm (see, for example, Becker and Murphy, 1988, Loewenstein et al., 2003, and O'Donoghue and Rabin, 2002).

By modeling the decision to use as the result of not one rational mind but two competing systems in the brain, the most recent models overcome this paradox and generate realistic behaviour. For instance, Bernheim and Rangel (2004) and (2005) draw upon literature from neuroscience, psychology, and clinical studies to clearly describe how the medical profession identifies it. Setting three key attributes as behavioural targets, the authors develop a model of cue-triggered addiction in which agents respond to stimuli, make mistakes, and are able to take actions that limit mistaken usage. They achieve this using a dual-system approach, where the individual normally operates in a rational "cold" mode and can enter an impulsive and uncontrollable "hot" mode when triggered by a stochastic external cue. A clear welfare criterion emerges, namely utility in the cold mode, and is used to evaluate several government policies from taxation and criminalization to improvement of rehabilitation facilities and drug subsidization. Loewenstein and O'Donoghue (2004) take a very similar approach with the exception that both systems are always active and that the influence of the affective system can be controlled by exerting "will power," a depletable but replenishable resource.

One crucial, but imperative, component missing from the models discussed above is the notion of social interaction. For instance, if a person wants to purchase an illicit drug she cannot just go to the local supermarket. Instead, access is most often gained through friends or acquaintances. This mechanism has the potential to be a powerful driving force in initial exposure. Besides access, incorporating networks can capture other important features such as peer pressure. Peer influence can be both positive and negative. Gardner and Steinberg (2005) find that individuals are more likely to engage in risky behaviours when in a group, especially in the case of teenagers and Maxwell (2002) argues that friends can influence an individual to use drugs but also help them to quit. Cooperation can also arise in the form of pooling resources to purchase drugs. This happens either because it is more convenient for one person from a group to meet with a dealer or because money runs low and users are unable to make the purchase individually (Needle et al., 1995). These three notions of interaction - access, influence, and resource sharing - can be important channels in modeling addiction development.

Social networks can also play an important role on the recovery side. For example, Hawkins and Fraser (1987) find that opiate addicts that return to their social network of users after leaving treatment recommence use rapidly, whereas those that alter their networks to include more non-users have a higher rate of success. More recently, Wooditch et al. (2013) explore data on probationers' neighbourhood in Baltimore and find that higher drug availability is associated with higher use, emphasizing the role of network effects in addiction recovery.

This paper bridges the gap between the empirical findings and the theoretical literature by explicitly modeling social interaction in the decision to consume an addictive substance. This novel feature can be interpreted in several ways: social acceptance, peer-influence, prevalence of drugs in a person's neighbourhood, etc. I consider a framework that incorporates network effects in a

dual-self model of addiction and use it to explore the implications on individual behaviour and policy. Depending on their state of addiction, neighbours can either reinforce or alter an individual's likelihood of use. Furthermore, susceptibility to network influence rises with the strength of network links. This model provides a theoretical explanation of why relocation after rehabilitation can improve an individual's probability of success and shows how the effectiveness of a drug-use deterrent policy can be altered by changes in the network. The structure of the network also has important implications for how quickly addiction can spread in a population. As the strength of connections decreases, adding more links actually leads to slower diffusion.

The paper is organized as follows. Section 2 describes the baseline theoretical model in a static setting with two players and Section 3 uses simulations to extend the analysis to a dynamic setting with multiple players. Section 4 concludes.

2 Model

Let the economy be populated by a finite set of N individuals $i \in \{1, 2, \ldots, N\}$ represented by nodes on a graph $\mathscr{G}$. Each individual has degree $d_i < N$, or d_i neighbours, represented by links, $k_{ij} \in \{0, 1\}$, between nodes. Agent i affects agent j's payoff if $k_{ij} = 1$. Assume that links are undirected, i.e. $k_{ij} = k_{ji}$, and that $k_{ii} = 0$. Let n_i represent agent i's neighbourhood, that is $n_i = \{j \in \mathscr{G} | k_{ij} = 1\}$. For example, a network with $N = 3$ and $d_i = 2$ is shown in Figure 1.

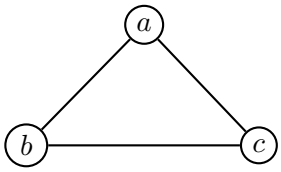


Figure 1: Example with 3 players (a, b, c), each with 2 links

Each period, agents face a choice of whether or not to consume an addictive good $x \in \{0, 1\}$. The good is discrete, so that $x = 1$ indicates the choice to consume and $x = 0$ indicates a decision to abstain. The price of the addictive good is q and it represents the monetary as well as the general resource cost associated with obtaining x. For instance, if the good is inexpensive but difficult to obtain then q will be high. Agents have a fixed amount of resources y. There is an additional good in the economy, c, which is non-addictive and can be thought of as an aggregation of all other goods available to the consumer. Let c be the numeraire.

As in Bernheim and Rangel (2004), the agent's behaviour depends on her state of addiction $s \in \{0, 1, \ldots, S\}$, where S is the maximum state of addiction and 0 is the "virgin" state. If consumption takes place, the agent's state of addiction increases by 1 and if consumption does not take place, the state decreases by 1. However, the agent can never return to $s = 0$ after having consumed at least once.

Agents are myopic and choose whether or not to consume x based on current preferences each period. The decision is determined by two systems as in Loewenstein and O'Donoghue (2004)

and Bernheim and Rangel (2004), as well as the network effect, which is the novel component introduced in this paper. The agent receives utility from consumption, u, which is calculated using the deliberative ("cold") system and simultaneously faces demands from an affective ("hot") system, f. In addition, the payoff is affected by a network effect, h, whose magnitude depends on the actions of the agent's neighbours, n_i, as well as her own choice.

As in Bernheim and Rangel (2004), the decision-making process is subject to mistakes. In their model of cue-triggered addiction, a mistake is characterized by entering the "hot" mode and consuming the addictive good even when the deliberative self prefers to abstain. The probability of making such a mistake is higher in more advanced states of addiction. A problem with this approach is that a shock influences behaviour in only one direction: activating the hot mode and shutting off the cold mode so that a mistake always implies consumption of the addictive good. Random events, however, can influence people in both directions. For instance, a sudden break-up or lay-off can make the use of an addictive substance more appealing. But, equally importantly, witnessing a friend overdose while using drugs can cause an individual to reevaluate her habits and stop use. Loewenstein and O'Donoghue (2004) propose a more general framework in which both systems are active at the same time. They explicitly model the interaction between the two systems as a costly struggle of willpower, but do not introduce a stochastic component. Keeping with the more general framework, in this model, I introduce a choice specific error term that enters the utility directly so that individuals calculate payoffs imperfectly. Both systems are always active and agents can make mistakes in the sense that the (deterministic) utility maximizing action is not always chosen. In this way, the error component can take on a broader interpretation.

The decision to use is a noisy binary choice problem and, as is common in these kinds of models (for an overview see McFadden, 1980), it can be framed using a random utility function, v. For individual i, the random utility function has the following form,

$$v^i(x, c, s, n_i) = \underbrace{u(x_i, c, s)}_{1} - \underbrace{f(x_i, s)}_{2} + \underbrace{h(x, n_i)}_{3} + \underbrace{e_x}_{4}, \tag{1}$$

where each component is described below.

1. $u(x_i, c, s)$ is the direct payoff for consuming the bundle (x_i, c) in state s. This can be seen as the **deliberative system**'s payoff and it is evaluated rationally, fully accounting for the possible negative effects of consuming the addictive good;

2. $f(x_i, s)$ is the mental/physical cost of addiction, e.g. withdrawal. This enters as a cost when the addictive good is not consumed. It can be seen as the willpower required to control the impulses of the **affective system**;

3. $h(x, n_i)$ is a payoff determined by the average behaviour in the agent's neighbourhood n_i, where $x = \{x_i, \{x_j\}_{j \in n_i}\}$. This is the **network effect**; and,

4. e_x is the choice specific **error term** that captures unobservable shocks which can make an action seem more or less attractive.

Although addiction can take on many forms and involve various substances as well as activities, the focus of this paper is on addiction to drugs, which exhibits declining health and increasing dependence with continual usage. Consumption of the addictive good provides a hedonic experience, but it too is declining in the state of addiction due to the build up of tolerance. These effects are evaluated by the deliberative system. At the same time, as use continues, the pain of withdrawal and force of neurological dependence drives the impulse to consume the drug. This urge becomes more difficult to resist and enters the decision-making process via the affective system. These effects are summarized by the following two assumptions.

Assumption 1 (Declining health and hedonic experience)**.** For individual i, $u(x_i, c, s') < u(x_i, c, s)$ for $s' > s$ and $x_i \in \{0, 1\}$.

Assumption 2 (Increasing urge to use)**.** For individual i, $f(0, s') > f(0, s)$ for $s' > s$ and $f(1, s) = 0$.

It is important to note that the cost of the affective system impacts the realized utility only when the urge to use is not satisfied, i.e. when $x_i = 0$. Otherwise, when the agent chooses $x_i = 1$, she avoids the painful withdrawal or willpower expenditure.

The next assumption concerns the novel feature introduced in this paper. As suggested by the empirical findings discussed in Section 1, utility from an action is increasing in the number of neighbours choosing that action. If the number of neighbours choosing each action is equal then the network effect has no impact on the decision.

Assumption 3 (Network effect)**.** For individual i,

1. if $\sum_{j \in n_i} x_j > \sum_{j \in n_i} (1 - x_j)$, then $h\left(\{1, \{x_j\}_{j \in n_i}\}, n_i\right) > h\left(\{0, \{x_j\}_{j \in n_i}\}, n_i\right)$,
2. if $\sum_{j \in n_i} x_j = \sum_{j \in n_i} (1 - x_j)$, then $h\left(\{1, \{x_j\}_{j \in n_i}\}, n_i\right) = h\left(\{0, \{x_j\}_{j \in n_i}\}, n_i\right) = 0$, and
3. if $\sum_{j \in n_i} \tilde{x}_j > \sum_{j \in n_i} x_j$, then $h(\{1, \{\tilde{x}_j\}_{j \in n_i}\}, n_i) > h(\{1, \{x_j\}_{j \in n_i}\}, n_i)$ and $h(\{0, \{\tilde{x}_j\}_{j \in n_i}\}, n_i) < h(\{0, \{x_j\}_{j \in n_i}\}, n_i)$.

In words, item 1 says that when there are more users than non-users in i's neighbourhood, then the network effect gives a higher payoff when i chooses to use. Next, item 2 says that if the number of users and non-users in i's neighbourhood is the same, then the network effect gives no payoff, regardless of i's choice. Finally, item 3 says that the higher the number of users in i's neighbourhood, the higher the network effect payoff for i choosing to use. Similarly, the higher the number of users in i's neighbourhood the lower the network effect payoff for i choosing not to use.

Each period, the agent maximizes expected utility subject to the budget constraint,

$$\max_{x_i \in \{0,1\}, c \in \mathbb{R}_+} \mathrm{E}[v^i(x, c, s, n_i)] \text{ subject to } y \geq c + q x_i.$$

To ensure that the budget constraint holds with equality, I make an additional non-satiation assumption:

Assumption 4 (Non-satiation)**.** $\frac{\partial v^i(x,c,s,n_i)}{\partial c} > 0$ and $\frac{\partial^2 v^i(x,c,s,n_i)}{\partial c^2} < 0$.

With this assumption, the solution to the agent's choice problem is a simple comparison of expected payoffs. The agent chooses to consume the drug if the expected marginal benefit from consuming the addictive good is positive:

$$\mathrm{E}[v^i(\{1,\{x_j\}_{j\in n_i}\}, y-q, s, n_i)] \geq \mathrm{E}[v^i(\{0,\{x_j\}_{j\in n_i}\}, y, s, n_i)]. \tag{2}$$

Let $\hat{v}_1^i(s,n_i) = v^i(\{1,\{x_j\}_{j\in n_i}\}, y-q, s, n_i) - e_1$ and $\hat{v}_0^i(s,n_i) = v^i(\{0,\{x_j\}_{j\in n_i}\}, y, s, n_i) - e_0$ be the utility without the error term for choices $x=1$ and $x=0$, respectively, and define the random variable $\varepsilon = e_0 - e_1$, that is, equal to the difference between the two choice specific error terms. Then the inequality in (2) can be rewritten as

$$\begin{aligned} &\mathrm{E}[\hat{v}_1^i(s,n_i) + e_1] \geq \mathrm{E}[\hat{v}_0^i(s,n_i) + e_0] \\ \Leftrightarrow\ &\mathrm{E}[\hat{v}_1^i(s,n_i) - \hat{v}_0^i(s,n_i)] \geq \mathrm{E}[e_0 - e_1] \\ \Leftrightarrow\ &\mathrm{E}[\widehat{MB}(s,n_i)] \geq \mathrm{E}[\varepsilon] \end{aligned}$$

where $\widehat{MB}(s,n_i)$ is the error-free marginal benefit from choosing to consume the addictive good in addictive state s and neighbourhood n_i. As is common in the binary choice literature, I assume that these error terms are drawn from a type 1 extreme value distribution.

Assumption 5 (Error component)**.** For each individual, e_x is independently drawn from a type 1 extreme value distribution with cumulative distribution function $F(e_x) = \exp(-\exp(-\lambda e_x - \gamma))$ for $x \in \{0,1\}$.

A direct consequence of this assumption is that ε is logistically distributed[1] $\Lambda(\mu,\lambda)$, with mean $\mu = 0$ and scale parameter λ (McFadden, 1980). Thus, the probability of individual i consuming the addictive good is given by,

$$\begin{aligned} \mathrm{P}(x_i = 1|s,n_i) &= \mathrm{P}(\mathrm{E}[\hat{v}_1(s,n_i) - \hat{v}_0(s,n_i)] \geq e_0 - e_1) = \mathrm{P}(\varepsilon \leq \mathrm{E}[\hat{v}_1(s,n_i) - \hat{v}_0(s,n_i)]) \\ &= \frac{\exp\left(\mathrm{E}[\lambda(\hat{v}_1(s,n_i) - \hat{v}_0(s,n_i))]\right)}{1 + \exp\left(\mathrm{E}[\lambda(\hat{v}_1(s,n_i) - \hat{v}_0(s,n_i))]\right)} = \frac{\exp\left(\mathrm{E}[\lambda\hat{v}_1(s,n_i)]\right)}{\exp\left(\mathrm{E}[\lambda\hat{v}_0(s,n_i)]\right) + \exp\left(\mathrm{E}[\lambda\hat{v}_1(s,n_i)]\right)} \\ &= \frac{1}{1 + \exp(-\lambda \mathrm{E}[\widehat{MB}(s,n_i)])}. \end{aligned}$$

The derivation above reveals that the magnitude of the marginal benefit directly affects the probability of choosing to consume x. In particular, the relative weight of each option determines its likelihood of being chosen. If the preferences for both options are close, then the agent is susceptible to small shocks. In fact, when $\mathrm{E}[\widehat{MB}(s,n_i)] = 0$, the agent is equally likely to choose either option. However, if one action is preferred over the other by a large amount, then the likelihood of it getting chosen is also much higher.

[1] with CDF $F(\varepsilon;\mu,\lambda) = \frac{\exp(\lambda(\varepsilon-\mu))}{1+\exp(\lambda(\varepsilon-\mu))}$.

Before considering interaction, I make an additional assumption that puts some structure on the individual's behaviour. The literature in neuroscience and biology identifies several stages of addiction (for an overview see Koob and Moal, 2006, chapter 1). In general, the user begins with some infrequent experimentation, moves to regular and risky use, and then graduates to dependence. In other words, initially the probability of use is low and it increases with continued usage, reaching a maximum at the highest state of addiction. Say that agent i is *likely* (*unlikely*) to consume x if $\mathrm{P}(x_i = 1|s, n_i) > 0.5$ ($\mathrm{P}(x_i = 1|s, n_i) < 0.5$). When the probability of consuming the addictive good is exactly 0.5, the agent is at her *tipping point*. In the context of this model, to fit the stages of addiction, the agent is *unlikely* to use at the beginning, becomes *likely* to use as her state of addiction increases beyond some point, and uses with high probability at state $s = S$. The following assumption ensures that this is the case.

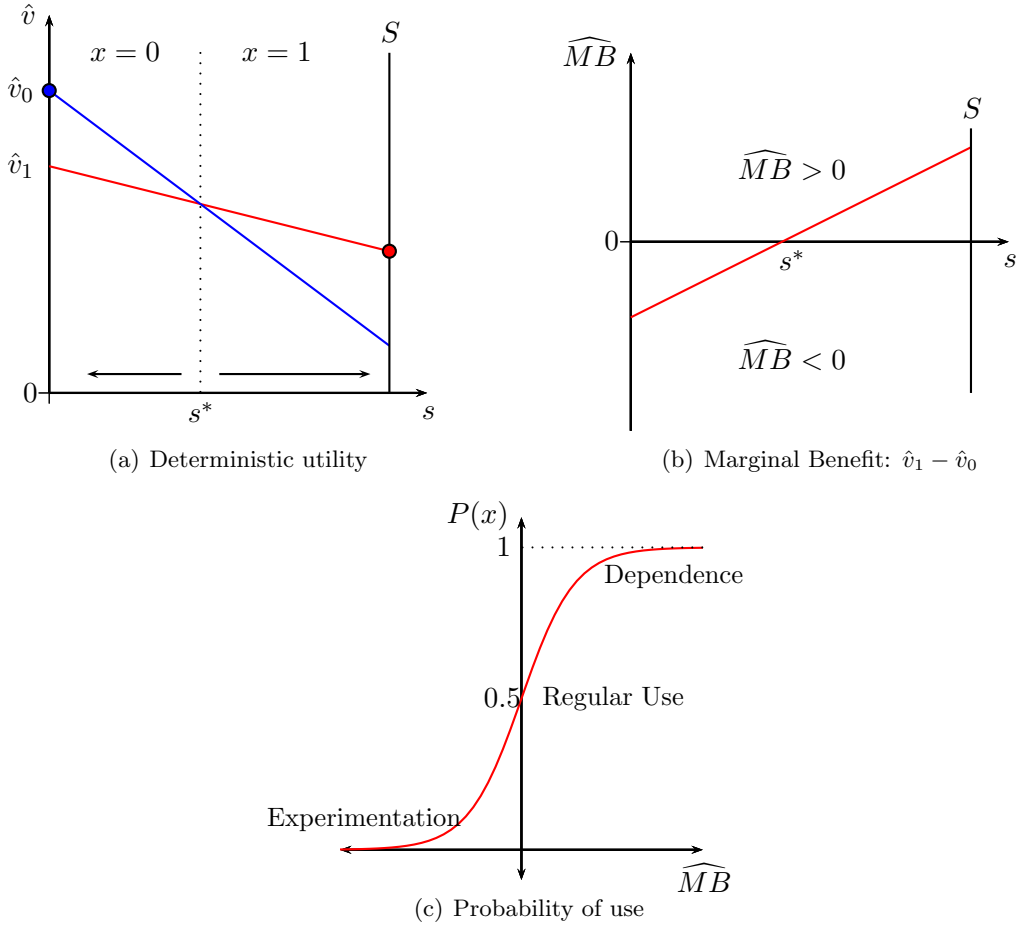
Assumption 6 (Stages of addiction)**.** For $n_i = \emptyset$, $\hat{v}_0^i(0, n_i) > \hat{v}_1^i(0, n_i)$, $\hat{v}_0^i(S, n_i) < \hat{v}_1^i(S, n_i)$, and $\hat{v}_0(s', n_i) - \hat{v}_0(s, n_i) > \hat{v}_1(s', n_i) - \hat{v}_1(s, n_i)$ for any $s > s'$.

The implication of assumption 6 are illustrated in Figure 2 below[2]. Note that, without network effects ($n_i = \emptyset$) the expected value of the network effect is zero and so $\mathrm{E}[\widehat{MB}(s, n_i)] = \widehat{MB}(s, n_i)$ because the remaining components are deterministic. Figure 2(a) plots the deterministic utility $\hat{v}_1$ and $\hat{v}_0$ as functions of s and no network influence for choices $x = 1$ and $x = 0$ respectively. The utility $\hat{v}_0$, is decreasing in s in response to growing demands of the affective system spurred by dependence. As the state of addiction increases, the individual must expend more willpower to resist consuming the drug and thus faces a growing utility loss. The utility $\hat{v}_1$ is also decreasing in s. The deliberative system rationally perceives the declining health effects and lowered hedonic experience. Eventually, the impulse to use becomes so strong that it overcomes the negative health effects in the individual's evaluation. This occurs at the *tipping point* s^*, where marginal benefit of consumption is exactly zero so that the agent is equally likely to consume the drug or not. Before the *tipping point*, the agent is *unlikely* to use the drug, but can still encounter shocks that push her to use. This is the "experimentation" stage. Once past the *tipping point*, the individual enters the "regular use" stage of addiction and becomes *likely* to continue to use until "dependence." In the final stage of addiction the individual can only return to a low state after a series of large negative shocks to the marginal benefit of use. Recall that the development of addiction is a gradual process and the agent only changes one state per period. Thus one large negative shock for someone in an advanced stage of addiction can result in them not using in a particular period, but it alone will not be enough for them to escape dependence.

Before discussing the network effect note that, for the individual, this model is consistent with several features of addiction. In particular, addicts often continue to use despite a rational awareness of decreasing utility (decreasing $\hat{v}_1$). Furthermore, they can express a desire to quit but be unable to do so ($\hat{v}_0$ decreasing faster than $\hat{v}_1$). Also, the model captures the stages of addiction.

To introduce influence, let the network effect depend on the average number of users in an

[2]Although utility is discrete in s and it could take on many shapes, it is plotted linearly here for simplicity.



(a) Deterministic utility

(b) Marginal Benefit: $\hat{v}_1 - \hat{v}_0$

(c) Probability of use

Figure 2: Stages of addiction for individual i with $n_i = \emptyset$

agent's neighbourhood n_i (as in Jackson and Yariv, 2007), so that

$$h(x, n_i) = g\left[x_i \frac{1}{d_i} \sum_{j \in n_i} x_j + (1 - x_i) \frac{1}{d_i} \sum_{j \in n_i} (1 - x_j)\right].$$

The network effects work in both directions. If the majority of neighbours are using, then the network effect rewards use in the sense that $h(0, n_i) < h(1, n_i)$. If, however, the majority of neighbours are not using, then $h(0, n_i) > h(1, n_i)$. The parameter g captures the magnitude of the network effect. For simplicity, I assume that g is equal across agents.

Since individuals have a random utility function, the equilibrium definition is non-standard. The following discussion draws heavily on McKelvey and Palfrey (1995) who define the equivalent of a Nash equilibrium for a game with players that calculate payoffs with a random utility function. Let $\Delta_i = \{p_i \in \mathbb{R}_+ | p_i \in [0, 1]\}$ be the set of all possible probabilities of choosing to consume the addictive substance for individual i and $\Delta = \prod_{i=1}^{N} \Delta_i$ be the set of possible probability measures for all N agents. Also, let $p_{-i} = \{p_j\}_{j \neq i}$ be a strategy profile and $\hat{v}_1^i(p_{-i})$ and $\hat{v}_0^i(p_{-i})$ the expected error-free utility (conditional on p_{-i}) for choices $x_i = 1$ and $x_i = 0$, respectively. Define agent

i's response set as $R_i(p_{-i}) = \{(e_0^i, e_1^i) \in \mathbb{R}^2 | \hat{v}_1^i(p_{-i}) + e_1^i \geq \hat{v}_0^i(p_{-i}) + e_0^i\}$. Thus, for a given strategy profile, individual i's statistical reaction function or quantal response function, is given by $\sigma_i(p_{-i}) = \int_{R_i(p_{-i})} \mathrm{d}F(e^i)$. In other words, $\sigma_i(p_{-i})$ is the utility-maximizing probability that individual i chooses to consume the addictive good given the strategy profile p_{-i}.

Definition 1 (Quantal Response Equilibrium)**.** A *quantal response equilibrium* is a mixed strategy $\pi^* \in \Delta$ such that $\pi_i^* = \sigma_i(\pi_{-i}^*)$ for all $i \in \{1, \ldots, N\}$.[3]

Furthermore, under the assumption that errors are independent across actions and individuals and drawn from the type 1 extreme value distribution described in Assumption 5, the probability of use depends on the relative weights of the expected utility from using and not using. Letting $v_0^i = \hat{v}_0^i - h(x, n_i)$, $v_1^i = \hat{v}_1^i - h(x, n_i)$, and $MB^i = v_1^i - v_0^i$, the probability of use for individual i is given by,

$$\pi_i^* = \frac{\exp\left[\lambda\left(v_1^i + \frac{1}{d_i}\sum_{j\in n_i}\pi_j^* g\right)\right]}{\exp\left[\lambda\left(v_1^i + \frac{1}{d_i}\sum_{j\in n_i}\pi_j^* g\right)\right] + \exp\left[\lambda\left(v_0^i + \frac{1}{d_i}\sum_{j\in n_i}(1-\pi_j^*) g\right)\right]}. \tag{3}$$

To understand the equilibrium and explore the implications of network effects, consider an example with two players i and j. The coordination game is shown in Figure 3 below. This is a simultaneous move game where player j does not know the choice (use (U) or do not use (D)) of player i when picking her own strategy. Since there are only two players, $h(x, n_i) = 0$ if they do not coordinate and $h(x, n_i) = g$ if they choose the same action.

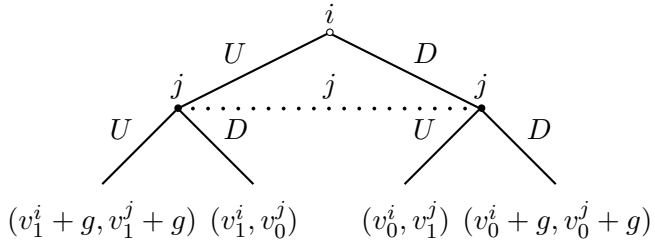


Figure 3: A two-player game of drug use

For player i, the expected utility from consuming the addictive good is given by $\pi_j(v_1^i + g) + (1-\pi_j)v_1^i$ and the expected utility from choosing $x_i = 0$ is $\pi_j v_0^i + (1-\pi_j)(v_0^i + g)$. Therefore, player i uses the drug when

$$\begin{aligned}
& \pi_j(v_1^i + g) + (1-\pi_j)v_1^i + e_1 > \pi_j v_0^i + (1-\pi_j)(v_0^i g) + e_0 \\
\Leftrightarrow \quad & (v_1^i + \pi_j g) - (v_0^i + (1-\pi_j)g) \geq e_0 - e_1 \\
\Leftrightarrow \quad & v_1^i - v_0^i - (1-2\pi_j)g \geq \varepsilon \\
\Leftrightarrow \quad & MB^i - (1-2\pi_j)g \geq \varepsilon.
\end{aligned}$$

[3]For more details and proof of existence see McKelvey and Palfrey (1995).

As a result, given definition 1, the equilibrium probabilities for players i and j are

$$\pi_i^* = \frac{\exp\Big(\lambda(\pi_j^*(v_1^i + g) + (1-\pi_j^*)v_1^i)\Big)}{\exp\Big(\lambda(\pi_j^*(v_1^i + g) + (1-\pi_j^*)v_1^i)\Big) + \exp\Big(\lambda(\pi_j^* v_0^i + (1-\pi_j^*)(v_0^i + g))\Big)} \tag{4}$$

and

$$\pi_j^* = \frac{\exp\Big(\lambda(\pi_i^*(v_1^j + g) + (1-\pi_i^*)v_1^j)\Big)}{\exp\Big(\lambda(\pi_i^*(v_1^j + g) + (1-\pi_i^*)v_1^j)\Big) + \exp\Big(\lambda(\pi_i^* v_0^j + (1-\pi_i^*)(v_0^j + g))\Big)}. \tag{5}$$

Note that (4) can be rewritten in the following way,

$$\pi_i^* = \frac{1}{1 + \exp\Big(-\lambda(MB^i + (2\pi_j^* - 1)g)\Big)}.$$

Thus, the probability to use for individual i is directly determined by the quantity

$$MB^i + (2\pi_j - 1)g. \tag{6}$$

As defined before, agent i is *likely* to use if $\pi_i > 0.5$. This occurs when the expression in (6) is positive. Likewise, agent i is *unlikely* to use if (6) is negative. A few remarks are in order. First, as in the case of no neighbours $n_i = \emptyset$, as the marginal benefit of use increases, the probability of use increases: $\pi_i(s') > \pi_i(s)$ for $s' > s$. Second, if $\pi_j = \frac{1}{2}$, the probability of use depends only on the marginal benefit from use and not the network effect.

Third, the effect of g, the magnitude of the network effect, depends on π_j,

$$\left.\frac{\partial \pi_i}{\partial g}\right|_{\pi_j > \frac{1}{2}} > 0, \qquad \left.\frac{\partial \pi_i}{\partial g}\right|_{\pi_j < \frac{1}{2}} < 0, \qquad \left.\frac{\partial \pi_i}{\partial g}\right|_{\pi_j = \frac{1}{2}} = 0. \tag{7}$$

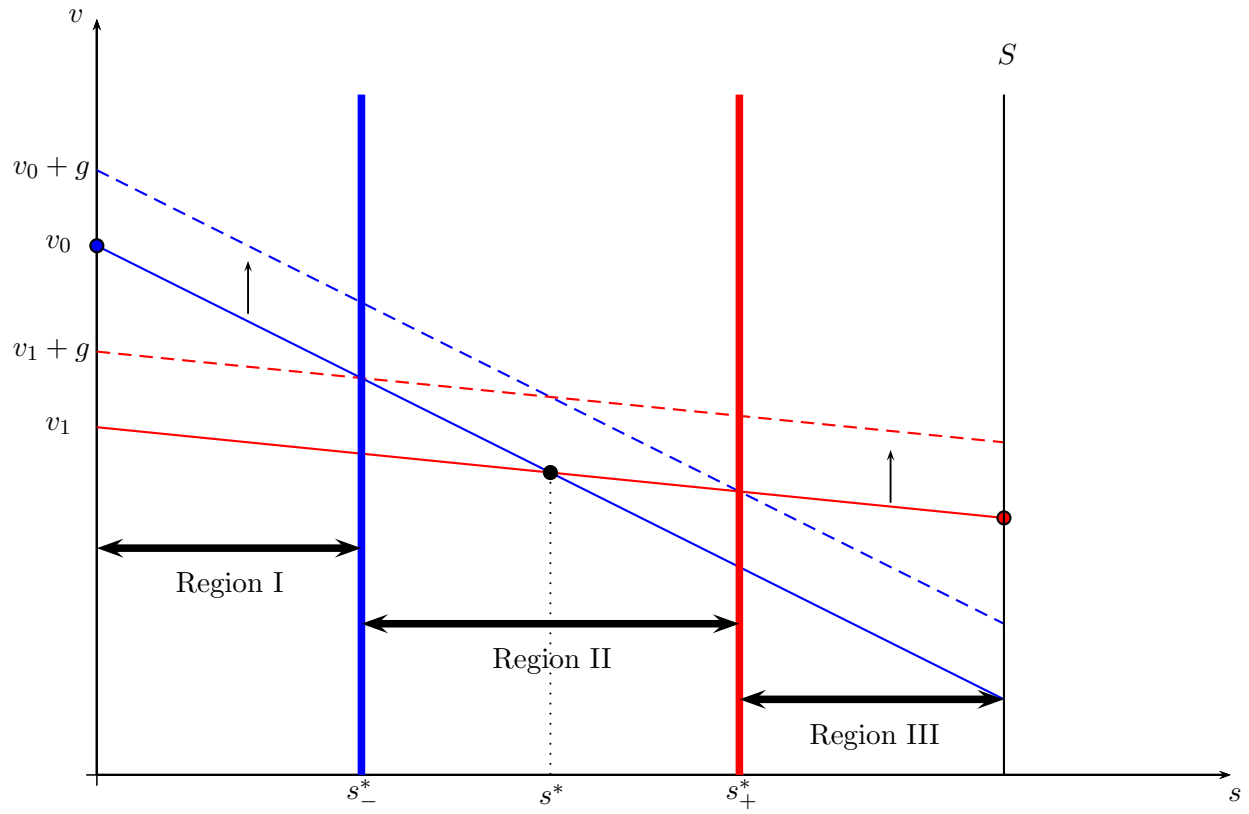
That is, if player j is *likely* to use, then increasing the parameter g raises the probability of use for player i. If, on other hand player j is *unlikely* to use, then an increase in influence lowers π_i. This effect captures the fact that influence can work in both ways. Also, it is through this mechanism that neighbourhood-related heterogeneity can lead to varying rates of recidivism and initial use for two otherwise identical individuals.

Fourth, the probability of use is unambiguously increasing in the other player's probability of use,

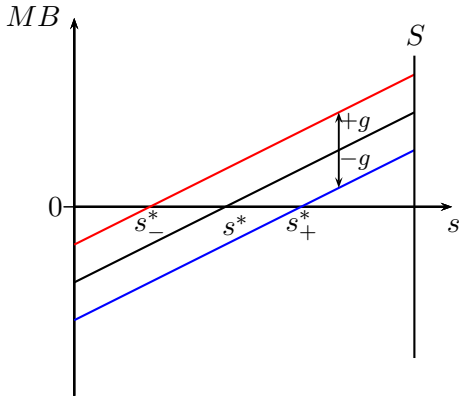
$$\frac{\partial \pi_i}{\partial \pi_j} > 0. \tag{8}$$

Fifth, if the expected network effect is as large as the marginal benefit, $MB^i = (1 - 2\pi_j)g$, then the player becomes indifferent, i.e. $\pi_i = \frac{1}{2}$. Figure 4(a) shows exactly how these effects influence individual i's utility. Coordinating actions with a player that chooses not to use shifts the v_0 curve up to $v_0 + g$. This is equivalent to shifting the MB down by g as shown in Figure 4(b). The result is a lower probability of use as demonstrated by a rightward shift of the probability curve

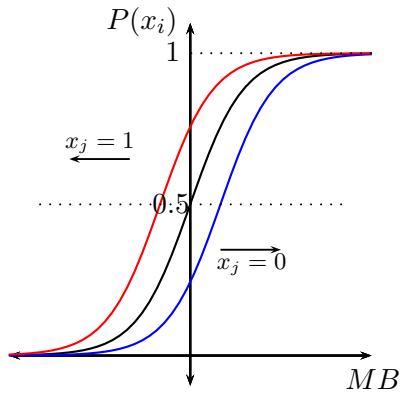
in Figure 4(c). Similarly, facing a player that chooses to consume the addictive good effectively shifts the v_1 curve up by the influence parameter g so that the marginal benefit increases for all s (upward shift in Figure 4(b)) and the probability of use is higher (leftward shift in Figure 4(c)).



(a) Deterministic utility with network effects



(b) Marginal benefit: $v_1 - v_0$



(c) Probability

Figure 4: Marginal benefit and probability of use with network effect

Incorporating the network effect creates three distinct regions in the agent's states of addiction. The borders of these regions occur at states where $|MB^i| = g$ and are marked by s^*_- and s^*_+ in Figures 4(a) and 4(b). As explained by the following definitions, these regions have important

implications for behaviour.

Definition 2 (Region I)**.** Region I is the set of states for which $-MB > g$. These states are $s \in [0, s^*_-)$ and if an individual is in this region then she is *unlikely* to use regardless of the behaviour of the other players.

Definition 3 (Region III)**.** Region III is the set of states for which $MB > g$. These states are $s \in (s^*_+, S]$ and if an individual is in this region then she is *likely* to use regardless of the behaviour of the other players.

These definitions are the direct consequence of the statistical reaction functions. Note that, player i is likely to use if

$$\frac{1}{1+\exp\left(-\lambda(MB^i+(2\pi_j-1)g)\right)} > \frac{1}{2} \tag{9}$$

$$\Leftrightarrow \quad MB^i + (2\pi_j - 1)g > 0 \tag{10}$$

$$\Leftrightarrow \quad \pi_j > \frac{1}{2} - \frac{MB^i}{2g}. \tag{11}$$

When player i is in region III, $MB^i > g$ so that the inequality in (11) is satisfied for any π_j because the right-hand-side is negative. In contrast, if she is in region I then $-MB^i < g$ and the inequality in (11) cannot be satisfied by any π_j because the right-hand-side is greater than one. Although the network effect alters the probability of use for agents in regions I and III, it is not large enough to make a *likely* user become an *unlikely* user and vice-versa. The set of states between regions I and III however, are different.

Definition 4 (Region II)**.** Region II is the set of states for which $|MB| \leq g$. These states are $s \in [s^*_-, s^*_+]$.

There are two cases when player i is in region II in which the network effect serves to *reinforce* behaviour. First, if player i is in addictive state $s \in (s^*, s^*_+]$ and player j is in region III, then player i is *likely* to use because (11) is satisfied (MB^i is positive and $\pi_j > 0.5$). Second, if player i is in addictive state $s \in [s^*_-, s^*)$ and player j is in region I, then player i is *unlikely* to use because (11) is not satisfied (MB^i is negative and $\pi_j < 0.5$).

Under what conditions can the network effect *alter* behaviour? Suppose player i is in region II in addictive state $s \in [s^*_-, s^*)$ and player j is in region III or i is in $s \in (s^*, s^*_+]$ and j is in region I. The next set of definitions describes certain conditions under which the player with addictive state in region II can become likely to conform to the other player's behaviour.

Definition 5 (Commitment Premium)**.** Consider a two-player drug use coordination game, where player i is in region II and player j is in region III. Player j is *likely* to use with a probability of at least 0.5. The *commitment premium*, given by $\frac{MB_i}{2g}$, is the additional level of commitment to use, from player j, that would make player i *likely* to use. That is, for player i to be *likely* to use, the probability of using for player j would have to be over the minimum by at least $\frac{MB_i}{2g}$. Similarly, if

player j is in region I, the *commitment premium* is the additional level of commitment not to use from player j that would make player i *unlikely* to use.

The *commitment premium* is determined by the marginal benefit of use for player i, the individual in region II. Therefore, we can identify a set of states for player i for which peer influence can be strong enough to alter behaviour.

Definition 6 (Zone of Susceptibility)**.** For a given state of addiction for player j, define the *zone of susceptibility*, Z, as the set of states for player i for which the probability to use π_j is large (small) enough to satisfy the *commitment premium*.

Thus if player i is in state $s \in Z$ then she is *likely* to choose the same action as player j even though alone she would be *likely* to do the opposite. Furthermore, the farther player i is from her tipping point, the greater the commitment premium.[4] This result is intuitive since the greater the marginal benefit in magnitude the higher the relative weight of either choice and the more unwilling is the individual to alter her decision. In order to be swayed, the probability of getting g must be high enough to account for this reluctance. Note that for an individual in regions I or III, $Z = \emptyset$.

In summary, for two players coordinating to use drugs we can expect the following results. If both payers are in region I then they are likely not to use the drug. If they are in region III, then consumption of the addictive good is likely for both players. If one player is in region II and the other player is in regions I or III then both are likely to choose the same action if the commitment premium is met. If the commitment premium is not met or both players are in region II, then the outcome is unpredictable because there are multiple equilibria.

Compared to the case of $n_i = \emptyset$, the presence of the network effect can reinforce or diminish the likelihood of an individual's action. The next series of theorems describes how changes in the model fundamentals affect the outcome.

Theorem 1 (Utility effects)**.** *A level increase (decrease) in the withdrawal and/or willpower cost affecting $v_0^i(s)$ causes the tipping point s^* to shift to the left (right), the size of region I (III) to decrease and the size of region III (I) to increase. A level increase (decrease) in the decline of health effects, hedonic experience, and/or resource cost q affecting $v_1^i(s)$ causes the tipping point s^* to shift to the right (left), the size of region I (III) to increase, and the size of region III (I) to decrease.*

Proof. Consider a level decrease of $v_0^i(s)$ (the proof for a level increase of $v_1^i(s)$ is the same). This causes the new marginal benefit, MB_n^i, to be greater than the original marginal benefit, MB_o^i, for all states $s > 0$. Thus, at the original tipping s_o^*, the new marginal benefit of use is greater than 0, and therefore, $MB_n^i(s_o^*) > MB_o^i(s_o^*)$. Since, by Assumption 6, MB^i is increasing in s and negative at $s = 0$, the new tipping point s_n^* must be at a lower state than the original one s_o^*. That is, $s_n^* < s_o^*$. Regions I and III are delimited by $|MB^i(s)| = g$. Since $MB_n^i > MB_o^i$ for all $s > 0$, $-MB_n^i(s_{n-}^*) = g$ and $MB_n^i(s_{n+}^*) = g$ must both occur at a lower state than in the original case (s_{o-}^*, s_{o-}^*). Consequently, the set $[0, s_{n-}^*)$ is smaller than $[0, s_{o-}^*)$ and the set $(s_{n-}^*, S]$ is larger than

[4] MB^i is zero at the tipping point and then increasing in magnitude for any state larger or smaller.

$(s^*_{o-}, S]$. The proof for a level increase of $v^i_0(s, n_i)$ (level decrease of $v^i_1(s, n_i)$) is exactly the same but with everything moving in the opposite direction. ■

According to Theorem 1, substances for which regular use begins at higher states of addiction (high s^*) make the individual vulnerable to network effects later than for substances with a faster onset of addiction (low s^*). For instance, it could be easier for someone who has consumed marijuana a few times to say no to a friend than it would be for someone who has started experimenting with heroin. Moreover, the network effect can be an important driving force in the transition from low to regular use. This is of particular importance for drugs that are often used in a social context such as tobacco and alcohol. A person who would normally abstain can be tempted to engage in social smoking or social drinking and develop an addiction more quickly when the probability of use in the neighbourhood is high. This effect works in both ways. Once regular use begins it is easier to stop when probability of use in an individual's neighbourhood is low. This mechanism shows that recidivism rates are likely to be higher for individuals in areas of high use, consistent with the empirical findings discussed in Section 1.

Theorem 1 also sheds some light on how government policy can affect an outcome. Suppose the government can alter the resource cost q of obtaining the addictive good. This can be achieved through policies such as higher police presence or direct disruption of the supply-chain. Note that by Assumption 4, an increase in q causes a level decrease of $v^i_1(s) \forall i$. For an individual close to her tipping point s^*, such a policy can alter her path away from addiction. On the other hand, for an individual in a state close to S, the policy might lower the probability of use but, unless the change is very large, not have a significant impact on long term behaviour. Furthermore, if the policy is only active for a small number of periods, then the individual is likely to return to regular use once it expires, even if it was large enough to get them to stop using for a few periods.

If two individuals are in region I, then the network effect serves to reinforce the policy that increases the resource cost in the sense that the policy is more effective when $n_i \neq \emptyset$. The opposite holds true if both individuals are in region III. In that case, the policy is more effective when $n_i = \emptyset$. In both cases, coordination offers a higher payoff. This finding implies that a policy aimed at reducing use by increasing the resource cost of a drug could be made more effective if complemented with a policy that encourages or discourages links between individuals. In low-use neighbourhoods, such policies could entail encouraging group activities and in high-use neighbourhoods links could be discouraged via curfews and restricting access to areas where individuals tend to gather.

The next theorem is mostly technical but serves to clarify the role of the error term.

Theorem 2 (Effect of error variance)**.** *As $\lambda \to \infty$ the zone of susceptibility $Z \to$ region II. As $\lambda \to 0$ the zone of susceptibility $Z \to \emptyset$ and $\mathrm{P}(x_i = 1|s, n_i) \to 0.5$.*

Proof. As $\lambda \to \infty$, the probability of use for player j, given by $\frac{1}{1+\exp(-\lambda(MB^j+(2\pi_i-1)g))}$, goes to zero for $MB^j+(2\pi_i-1)g < 0$ and one for $MB^j+(2\pi_i-1)g > 0$. Therefore, player j uses with probability one in region III and with probability zero in region I. Since the commitment premium for player

i in region II is $\frac{MB^i}{2g} \in \left[-\frac{1}{2}, \frac{1}{2}\right]$, it is always satisfied and the zone of susceptibly covers the entire region II. As $\lambda \to 0$, the probability of use for player j, $\frac{1}{1+\exp(-\lambda(MB^j+(2\pi_i-1)g))} \to \frac{1}{1+\exp(0)} = \frac{1}{2}$. ■

Recall that λ is the inverse of the variance of the random variable ε. As the variance of the error term gets very large ($\lambda \to 0$), all of the other effects get washed out and the individual behaves randomly choosing to use with a probability of 0.5 regardless of her state or her neighbourhood. As the variance collapses to zero ($\lambda \to \infty$), decisions become deterministic. For an individual in region II the probability of coordinating with a region I or region III player becomes 1. At the same time, a very low variance eliminates all of the dynamics in the model. Agents in region I stay in region I and agents in region III stay in region III with probability 1. Clearly λ is an important parameter to consider if this model is taken to the data.

Theorem 3 (Strength of link)**.** *An increase (decrease) in the influence parameter g results in an increase (decrease) in the size of region II and a decrease (increase) in the size of regions I and III.*

Proof. Consider an increase in the influence parameter $g' > g$. Regions I and III are delimited by $|MB^i(s)| = g$. Let the original delimiting states be s^*_{o-} for region III ($-MB^i(s^*_{o-}) = g$) and s^*_{o+} for region I ($-MB^i(s^*_{o+}) = g$). Since MB^i is increasing in s, it must be the case that the new delimiting states are $s^*_{n-} < s^*_{o-}$ for region I ($-MB^i(s^*_{o-}) = g < g' = -MB^i(s^*_{n-})$) and $s^*_{n+} > s^*_{o+}$ for region III ($MB^i(s^*_{o+}) = g < g' = MB^i(s^*_{n+})$). As a result, the set $[0, s^*_{n-})$ is smaller than $[0, s^*_{o-})$ and the set $(s^*_{n-}, S]$ is smaller than $(s^*_{o-}, S]$. ■

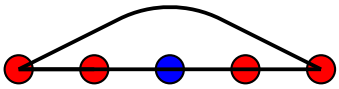
Theorem 3 describes how the strength of links affects behaviour. First, note that if $g = 0$ there is no added payoff for coordination and an individual behaves as if her neighbourhood is empty. As g gets larger, region II and the zone of susceptibility, increase in size. For two players in region I or region III facing a very large g there is a strong incentive to coordinate. Therefore, we expect individuals in these situations to stabilize around a low ($s = \{0, 1\}$) or high state ($s = S$) of addiction, respectively. Although there is a very low probability that both individuals receive a large enough shock (or series of shocks) so that they decide to consume the addictive good and end up in region II, if this does occur then the outcome is very volatile. Once in region II, due to the multiple equilibria, behaviour becomes volatile and unpredictable. Two players could quickly end up in region III where they would be likely to remain until another very large shock simultaneously arrives for both of them to stop use. Thus, for large g the system exhibits strong absorption - if both agents are in states $s = \{0, 1\}$ or $s = S$ then there is low probability of them leaving - and more volatility - the set of states for which multiple equilibria arise is large. At the other extreme, for a very small g the system exhibits weak absorption - not much stronger than if the individuals behave independently - and low volatility. Since region II is relatively small, even after a few instances of use agents can return to a low use state.

The strength of the network parameter g has immediate effects on system stability and diffusion of addiction. To explore these effects further, the next section uses simulations to examine behaviour and dynamics of multi-player games under various conditions.

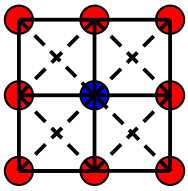
3 N-player Behaviour

The theoretical model for two players provides a lot of intuition for the impact of influence on behaviour but it limits analysis to comparisons of static equilibria. Using a computational model, this section extends the analysis to N-players and shows the evolution of aggregate behaviour and the impact of changing network topologies. I analyze the spread of addiction with different numbers of links d_i and various magnitudes of the influence parameter g. Simulation requires explicit descriptions of the space, agent attributes, methods of interaction, initial conditions, as well as decision rules. Definitions of these components are provided below and followed by a discussion of the simulation strategy and results.[5]

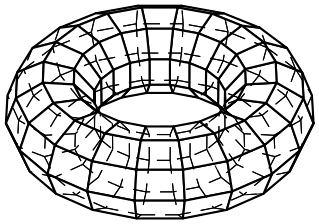
For simplicity, I only consider regular networks, i.e. where each node has the same degree. The most basic network formation is a circle which is represented by a line of agents with the two endpoints connected. In this configuration, as shown in Figure 5(a), each agent has degree two, sharing one link with each adjacent neighbour. Adding one more dimension results in an alternative network formation represented by a torus. This shape, shown in Figure 5(c), is a rectangle wrapped in such a way that its opposing sides are connected to each other. Agents can have either 4 links - one for each agent to the north, south, east, and west - or 8 links - adding connections to neighbours to the north-west, north-east, south-west, and south-east. Figure 5(b) shows these two configurations as they would appear on a section of the torus surface. In addition, for comparison, I also provide results for the N-player game with $N-1$ links, i.e. with all players connected to each other.



(a) Circle with each agent connected to a neighbour on both sides



(b) Section of a torus with 4 links (solid lines) and 8 links (solid and dashed lines) from the perspective of the centre node



(c) Torus

Figure 5: Network structures

In the static model, when more than one agent is in region II, multiple equilibria arise and there is no criterion for selecting one equilibrium strategy over another. Therefore, in order to consider a dynamic version of the model, agents best respond to the information received from the previous period. This is a similar process to diffusion over a network discussed by Jackson and Yariv (2007). In that paper, the authors look at what network properties affect the adoption of

[5]For a detailed discussion of parameterization see Appendix A.

a new technology or behaviour in a population. Once behaviour converges to a stable point, this steady state corresponds to an equilibrium of the static game. While their paper has only two possible outcomes - complete adoption or failure to adopt - my model has several and I compare them by observing how the system evolves under various conditions.

Recall (3) from definition 1. The equilibrium probability of consuming the addictive good depends on the marginal benefit of use MB^i and the expected value of the network payoff. I assume that each agent uses last period's information as a prediction for the expected network payoff in the current period. Thus, the probability of use is determined by

$$MB^i + \frac{1}{d_i}\sum_{j\in n_i}(\pi_j)g - \frac{1}{d_i}\sum_{j\in n_i}(1-\pi_j)g = MB^i + \frac{g}{d_i}\sum_{j\in n_i}(2x_j - 1), \tag{12}$$

where n_i is the set of agent i's neighbours, d_i is the size of that set, and all x_j's are neighbours' actions from the previous period.

I consider an environment with eleven stages of addiction $s \in \{0, \ldots, 10\}$ with the tipping point at $s^* = 5$. The objective is to explore how changing the strength of the influence parameter g and increasing the number of links alters the evolution of addiction for various initial conditions. These initial conditions include two scenarios: (1) the majority of the population is in advanced stages of addiction, and (2) the majority of the population is in the experimentation stages of addiction.

Simulation proceeds as follows. For a given network, agents are each assigned to a particular node, implicitly defining their neighbourhoods. Agent attributes take on two forms, private and public, and only the latter is used in communication with neighbours. Private attributes include the agent's current state of addiction, used to calculate MB^i, while public attributes contain the agent's location and the previous period's choice x_i. Agents all begin with same addictive state, which depends on the experiment, and are all identical at $t = 0$. For all of the experiments, the number of agents is $N = 100$. At each iteration, there are two steps: (1) an intra-period step where information is gathered and payoffs are calculated, and (2) an inter-period step where agents act based on the probabilities obtained in step (1). To generate some variation in the population, for experiments where $g > 0$, the network effect only becomes active after 300 periods. Simulations run for $T = 1000$ periods and in each case, the benchmark is given by the simulation with $g = 0$, i.e. no network influence. The length of simulation is chosen so that a pattern of behaviour can be clearly identified but is otherwise arbitrary. Each experiment is repeated 5000 times and the reported results are given as simulation averages.

Table 1: Average number of users, $\frac{1}{N}\sum_{i=1}^{N} x_i$, at $t = T$ in different initial environments

	High-use ($s = 6$)				Low-use ($s = 4$)			
	$d_i = 2$	$d_i = 4$	$d_i = 8$	$d_i = 99$	$d_i = 2$	$d_i = 4$	$d_i = 8$	$d_i = 99$
$g = 0$	0.8306	0.8306	0.8306	0.8306	0.1695	0.1695	0.1695	0.1695
	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)
$g = 2$	0.8928	0.8877	0.8883	0.8635	0.1077	0.1124	0.1119	0.1370
	(0.0017)	(0.0016)	(0.0017)	(0.0022)	(0.0017)	(0.0016)	(0.0016)	(0.0020)
$g = 3$	0.9304	0.9421	0.9421	0.9948	0.0690	0.0588	0.0590	0.0054
	(0.0018)	(0.0014)	(0.0014)	(0.0006)	(0.0019)	(0.0015)	(0.0014)	(0.0006)
$g = 4$	0.9345	0.9844	0.9842	0.9997	0.0650	0.0156	0.0161	0.0004
	(0.0017)	(0.0006)	(0.0006)	(0.0000)	(0.0017)	(0.0006)	(0.0007)	(0.0000)

Notes: Standard errors are provided in parenthesis below each average. All statistics are given for period T=1000, averaged over 5000 replications.

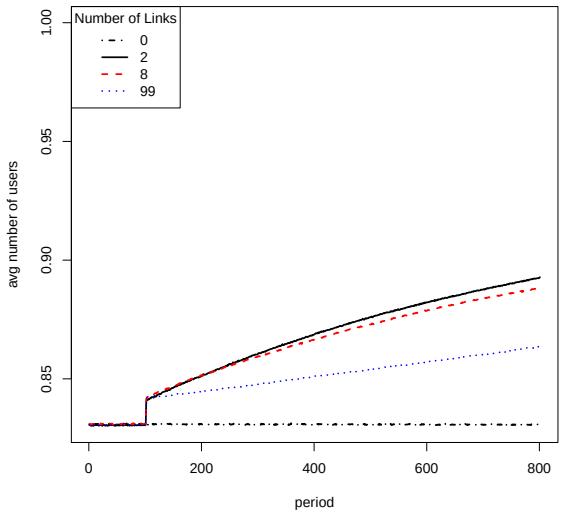
Table 1 reports the average number of agents choosing to consume the addictive substance at the end of the simulation for two scenarios with different initial conditions.[6] The first scenario has the state of addiction set to 6 across all agents and represents a high initial use environment and the second sets it to 4 to mimic a low initial use environment. The various columns show the results for different degrees and the various rows correspond to different strengths of the network effect.

In the high-use environment about 83.1% of the population consumes the addictive good when there are no network effects present. As predicted by the results in the two-player game, for a given number of links, increasing the strength of the network parameter increases the proportion of agents consuming the addictive good. However, the effect of increasing the number of links for a given network strength is not as apparent. First, moving from 2 to 4 links appears to change the results, but moving from 4 to 8 has no significant impact. When the number of links increases significantly, as in the case of $d_i = 99$, the difference in behaviour is much clearer.

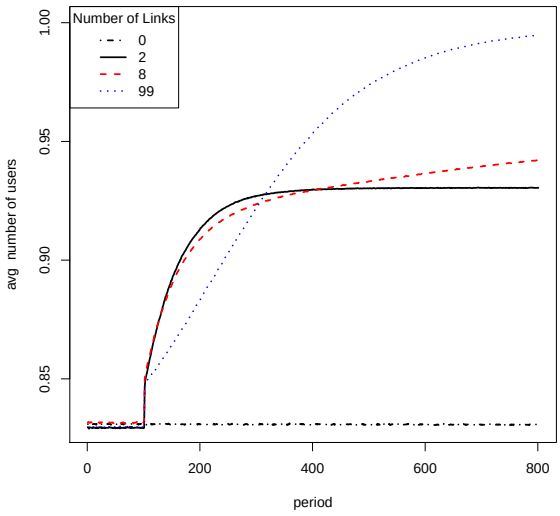
When the strength of the network parameter is relatively weak, $g = 2$, increasing the number of links lowers the average use in the economy. However, when the strength of the network parameter is relatively strong, $g = 4$, increasing the number of links increases the average use in the economy. In fact, when there are few strong links ($g = 4$, $d_i = 2$), the diffusion of addiction is faster, with 93.5% of the population using at the end of the simulation, than when there are many weak links ($g = 2$, $d_i = 99$), with 88.8% of the population using.

These results are similar for the low initial use environment. The majority behaviour is reinforced as the strength of the network parameter increases for a given number of links and drug use is eliminated more quickly when there are few strong links, with only 1.6% of agents using at the end of the simulation, than when there are many weak links, with 13.7% of the population using.

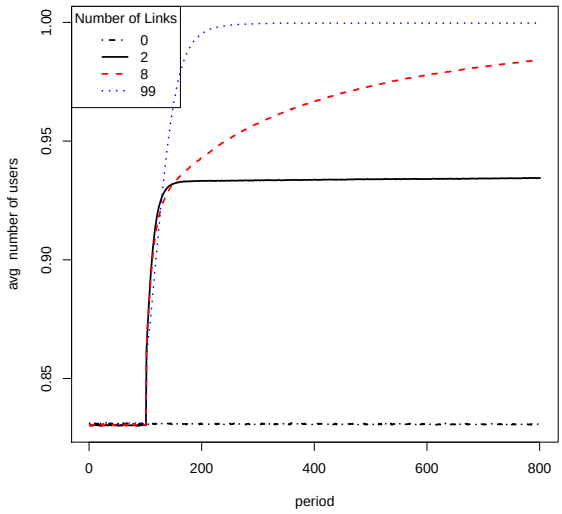
[6]Several other summary statistics were considered but are not reported here. These include the average addictive state, the average number of agents in the highest state of addiction, and the average probability of use. The results using these other measures were qualitatively the same. Furthermore, other regular networks (with $d_i = 20$, 40, and



(a) $g = 2$



(b) $g = 3$



(c) $g = 4$

Figure 6: Simulation results for high initial use

To gain a better understanding of this result, Figure 6 plots the simulation paths for various numbers of links and network strengths in the initial environment with high use. As shown in Figure 6(a), addiction spreads faster with fewer links when the connections are weak. Figure 6(c) shows that this difference becomes more pronounced and reverses order when the connections are strong. Finally, Figure 6(b) reveals that when the strength of the links is in between, addiction

60) were also considered and led to the same conclusions. All of these additional results are available upon request.

initially spreads faster with fewer links but by the end of the simulation the environment with many links has more addicts. For the environment with low initial use, the same plots appear as mirror images of the ones shown in Figure 6 and reveal the same dynamics (see Figure 7 in Appendix B).

The simulations shown here imply that the speed of diffusion is critically related to both the number of links and their strength. Although addiction can spread the fastest when there are many strong links, this rate of diffusion can be greatly reduced as the network effect is weakened. This finding has implications for the role of networks in understanding the spread of addiction in different communities. For instance, rural towns with few inhabitants that know each other very well (few strong links) might be more susceptible to the diffusion of addiction than those living in large urban areas and interacting with many strangers (many weak links). Furthermore, knowledge of the network configuration can provide important insights for a policy maker dealing with addiction. For example, as part of a drug-deterrent policy, disrupting links can have a greater effect when there are few strong links than when there are many weak links.

Interestingly, the environment that is best at keeping use low is also the one that is most susceptible to the fastest spread of addiction. Thus, it is important to keep in mind that network effects are only one factor in explaining differences in addiction across environments. Nevertheless, as this section reveals, understanding the role of the network configuration can be an important tool in assessing the threat of drug addiction or dealing with its spread.

4 Conclusion

This paper introduces network influence into a dual-self model of addiction. On the individual level, it is consistent with previous studies that model decision making under addiction as a trade-off between two competing systems in the brain. Furthermore, it captures the dynamics of the transition from experimental use to dependence.

I analyze interaction in the two-player game using a quantal response equilibrium. As the strength of influence rises so does the zone of susceptibility or the set of states in which the agent is susceptible to behaviour-altering network effects. Also, as influence rises so does the absorption into dependence or abstention. If both players are in low states, they are more likely to stay there. If they are both in advanced states of addiction, then their connection reinforces their behaviour and they are less likely to stop use than if they were acting alone.

Next, I explore a dynamic version of the model using simulations. For standard network topologies, increasing the strength of the network parameter reinforces the majority behaviour. Changing the number of links however, has different effects based on the strength of network influence. The majority behaviour spreads faster with less links when connections are weak and with more links when connections are strong.

These results have several implications for policy. For example, the network based explanation of heterogeneity in relapse rates provides a theoretical foundation for implementing post rehabilitation relocation programs aimed at improving the probability of recovery. Also, in high use

environments, depending on the number of links and their strength, disrupting connections can make drug deterrent policies more effective.

In comparison to previous literature, criminalization has similar effects in my model as in Bernheim and Rangel (2004). If I adopt the same welfare criterion as they do, namely the utility as determined by the deliberative system, then laissez-faire is the optimal policy so long as an individual's hedonic experience exceeds the negative health effects of consuming the addictive good. When the reverse is true but the affective system imposes a large enough cost to make the individual likely to use, then policies limiting access are optimal. However, by incorporating the network effect, my model adds an important dimension to the policy analysis and shows how the environment can impact effectiveness.

Furthermore, the affective or "cold" system in Bernheim and Rangel (2004) is very simple and always desires consumption of the addictive good, regardless of the state of addiction. But as Loewenstein and O'Donoghue (2004) point out, choosing the welfare criterion becomes complicated when the affective system is more complex. Specifically, if we also consider the utility lost from expending the willpower required to appease the affective system then it is no longer clear that the deliberative system alone is an appropriate welfare criterion. For this reason, the focus in this paper is on positive rather than normative analysis.

The model presented in this paper serves as an important stepping stone for future exploration of the role of networks in drug addiction. There are two main directions for future work. One is to endogenize the network. In all of the discussion in this paper, the network structure has been imposed exogenously. Nevertheless, there are many instances where people do not have much influence over their networks. For instance, growing up in a certain neighbourhood exposes an individual to peers particular to that area. However, drug addiction can shatter friendships and spawn new ones, creating dynamics that are far more complicated than the ones considered in the current model. The other element that has been exogenous in this paper is the supply side. It is possible that certain areas of a network have easier access to drugs or that drug-suppliers target areas where addicts conglomerate. This could create heterogeneity in the resource cost of the addictive good that would translate to pockets of agents across the network with increased susceptibility to use.

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A Computational Model Parameterization

Building a computational model requires explicit parameterization of the entire environment. I start by describing the individual behaviour rules and then discuss the model parameters.

Recall from (12) that probability of use is entirely determined by the quantity

$$\frac{1}{1+\exp(-\lambda(MB^i + \frac{g}{d_i}\sum_{j\in n_i}(2x_j - 1)))}, \tag{13}$$

where x_j is the only endogenous variable. The number of links d_i and neighbourhood n_i are specified by the network structure as discussed previously. The components that need to be given values are MB^i, g, and λ. Furthermore, since MB^i is a function of the addictive state s, I must also specify the maximum state of addiction S.

I assume that the function $MB^i(s)$ takes on the following form,

$$MB^i(s) = MB_0 + \psi s.$$

Here, MB_0 represents the marginal benefit from use in state $s = 0$. The other parameter, ψ, captures the difference between the response of the deliberative system and the affective system to a change in degree of addiction s. If the difference is positive, the marginal benefit of use increases each time the agent uses. If ψ is negative, the decline in utility from continual use is greater than the willpower cost of controlling the affective system, and marginal benefit is decreasing in s. For Assumption 6 to be satisfied, MB_0 must be negative and $MB_0 + \psi S$ must be positive.

An agent is fully characterized by five parameters: $\{\lambda, g, S, MB_0, \psi\}$. I let S and λ be homogeneous over the entire population. S is the maximum degree of addiction and it affects the length of the addiction-state-interval and thus how long it takes for an agent to reach her tipping point s^* and develop dependence. For a given S, s^* can also shift as a result of changes to parameters MB_0 and ψ. If the tipping point is close to the middle of the interval $\{0, \ldots, S\}$, then a larger S implies a longer path to the tipping and a longer descent into state S once the tipping point has been passed. However, the speed of moving to s^* and eventually S can be determined by MB_0 and ψ because they directly impact the probability of transitioning between states. Therefore, I set $S = 10$, and, let the other parameters determine the speed and likelihood of descent into heavy use.

The scale parameter, λ, governs how responsive the agent is to shocks. For $\lambda \to \infty$, the error term plays no role and decisions are based solely on the sign of the marginal benefit. For $\lambda = 0$, behaviour is completely random and the marginal benefit has no impact on an agent's choice.

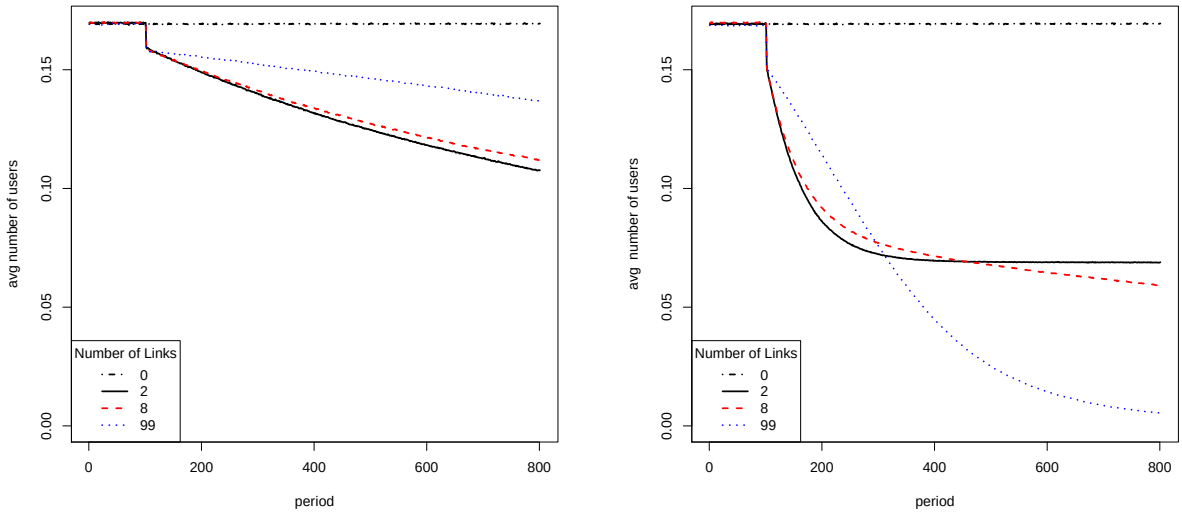
In order to obtain similar absorption strengths for $s = 1$ and $s = 10$, I choose $MB_0 = -5$ and $\psi = 1$. Furthermore, to add some more dynamics to the model, I set $\lambda = 0.8$. The resulting

transition probabilities are

$$P(s'|s) = \begin{bmatrix} 0.98 & 0.02 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.96 & 0.04 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.92 & 0 & 0.08 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0.83 & 0 & 0.17 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0.69 & 0 & 0.31 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.50 & 0 & 0.50 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0.31 & 0 & 0.69 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0.17 & 0 & 0.83 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.08 & 0 & 0.92 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.04 & 0 & 0.96 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.02 & 0.98 \end{bmatrix}$$

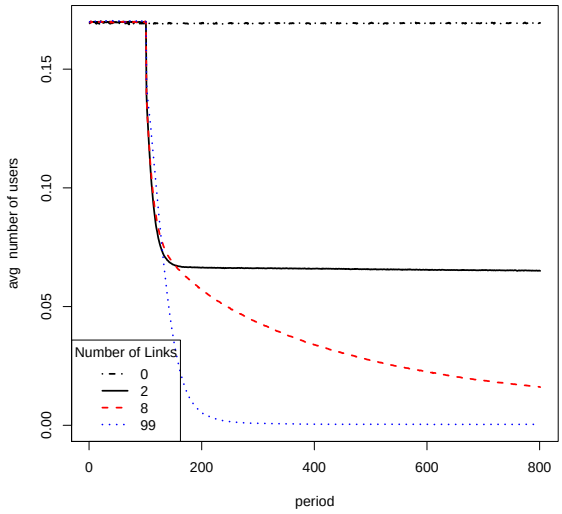
where each element P_{ij} represents the probability of an agent in state i moving to state j.

B Additional Simulation Results



(a) $g = 2$

(b) $g = 3$



(c) $g = 4$

Figure 7: Simulation results for low initial use