

# **SOCIAL INFLUENCES AND SMOKING BEHAVIOR**

FINAL REPORT TO THE AMERICAN LEGACY FOUNDATION

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SOCIAL INFLUENCES AND SMOKING BEHAVIOR

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## I. FINAL NARRATIVE

The objectives of this project were to conduct a comprehensive study of social influences on smoking behavior using an agent-based modeling approach. The results of the research can be divided into three parts. The first part was devoted to understanding the physiological and psychological bases of nicotine addiction in order to integrate them into the model. This work is summarized in two papers, "Social Influence, Reactance, and Tobacco," by Ross Hammond, and "Addiction and Cessation Functions in the Agent-Based Smoking Model" by Zirui Song. The second part of the research involved a statistical analysis of social network structures among teenagers, which function as conduits for initially taking up smoking and also for quitting based on targeted interventions. These findings are reported in the paper "How to Form a Network of Junior High School Students," by Ben Klemens, and form an integral part of the computer models described below.

The third and principal component of the research was to construct agent-based computer models that can be used to simulate the impact of various types of policy interventions. This work was carried out by Robert Axtell, Joshua Epstein, Ross Jon Parker, and Peyton Young. The project produced two models that are designed to study different types of effects.

The first model developed by Joshua M. Epstein, Ross Hammond, Jon Parker and H. Peyton Young is entitled "An Agent-Based Smoking Policy Model." It is designed to investigate the effects of highly targeted interventions, such as smoking cessation campaigns in schools. This model incorporates the physiological and psychological reactance variables identified in the studies mentioned below. It allows the user to simulate social networks among teenagers, and thus to investigate the expected effects of targeted policy interventions and information campaigns aimed at reducing smoking. In particular the model demonstrates how social network structure, and the different roles played by friends and opinion leaders, dramatically affects the rate at which smoking norms take hold in a given social setting. It also

provides a way of testing the effectiveness of different messages, given the peer network structure, and the distribution of reactance in the network.

This is a fully interactive web-based model with an accompanying user manual. The model can be used by other researchers and policy makers to examine the effects of specific types of interventions, allowing users to vary the parameter values as they see fit, including such factors as density and strength of network interactions, degree of reactance to messages, and physiological differences in rate of addiction. This flexibility in model design means that the predictions do not hinge on any one set of assumptions about the true parameter values. Instead the user can explore the robustness of a given type of intervention under alternative hypotheses.

The second model, developed by Robert Axtell and entitled “Smoking Behavior via Peer Effects in Social Networks,” allows the user to examine how smoking behavior spreads within specific subgroups of the population, based on the density of network interactions both within and across subgroups. This type of model can be calibrated against aggregate data on smoking rates by age, gender, and ethnicity, and can be used to predict how these rates respond to large-scale interventions such as truth campaigns.

## RESEARCH PAPERS

### **A. “Addiction and Cessation Functions in the Agent-Based Smoking Model” by Zirui Song.**

This paper proposes mathematical relationships for the probability of addiction as a function of initial smoking rates among adolescents, and also the probability of being able to quit as a function of the duration and intensity of smoking. The estimates are based on an extensive review of the public health literature. The evidence suggests that both addiction and cessation rates may vary substantially according to gender and ethnicity. The estimated functional forms are incorporated into an agent-based smoking model that simulates the evolution of smoking behavior among networks of adolescents.

**B. “Social Influence, Reactance, and Tobacco” by Ross Hammond.**

Psychological reactance theory refers to the tendency of individuals to respond negatively to certain messages if they believe their freedom of action is threatened. Reactance includes a desire to engage in the proscribed, detrimental behaviors and to engage in even more extreme versions of the behavior. It may also prompt individuals to persuade their peers to engage in the behavior. This paper reviews the scientific literature on reactance theory and discusses its implications for designing public health campaigns to reduce smoking among young people.

**C. “How to Form a Network of Junior High School Students,” by Ben Klemens.**

This paper describes a statistical method for producing artificial networks whose topological characteristics match those of actual social networks in junior high school classrooms. The model was developed using data on social network structure in a large sample of southern California junior high schools. We use the approach to model the density of links in such networks, and find that the Gamma distribution gives the best results among several alternative functional forms.

**AGENT-BASED MODELS****A. Agent-Based Smoking Policy Model**

*Joshua M. Epstein, Ross Hammond, Jon Parker and H. Peyton Young*

As noted above, the Policy Model integrates much of the scientific data presented in the above papers. It builds up an individual (agent) smoking decision function, incorporating individual biology (the addiction function), individual psychology (reactance, and skepticism toward USG and NGO messages), weighted social networks, and messages conceptually arrayed from -1 (maximally negative) to 0 (neutral). The

precise functional form of this decision function (to smoke or not to smoke) is given in the attached Power Point presentation (See Section V).

The main points of this modeling exercise are as follows: In networks where low reactance predominates, or where high reactance kids are low weight and/or low degree, the extreme message of -1 may be effective. However, in networks where high reactance kids have high weight and high degree, a message of -1 can backfire, and actually increase smoking. Here, the policy aim is to find a message strong enough to deter the uncommitted but not strong enough to induce the reactance (forbidden fruit) epidemic. This is the policy “sweet spot.” It will be network-specific, enforcing our claim that *optimal messages must be heterogeneous, tailored to specific communities, and adaptive over time.*

In addition to the power point presentation on these policy conclusions (See Section V), we include a User Manual (Section II) for the software located in Section IV.

**B. “An Agent-Based Computational Model of Smoking Behavior Among Adolescents Due to Peer Effects: Coordination with Addiction in Social Networks,”** *Robert L. Axtell.*

Adolescent smoking behavior is modeled as a coordination game played by purposive individuals within social networks. Payoffs to coordinated plays of the game change over time to reflect addiction. This model is a variant of a more general stream of research involving coordination games played adaptively by agents in social networks. A key feature of this model is that the population consists of different *types* of agents: some adapt to their environment, while others are non-adaptive either because they are stubborn (have fixed behaviors) or because they act unpredictably. The contribution of the paper is to show how population heterogeneity affects the spread of a “contagious” behavior such as smoking.





## PERSONNEL

### **Principal Investigators**

Robert L. Axtell, Senior Fellow, The Brookings Institution: A pioneer in the field of agent-based computational modeling. Co-author with Joshua Epstein of *Growing Artificial Societies: Social Science from the Bottom Up*, Cambridge MA: MIT Press.

Joshua M. Epstein, Senior Fellow, The Brookings Institution and External Faculty Member, The Santa Fe Institute. Leading authority on agent-based computational modeling and its application to economics, political science, and public health issues. Author of numerous articles and books, including *Nonlinear Dynamics, Mathematical Biology, and Social Science* (Wiley, 1997) and (with Robert Axtell) *Growing Artificial Societies: Social Science from the Bottom Up*, Cambridge MA: MIT Press.

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### **Internal Support**

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## II. RESEARCH PAPERS (1 OF 3)

# Addiction and Cessation Functions in the Agent-Based Smoking Model

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March, 2005

### Abstract

Few studies have studied the relationship between nicotine addiction and smoking for adolescents in a quantitative manner. This paper looks at the agent-based approach to modeling smoking behavior and proposes mathematical relationships for the probabilities of addiction and cessation as functions of smoking rates in adolescents. Published data is used to build their functional forms. Incorporating adolescent addiction and cessation functions into the agent-based model improves its ability to illustrate the dynamics and evolution of smoking behavior within adolescent peer networks over time.

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## **PART 1: SMOKING, DATA, AND THE MODEL**

### **1.1. Epidemiology**

Cigarette smoking is the leading preventable cause of death in the United States, responsible for approximately 440,000 annual mortalities (CDC, 2002). Although the overall prevalence of smoking has declined slightly in recent years—from 42.4% in 1965 to 23.4% in 2001 for adults, and from 36.5% in 1997 to 21.9% in 2003 for adolescents (CDC, 2004; CDC, 2003; CDC, 1999)—and drastic improvements have been made in the fields of surveillance, public policy, education, and tobacco research, the imminent threat of smoking to public health in the coming decades is only growing. Smoking damages every organ of the human body (USDHHS, 2004), and approximately five million of today's young adults will die prematurely from smoking-related illnesses (CDC, 1996), with each losing close to fifteen years of potential life (CDC, 2002). Smoking also costs \$157 billion in health-related expenditures annually, contributing to the 14.6% of U.S. GDP spent on health care, which is by far the most in the world (OECD, 2004).

The most important step smokers can take to enhance their quality of life is quitting (USDHHS, 1990). Smoking exposes the body to more than 4000 chemicals, more than 100 of which are carcinogens and mutagens (Hecht, 2002; Shields, 2002). Yet despite the well documented consequences of smoking, despite the unequivocal benefits of quitting, and despite facts such as that 1150 people die from smoking prematurely each day in America (Fiore et al, 2004), there were still 46.5 million adult smokers in the U.S. alone at the turn of the century (CDC, 2000). Surveys show that 70% of adult smokers and two thirds of adolescent smokers wish to quit, but while the majority do make an attempt, less than 5% are able to maintain abstinence for three or more months and only 2.5% are believed to quit permanently each year (CDC, 2002; CDC, 1994; CDC, 1993). With almost a quarter of the national population still smoking and the latency of cancer in the decades, the true costs have yet to unfold. It is an urgent public health need to help current smokers quit and, most importantly, discourage adolescents from uptake.

## 1.2. Addiction and cessation

Smoking is an addiction-driven behavior that is both a personal habit and a social phenomenon. Its difficulty of cessation can be explained in part by the addictive nature of nicotine in tobacco as well as by various psychosocial reinforcements (Baker et al., 2004). Genetics also plays a large role (Tyndale and Sellers, 2002). While nicotine consumption is the lone cause of addiction, evidence suggests there are many predictors of smoking perpetuation and cessation, including the smoking rate, genetic profile, sex, age, education, socioeconomic status, cigarette price, youth access laws, nicotine replacement therapy, parental smoking, and peer influences (Wetter et al., 2004; Gruber, 2003; Kobus, 2003; Ross and Chaloupka, 2003; Sellers et al., 2003; Lindström et al, 2002; Lu et al., 2001; West et al., 2001; Zhu et al., 1999; Osler and Prescott, 1998; Rose, et al., 1996; Chassin et al., 1996; Cohen et al., 1989).

## 1.3. Agent-based model

Agent-based modeling differs from the traditional study of equilibria in two ways. First, the two approaches differ in the theory. Where the traditional method looks at the static equilibrium of a social or economic problem, such as the market-clearing situation, the agent-based method focuses on the dynamics behind reaching the end result, such as the evolution of solutions, the aggregation of individual decisions on the whole, and the influence of social networks on behavior. Consider two competing gas stations at the same busy intersection. The equilibrium approach is useful for equating supply and demand to compute the market-clearing price and quantity that maximize utility and profit at each station. The agent-based approach, however, looks at how people behave and why prices change. It is able to incorporate such exogenous influences as product loyalty, peer influences, and social norms. The result may neither be market-clearing, optimal, or even desired, but is possible given the initial parameters. For example, both gas stations may claim equal market share in theory, but in reality, the taste of one's coffee may draw the majority of morning customers, building a reputation over time

that will eventually put the other out of business. The study of equilibria can yield solutions that are inaccurate in practice or fail to tell the whole story. Equilibria are only special cases within sets of all possible solutions, and could occur very infrequently or never at all, for perfect information and rationality are rare conditions. An equilibrium is merely an acknowledgment that there are adjustment processes (Schelling, 1978). Similarly, rationality is not a requirement of economic analysis (Arrow, 1986).

A second set of differences lies in practice. Where equilibrium analysis assumes that all agents in a specific population are homogeneous in tastes and utilities, agent based modeling allows for a large degree of heterogeneity among them. This is essential for studying such individualized behaviors as smoking because it allows each agent to have unique characteristics: age, sex, smoking frequency, etc. Differentiating each agent from the rest allows these factors to affect the dynamics in a realistic way.

After assigning a unique set of parameters for each agent, deriving solutions simply involves running the model repeatedly through a computer. Rather than solving a system of equations once to obtain an equilibrium solution, agent-based models can be executed countless times to show the entire spectrum of possible solutions (Axtell, 2000). Probabilities of occurrence for each solution can also be calculated.

The agent-based approach is especially useful in studying social groups. With networks comprised of heterogeneous agents who exert influence on each other and who make decisions on the micro level without regard to the aggregate, tracking the evolution of their behavior can provide surprising and novel outcomes. Social networks can act as multipliers to behavior as it diffuses from agent to agent, providing pathways of diffusion much like vectors in the spread of disease. As one chooses to smoke, a friend could easily adopt the behavior, and his or her friend could follow. Soon, a social identity may evolve from this common behavior, rendering smoking an acceptable activity. Of special interest are unexpected results that may surface as a result of the multiplicative property of social networks. One such type of outcome is stochastically stable behavioral or social norms, because their stability promotes permanence

and resistance to change (Young, 1998). In the case of smoking, it is wise to consider such types of results.

#### 1.4. Data

The agent-based smoking model was built to analyze data from a nested prospective cohort study of 1,961 six-grade students in 16 schools and 84 classrooms conducted by Valente and colleagues in Los Angeles County, California (Valente et al., 2003). It compared the effectiveness of three in-school anti-smoking intervention programs in changing the students' attitudes toward smoking, and concluded that peer leader groups were the most effective way to design the program, ahead of teacher assignment and random assignment. The study administered three surveys: a baseline survey of sex, ethnicity, etc., a precurriculum survey measuring attitudes toward smoking, and an identical post-curriculum survey three months after that. Social networks were constructed by asking each child to list his or her five best friends in the classroom, as well as five peers whom he or she believed would make the best peer leaders in a group project.

Taking the baseline characteristics and pre-curriculum attitudes of each child as exogenous variables, model simulations show the changing of smoking attitudes through the three-month intervention period as a function of the child's social network position, his network status (group leader, group follower, popular, lonely, etc.), and the smoking attitudes of his or her friends.<sup>1</sup> In all, the data provided the following empirical variables for each agent in the model: age, sex, five friends, five chosen group leaders, pre-program attitude towards smoking, and post-program attitude towards smoking.

But to specifically model smoking behavior, the model required a variable absent in the empirical data: a measure of nicotine addiction. It was needed to: (1) describe the reinforcing nature of smoking; (2) describe the difficulty of cessation; (3) establish an asymmetry between the probabilities of smoking continuation and that of cessation<sup>2</sup>; and (4) differentiate smoking

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<sup>1</sup> Attitudes toward smoking in this study were taken as an indirect measure of actual smoking behavior.

<sup>2</sup> It is postulated that peer influence alone could be enough to induce an adolescent to smoke, but peer influence alone is probably not always enough to cause the same adolescent to quit.

from other habitual behaviors or social fads, such as fashion trends, that the model could as well be describing with network variables alone.

## **PART 2. ADDICTION FUNCTION**

### **2.1 Definition of addiction**

Nicotine addiction, or nicotine dependence, can be qualitatively defined as the perpetuation of smoking despite the conscious desire to stop. Empirically, its evolution can be traced through the proliferation or decline of smoking intensity. Nicotine addiction directly correlates to smoking intensity, and is quantified in most epidemiological studies with an accepted scale.<sup>3</sup> Research on the validity of the scales show a consistent positive relationship between scores indicating nicotine addiction and actual smoking intensity, suggesting that addiction can be inferred from empirical data on the smoking rate. In the agent-based model, the probability of addiction is defined as the probability of choosing to smoke at time  $t_2$  having been a smoker throughout  $t_1$ , assuming that the agent would have chosen to not smoke at  $t_2$  had he been a nonsmoker at  $t_1$ . The first statement captures the time requirement of addiction—that addiction is the perpetuation of a behavior—while the latter assumption captures the defiance of rationality (it does not say that smokers are irrational, but rather they acknowledge the benefit of quitting but, compelled by addiction, continue to smoke anyway); that is to say, addiction has the property of continued smoking despite the desire to quit. Time periods are defined as iterations in the model, which can be days, weeks, months, etc. A good benchmark for the appropriate length of  $t_1$  is 183 days for boys and 21 days for the girls, which are reported in the studies used to construct the function as the median lengths of time between initial smoking and the onset of addiction symptoms.

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<sup>3</sup> Popular scales include the Fagerström Tolerance Questionnaire, the Diagnostic and Statistical Manual (DSM-IV) symptoms of nicotine dependence, and the Hooked On Nicotine Checklist (HONC).



## 2.2. Endogenous variables

Smokers become addicted to smoking because of the nicotine in cigarettes. While studies have disagreed on the quantity of nicotine from smoking required for addiction<sup>4</sup>, the causal relationship between nicotine and addiction has been proven (USDHHS 1988). The probability of addiction, denoted  $P(A)$ , is a function of nicotine intake and the genetic profile. Where social factors are strongly suggested to be the key determinant of smoking uptake, some pharamacogenetic studies suggest that the genetic profile is most influential in the actual development of addiction (Slama 2004). In other words, the likelihood of acquiring addiction is predisposed behaviorally and genetically. The derivation of the addiction function begins with this statement of causation.

$$P(A) = f(\text{nicotine intake, genetic predispositions}) \quad [1.1]$$

Empirically, the most accurate measures of nicotine intake are “how much” and “how often.” It is best written as a rate: quantity over time. Genetically, the most influential factors seem to be sex and ethnicity, both affecting the capacity for nicotine metabolism.

$$P(A) = f(\text{rate of nicotine intake, nicotine metabolism capacity}) \quad [1.2]$$

Nicotine intake can occur in many ways, only one of which is cigarette smoking. For cigarette smokers, the rate of nicotine intake is equal to the rate of cigarette smoking, assuming other forms of nicotine consumption are not practiced or not significant.<sup>5</sup> The rate of smoking is commonly measured in cigarettes per day (CPD).

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<sup>4</sup> It was previously believed that consistent regular smoking was required for addiction (USDHHS 1994). However, recent evidence suggests that addiction can develop within days or weeks of occasional use, prior to daily smoking (DiFranza et al., 2000).

<sup>5</sup> This assumption is justified by a physiological explanation. Cigarette smoking delivers the largest dose of free-basing nicotine to the brain over the shortest time period compared to any other product, providing maximum pleasure with minimum delay. Unless there is an outside incentive to choosing an alternative method of nicotine intake, a smoker will stick to cigarettes to receive the most “smoking utility.”

$$P(A) = f(\text{rate of smoking, nicotine metabolism capacity}) \quad [1.3]$$

How much and how often to smoke depend on many factors. Four in particular are well understood: (1) a physiological component governing nicotine absorption in the brain and the degree of subsequent euphoria, (2) a psychological component describing reinforcement from smoking and activities paired with smoking, (3) an economic variable adjusting demand to the price of cigarettes, and (4) a network component describing the effect of peer influences on smoking behavior.<sup>6</sup> Let  $R$  be the rate of smoking, and let  $G$  be the genetic predisposition.

Broadly:

$$R = g(\text{PHYSIOLOGY, PSYCHOLOGY, PRICE, PEER INFLUENCE}) \quad [1.4]$$

$$G = h(\text{GENETIC PROFILE}) \quad [1.5]$$

In detail:

$$R = g(\text{PHYSIOLOGY: rate of nicotine absorption in the brain,} \\ \text{PSYCHOLOGY: positive and negative reinforcements,} \\ \text{PRICE: elasticity of demand / price responsiveness,} \\ \text{PEER INFLUENCE: stature of agent i.e. leader/follower,} \\ \text{connectivity of agent, smoking behavior of others}) \quad [1.6]$$

$$G = h(\text{CHROMOSOME 23: the sex gene,} \\ \text{CHROMOSOME 19: the nicotine metabolism gene})$$

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<sup>6</sup> While the physiological absorption of nicotine and its psychological reinforcements are intrapersonal components of nicotine, peer influence and price are interpersonal. The varied nature of these components further captures the holistic property of addiction.

Thus, the probability of addiction can be summarized in the following expression.

$$P(A) = f(R,G) = f [ g (PH, PS, PR, PE), h (C23, C19) ] \quad [1.7]$$

### 3. Explanation of variables

#### 2.3.1. Physiology of addiction

Nicotine addiction follows a distinct pathway. The pharmacokinetics of nicotine, which refers to the mechanisms by which nicotine levels vary in different parts of the body over time, is heavily dependent on the route of administration (Baker et al., 2004b). Inhaled nicotine from cigarette smoke is the most addictive form of administration because about 80% is absorbed in the lungs (Armitage et al., 1975). This high absorption rate takes advantage of the large alveolar surface area in the lungs, from where it quickly enters pulmonary circulation and proceeds to the brain for further rapid absorption 12 to 15 seconds later (Le Houezec, 2003; Benowitz, 1994; Maziere et al., 1976). Other routes of nicotine administration, namely oral, nasal, and dermal, fail to deliver a similarly high dose to the brain, as they allow for greater initial distribution to other parts of the body (Baker et al., 2004b).

Following initial increases in brain and arterial blood nicotine levels, declines follow within the subsequent half hour as redistribution to skeletal muscle and other tissues occurs with an elimination half-life of two hours (Baker et al., 2004a). Prior to bodily elimination, nicotine is metabolized in the liver, lungs, and kidneys primarily into cotinine, a traditional biochemical marker of nicotine intake (Le Houezec, 2003). In the brain, nicotine binds to and activates  $\alpha 4\beta 2$  nicotinic acetylcholine receptors (nAChR) located throughout the central and periphery nervous systems (Camí and Farré, 2003). nAChRs serve as ligand-gated channels to release dopamine and  $\gamma$ -aminobutyric acid neurons into the ventral tegmental area and the nucleus accumbens, where they mediate both the subsequent rewarding and aversive psychological effects of nicotine (Laviolette and Kooy, 2004).

Accumulating a tolerance for smoking, namely a tolerance for the nausea, coughing, and dizziness associated with initial puffs, leads to increased smoking that eventually develops into a physical dependence (Leventhal and Cleary, 1980). Quitting while physically dependent causes withdrawal symptoms such as irritability, restlessness, concentration, impairment, increased appetite, and feeling miserable, which can last weeks or even months, as is the case with hunger (Jarvis, 2004). The intensity of these symptoms depends on one's level of nicotine dependence (Marlow and Stoller, 2003), and nicotine administered to withdrawn smokers consistently reduced sadness, anxiety, and irritability (Jorenby et al., 1996; Zinser et al., 1992).

Aside from motivating further nicotine self-administration (Piasecki et al., 2000), nicotine dependence also increases the threshold required for subsequent rewarding neural stimulation (Epping-Jordan et al., 1998), meaning that relapse usually occurs with even more intense smoking. Combined with intense cravings, withdrawal tends to thwart quit attempts, which helps to explain why most successful quitters attempt quitting many times before ultimately achieving the feat (Le Houezec, 2003).

### 2.3.2. Psychological reinforcement

There are two types of reinforcements, positive and negative. In short, the former is defined as the strengthening of a reward, and the latter is defined as the elimination of stress or punishment. Both are forms of classical conditioning that pair a stimulus, be it food or cigarettes, to a sensory outcome in a Pavlov's dog-like manner<sup>7</sup>. The most common positive reinforcement from smoking is the initial "buzz" or elation that derives, most intensely, from the first cigarette. This pleasurable response, when experienced over several cigarettes in a matter of days<sup>8</sup>, creates an inner psychological desire for its reproduction that drives further smoking. Negative reinforcement in smoking is characterized by the removal of depression,

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<sup>7</sup> This famous psychology experiment paired the sound of a bell to the sensation of eating for a dog, which resulted in the conditioned response of salivation when the bell was heard without food present.

<sup>8</sup> A quantitative justification of the cigarettes and time required for the onset of addiction has not been elucidated in the literature. For clues, a good place to begin is Cohen S, et al. "Debunking Myths About Self-Quitting." *American Psychologist* 1989; 44(11): 1355-65.

anxiety, and stress, often putting the smoker “at ease.” Studies with human subjects show that smoking reduces distress and depressed individuals are approximately twice as likely to be smokers. Although the directionality of causation is not certain, national studies in both the United States and Australia have found a high degree of co-morbidity between depression and smoking (Anda et al., 1990; Degenhardt and Hall, 2001; Kendler et al., 1993).

### 2.3.3. Pricing and demand

Substantial research in economics has established that aggregate propensities to smoke and cigarette excise taxes are inversely related, with the overall adult price elasticity <sup>9</sup>in the range of -0.3 to -0.5 (USDHHS, 2000). Moreover, it has been repeatedly shown that adolescents and young adults are much more price responsive than adults and therefore have greater price elasticities toward cigarettes. A recent study found that a ten percent increase in the price of cigarettes would increase the likelihood of young adult smoking cessation by 3.5% (Tauras, 2004). Economic data continues to strongly suggest that a significant increase in excise taxes across the United States would be an extremely effective means of reducing the social and economic burden of smoking.

In 1993, for example, the Philip Morris Company reduced the price of Marlboro cigarettes such that it more than offset the twice-raised Federal cigarette excise tax from the beginning of the 1990s, resulting in a decline in the real price of cigarettes by 10% and, consequently, higher prevalence of smoking (Ross and Chaloupka, 2003).

### 2.3.4. Peer influences

There is consensus in the literature around the idea that social group identity is a powerful force in the perpetuation of social norms, especially among youths. A smoker’s identity plays a direct role in explaining both current smoking behavior as well as the intention

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<sup>9</sup> Price elasticity is a measure of the flexibility of demand with respect to changing prices. A greater price elasticity corresponds to a greater flexibility to lower demand in times of higher prices. Necessities tend to have low price elasticities.

not to smoke (Falomir and Invernizzi, 1999). Social identity can be formed and subsequently molded in a number of ways. Various data sets on adolescent smoking networks show that smokers tend to associate with smokers and nonsmokers tend to associate with nonsmokers. Findings indicate that simply noticing other teens smoking is positively related to smoking behavior among adolescents across three different levels of smoking frequency (Eisenberg and Forster, 2003). Peer influences are most powerful on adolescents, with both self and group identity heavily shaped by peer affiliation (Jones et al., 2004). Moreover, adolescents with a strong smoker identity experience fewer quit attempts and become defensive when faced with antismoking messages (Jones et al., 2004). In all, friends who smoke develop a social culture around tobacco (Sargent and DiFranza, 2003), and behavioral norms other than smoking can also strengthen group unity, further reinforcing the smoker identity.

Loneliness—the lack of participation in social networks—can decrease the propensity of an agent to switch behaviors through a behavioral pathway as well. There is a body of literature on the relationship between social interactions and mental health that establishes a positive correlation between loneliness and depression. In a prospective cohort (longitudinal) study on risk factors associated with adolescent development of depression, Carbonell and colleagues (2002) found that the depressed group had a greater tendency to experience interpersonal problems such as loneliness or conflict with others, and that it is beneficial to have people to spend time with, suggesting that peer groups are a protective factor against depression. Various other studies have also identified, among others, positive peer interactions as a protective factor (Dumont and Provost 1999; Herman-Stahl and Peterson 1996).

#### 2.3.5. *The role of genetics*

The heritability of the risk for addiction has been estimated to be between 50-84%, and genes play an even larger role, 70-86%, in the intensity and perpetuation of smoking (Tyndale and Sellers, 2002). The protein CYP2A6, a polymorphic enzyme encoded on the 19th chromosome, is responsible for 80-90% of nicotine metabolism (Sellers et al., 2003; Tyndale and Sellers, 2002). Nicotine metabolism proceeds through the stepwise inactivation of nicotine into

cotinine, catalyzed by CYP2A6, and then from cotinine into trans-3'-hydroxycotinine. Seventeen allelic variants of CYP2A6 have been found, with different frequencies of appearance among different ethnic groups (Sellers et al., 2003; Xu et al., 2002).

Certain alleles enable carriers to metabolize nicotine quickly, while others delay the process. That different alleles exist across different ethnicities means there is genetic segregation of nicotine metabolism capacity, creating genetically predisposed potentials for the development of nicotine addiction. Different allelic variants can be acquired through mutations in the genetic sequence, which may introduce a risk factor or a protective factor into an individual's probability of developing nicotine addiction. This is achieved through the altered production of the CYP2A6 protein (Xu et al., 2002). Slower nicotine metabolism has been shown to decrease the risk of smoking initiation and dependence, decrease the amount smoked, and decrease the risk of tobacco-related cancers and mutations (Sellers et al., 2003).

Studies also show a difference between males and females, as well as between adolescents and adults in the development of nicotine addiction. Adolescents under the age of 19 are twice as likely to experience dependence and difficulty of quitting before smoking 100 cigarettes than those 19-22 years of age (Barker, 1994). This may be caused by the still developing adolescent brain, which is more vulnerable to nicotine. Girls also report symptoms of addiction much earlier than boys, and this is consistent with the data used to construct the addiction functions in section 2.5 (DiFranza et al., 2002a).

## **2.4 Constructing the function**

Finding a functional form for  $P(A) = f(R,G)$  requires data from epidemiological studies that were prospective or retrospective in nature and which contains a cohort of adolescents who fulfilled several conditions: (1) they smoked at a discernible rate, (2) they were not addicted to smoking at the beginning of the study period, but developed addiction by the end of the period, and (3) their genetic profiles were known. A review of currently published work revealed no

studies that collected data for all three conditions. However, several studies looked at the first two conditions, allowing for the construction of an addiction function based solely on R, the rate of smoking.

$$\underbrace{P(A) = f(R)}$$

Limited data  
for derivation

$$\underbrace{P(A) = f(R,G)}$$

No data available  
from the same study

Furthermore, studies that have looked at CYP2A6 in human subjects have been predominantly case control in design, bringing into question the influence of a variety of biases in the data, including crude phenotype assessments, undetermined co-morbidity, variable genotyping methodologies, and perhaps most importantly, recall bias. In fact, in a study involving the same McGill University study data used below, O'Loughlin and colleagues found that none of the allelic variants of CYP2A6 that code for slow nicotine metabolism actually protected subjects from developing addiction. On the contrary, those carrying one or two copies of the slow metabolism gene were actually at a substantially increased risk of addiction (O'Loughlin et al., 2004).

An important difference between the two variables R and G is that R is dynamic, whereas G is static. That is, the rate of smoking changes over time but an individual's genetic profile is like an exogenous variable. Mathematically, R should be the variable that determines the shape of the P(A) function, while G should act more like a coefficient, merely increasing or decreasing the function by a constant factor for each individual. Therefore,  $P(A) = f(R)$  should provide enough information for the shape of the curve.



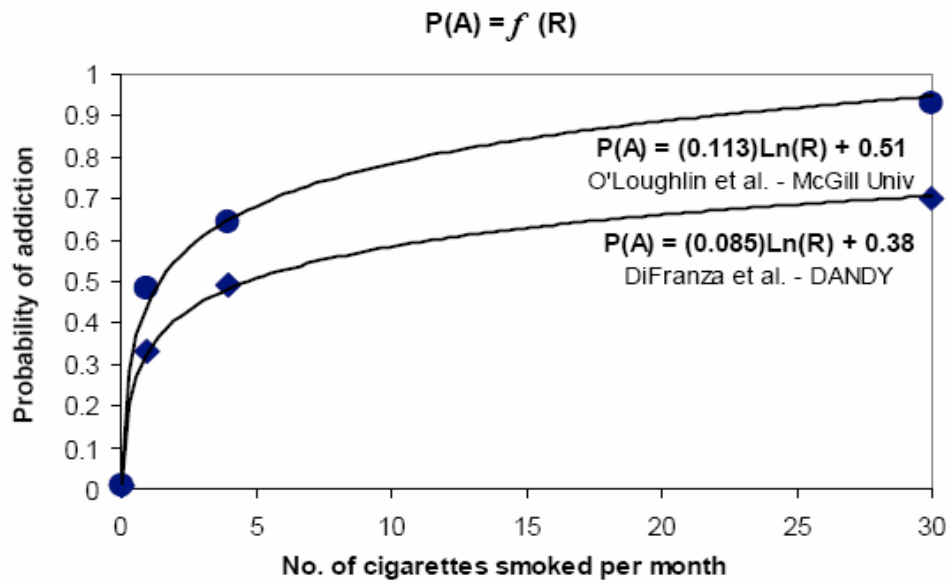
$$G = h (\text{GENETIC PROFILE}) = \text{constant "C"} \quad [1.8]$$

$$P(A) = f(R,G) = C f(R) \quad [1.9]$$

It is unknown whether R and G can work together to induce an interactive effect in influencing P(A). If they can, then the above equation 1.8 does not hold and a true P(A) function would require a prospective study that tracks both R and G simultaneously. In the next section, P(A) is derived as a function of R only with the assumption that it is an accurate description of the function's shape.

#### 2.4. P(A) = f(R)

DiFranza et al. (2002a) and O'Loughlin et al. (2003) were used to generate data for  $P(A) = f(R)$ , which is graphed in Figure 1. These two studies were chosen for their strong study design, use of accurate indicators for nicotine dependence symptoms, and wide range of data on rates of smoking. Study subjects were also similar across the two studies, which reduces the chances of confounding factors. However, the one consistency that allows them to be graphed on the same plane is that both studies used the same indicator for addiction: the Hooked on Nicotine Checklist (HONC). Two studies by DiFranza, O'Loughlin and colleagues on the HONC and other indicators determined that the HONC "demonstrated excellent psychometric properties" (O'Loughlin et al., 2002a) and "performed well psychometrically" (O'Loughlin et al., 2002b). The internal reliability for the HONC was 0.94 (DiFranza et al., 2002b).



**Figure 1.** Probability of addiction as a function of smoking rate. Girls achieved symptoms of addiction in a median of **21 days**. Boys achieved symptoms of addiction in a median of **183 days**.

Several features of addiction are apparent from the graph. First,  $P(A) = f(R)$  is logarithmic and asymptotes to a probability less than one. This means that even at very high smoking rates, there is still a probability that addiction will not develop in a smoker.

Second, nicotine is a non-threshold toxin, meaning there is no safe dose under which addiction symptoms will not develop. This is shown by the point (0,0) and is taken from DiFranza's conclusion that "There does not appear to be a minimum nicotine dose or duration of use as a prerequisite for [addiction] symptoms to appear." The graphs show that smoking just one or two cigarettes per month corresponds to a 0.3 to 0.5 probability of addiction. DiFranza et al. (2002a) also reports that the median smoking rate at the onset of addiction is two cigarettes, one day per week.

Third, the two graphs are similar, despite being derived from two different studies. Data in DiFranza et al. (2002a) came from the Development and Assessment of Nicotine Dependence in Youth (DANDY) study, conducted in central Massachusetts with a cohort of 679 seventh grade students for three years beginning in 1998. Data in O'Loughlin et al. (2003) comes from

the McGill University Study on the Natural History of Nicotine Dependence, a six-year project that began in 1999 with 1267 seventh grade students in 10 Montreal high schools. That the curves are similar despite these differences strengthens the hypothesis that  $P(A) = f(R)$  is actually shaped this way.

However, the discrepancy still needs explanation. It is likely not entirely due to random error alone, since one curve is uniformly displaced in the same direction from the other at all points. Rather it is likely explained by a systematic difference between the two studies, creating a multiplicative difference between the two curves. One possible explanation is sex. In the DANDY study, females represented 49% of the cohort, while in the McGill University study, 66% of study subjects were female. There is strong evidence that females are more prone to addiction than are males. In the same DANDY study, girls reported a much faster onset of addiction symptoms (median = 21 days) compared to boys (median = 183 days). Girls also reported more frequently on each of the 11 HONC symptoms of addiction, and reported an average of 2.9 symptoms versus 1.6 for boys among all subjects who had tried tobacco, and an average of 5.7 symptoms versus 4.0 for boys among all subjects who smoked monthly. In the same McGill University study, girls consistently scored higher than boys on most nicotine dependence indicators. Thus, the fact that the McGill University study sample had 34% more females than did the DANDY sample may explain why its curve asymptotes to a greater value along all smoking rates.

Because the Valente et al. (2003) data consisted of 50% females at baseline and 47.3% female after the baseline and curriculum surveys, the DANDY study curve should be used as the addiction function for the agent-based model.

## PART 3: CESSATION FUNCTION

### 3.1. Theory and derivation

Where the probability of acquiring addiction is determined by the rate of smoking alone, as nicotine consumption is the lone necessary and sufficient cause of addiction<sup>10</sup>, the probability of quitting smoking depends on a range of variables, only one of which is the current rate of smoking. In other words, where addiction is a one-way street, cessation has multiple pathways, and the most significant predictor for each individual agent differs based on each agent's unique set of characteristics (parameters in the code).

Known predictors of adolescent cessation include the rate of smoking (R), length of past quit attempts, parental smoking status, depression symptoms, sex, age, education, socioeconomic status, cigarette price, motives for quitting, conditioned behavior, access laws for minors, time spent with friends, and the use of nicotine replacement therapy (NRT) (Bottorff et al., 2004; Hughes et al., 2004; Jones et al, 2004; Buller et al., 2003; Horn et al., 2003; Klesges et al., 2003; Zhu et al., 2000; Zhu et al., 1999; Rose et al, 1996). As such, the probability of cessation is a function of R, G, and other variables.

$$P(C) = f(R, G, \text{age, sex, education, NRT, ...etc.}) \quad [2.1]$$

It is unclear which predictor of cessation is most important for adolescents. There are contradictions in the literature. For example, Zhu et al. (1999) found that there was no significant difference in cessation by sex, while Rose et al. (1996) showed that sex was a significant predictor of cessation. Given this uncertainty, however, almost every study, both on adults and adolescents, has found that the rate of smoking (R) is a strong predictor of cessation. Across entirely different study designs: different measurements of R, different addiction indicators, different study subjects, different time periods, etc., the rate of smoking has consistently predicted cessation with very few exceptions. From this consistency, it is likely that

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<sup>10</sup> Nicotine consumption is a necessary cause of addiction because one must consume nicotine to become addicted. It is also a sufficient cause of addiction because without nicotine, one cannot become addicted.

the R is the most important predictor for the probability of successful cessation. To isolate R, the other variables are combined into a noise term K.

K acknowledges the influence of other variables, although its value is unknown.

$$P(C) = f(R) + K \quad [2.2]$$

Since  $P(A)$  is a positive function of R,  $P(C)$  must be a negative function of R. This is because the probabilities of addiction and cessation must sum to 1, with noise. At each iteration of the model simulation, each smoking agent either continues smoking to the next iteration (on the addiction curve) or stops smoking at the next iteration (on the cessation curve). Each nonsmoking agent either continues to not smoke or starts to smoke.

In other words, each agent has a unique probability of addiction and cessation, but it must be one or the other. As such,

$$P(A) + P(C) = 1 \quad [2.3]$$

Solving for the cessation function,

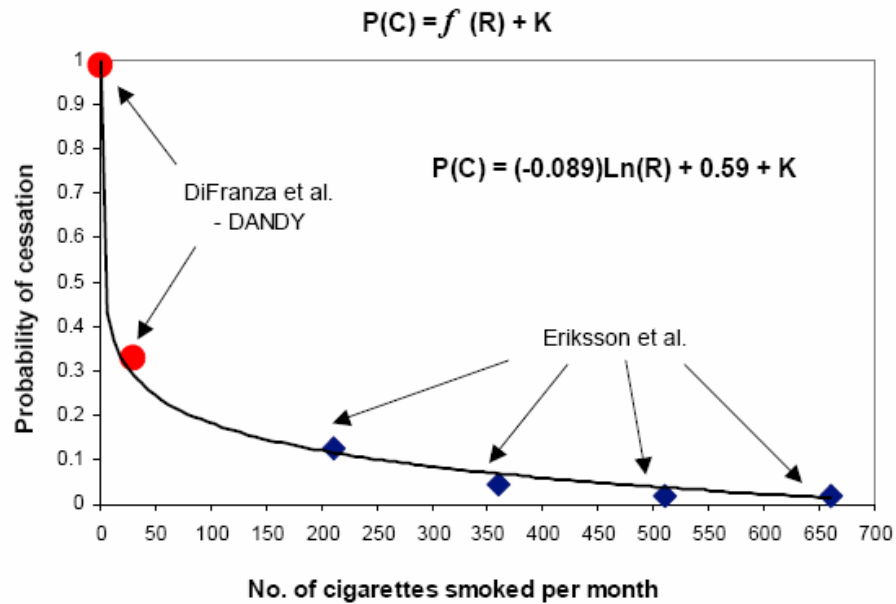
$$P(C) = 1 - P(A) \quad [2.4]$$

### 3.2. $P(C) = f(R) + K$

The cessation function was derived in two parts, because there was a shortage of data in the literature describing cessation as a function of smoking at a rate less than daily. Data for the first part, which describes cessation as a function of R from 0 to 30 cigarettes smoked per month, comes from the same DANDY study used to derive  $P(A) = f(R)$ . It is simply calculated from equation 2.3. Figure 2 shows this section of the curve. The curve in Figure 2 was plotted with no noise ( $K=0$ ), similar to the addiction function. Including K means that the more realistic curve is displaced uniformly in either direction. The second portion of the curve is simply an extension of the first, but the data comes from Eriksson et al. (1998), a study conducted in Norway on

pregnant women. Therefore, it should only be interpreted as a rough continuation of the first part. Figure 3 shows both portions of the curve combined, with common data points denoted as circles.

It is presented here only to give an idea of what the curve may look like as the rate of smoking increases up to more than 600 cigarettes per month. It should not be interpreted as a part of a generalized cessation function nor used for subjects in the model. The logic behind selecting this study to draw an extension of the curve is that if it falls in place with the first portion, it would strengthen the notion that the first portion is at least correctly shaped, and that the derivation makes sense. Moreover, it takes time to develop from a light smoker to a chronic one, so the fact that the first portion comes from a study on adolescents and the second from a study on young adults makes temporal sense. Only the DANDY portion of the curve, however, should be used in the agent-based model.



**Figure 3.** Probability of cessation as a function of smoking rate (0-660 CPM)

## PART 4: DISCUSSION

### 4.1. Application in the model

The addiction and cessation functions serve the same purpose in the agent-based model: to make the agents more realistic. They are gatekeepers in the evolution of agents between the smoking and nonsmoking states. Whenever the parameters of an agent are such that the agent is ready to swap states at the next iteration, the appropriate probability function intervenes to stop a portion of the swaps. Assume a nonsmoking agent in the Valente et al. (2002) data at baseline ( $t_0$ ). At  $t_1$ , the agent's peers in his social network, say one of his chosen group leaders, persuade him to try a cigarette. Will he then be a smoker at  $t_2$ ? The addiction function says there is a very low probability, since his rate of smoking at  $t_1$  is low. Chances are he will still be a nonsmoking agent at  $t_2$ , even though his peer influence parameters are such that he could be labeled a smoker otherwise. If nothing changes in his social network, that is, if his peer influence parameters continue to be such that his smoking rate increases at  $t_2, t_3, t_4, \dots$ , then his probability of becoming a smoker increases logarithmically at each iteration. Thus, each agent is affected by its social environment (surrounding agents). If the social environment is dynamic and evolving, then each agent's smoking behavior will be dynamic and evolving. If the social environment is static, then each agent's smoking behavior will only evolve according to the smoking rate function  $R = g(PH, PS, PR, PE)$ , which is still unknown but assumed to be dynamic.

Now assume that the agent is a smoker at  $t_5$ . If his social network continues to be smoking-friendly, then he will continue to be a smoker, as his smoking rate increases and his probability of addiction increases at each iteration. A function for this periodic growth in the smoking rate is still unknown. It is discussed in the following section 3.4. Suppose that after five more iterations of being a smoker, the agent experiences a change in his social environment, as several of his smoking friends move out of town. Now a majority of his network are non-smokers, and they persuade him to quit smoking. Will he be a non-smoker at  $t_{11}$ ? The cessation

function says that is unlikely, assuming his smoking rate is high at  $t_{10}$ . Although his peer influence parameters may be enough to persuade him to quit, his smoking rate acts against cessation with a probability  $P(C)$ . This is where the asymmetry between uptake and cessation comes alive in the model.

#### **4.2. Growth rate of R**

A growth rate of the smoking rate,  $dR/dt$ , is still needed for the model to describe the evolution of R for agents who continually smoke for many iterations. Such agents are expected to experience an increase in R, leading to a greater probability of addiction and continued smoking at each subsequent iteration.

There is a physiological and psychological explanation for this expected increase, and studies have shown that the majority of smokers who are addicted as adults began smoking during adolescence (USDHHS 1994).

However, the functional form of  $dR/dt$  depends on the equation R, which remains unknown at this point. Future studies should empirically test the components of R to build its functional form. The ability to solve for the first and second order conditions of an agent's rate of smoking at any given time will further improve the agent-based model. An interesting difference between the rate of smoking and the genetic profile is that G does not change over time,  $dG/dt=0$ . Thus, the probability of addiction is influenced by both R and G, but its evolution is mainly controlled by R.



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## II. RESEARCH PAPERS (2 OF 3)

“Assail my sense of personal control by telling me I cannot do something and I will want to do it all the more”

(Phares 1991, p. 473)

# Social influence, Reactance, and Tobacco

Ross Hammond

October 2005

Freedom is an important theme in psychology. Threats to freedom (actual or imagined), and the responses they evoke, are the central subjects of the theory of psychological reactance. Reactance can increase an individual’s tendency to engage in any behavior that is proscribed or portrayed negatively, sometimes leading public health campaigns and other attempts to influence behavior to “backfire”. This paper reviews the theoretical and empirical scientific literature on reactance theory, and discusses the implications of the theory for a social influence model of adolescent tobacco use.

### I. The theory of psychological reactance

The theory of psychological reactance was first articulated by Brehm (1966). Reactance, in Brehm’s theory, is a motivational state aroused whenever “a person’s behavioral freedom is reduced or threatened with reduction” (p. 2). Motivation is directed toward re-establishing the lost or threatened freedom, and the general effect of a reactance response is “to produce tendencies opposite the actual or threatened restrictions” (p. 13). Brehm’s research suggested

that reactance can be a hidden or even unconscious effect, and that a person in whom reactance is aroused might deny being motivated by a desire to restore freedom. Nonetheless, Brehm found reactance to have a strong effect on behavior—as external pressure to comply with a behavioral limitation increased, so did pressure *not* to comply (generated by reactance). Brehm found that “the resultant effect on the individual’s final response” was often difficult to predict.

Brehm found that “verbal appeals” to change behavior could also elicit reactance and “thereby minimize compliance” (p. 90). Subsequent research has built on this result, extending reactance theory beyond situations in which behavioral freedom is literally threatened, to apply to attempts to *influence* behavior through persuasion. Freedom to decide for oneself is in itself a widely valued freedom, and so any attempt to induce behavioral change “may meet with resistance, *even if the recipient of the effort agrees in essence with the communication*” (Fogarty 1997). A reactance response, pushing one away from the position being advocated, can be triggered whenever an “intent to persuade” is perceived and does not require “that the source of intent to influence is ill-motivated, reprehensible, hostile, or dissimilar” (Wicklund 1974, Brehm 1966).

The strength of responses to perceived threats (either to behavior or free choice) is, according to reactance theory, a function of three factors. The first of these is the perceived importance of the freedom that is threatened. If a freedom is irrelevant<sup>11</sup> to an individual, threats to it are less likely to elicit reactance from him or her. However, Brehm notes that reactance can cause a behavior to increase in perceived importance simply *because of* the threat to it (p. 11). The second factor affecting the strength of reactance is the proportion of the total set of available behaviors that is threatened—the more restrictive the threat appears, the more reactance it is expected to elicit. The third factor affecting reactance is the perceived “magnitude” of the threat. In the case of a social pressure to comply, the perceived magnitude of the threat will “center on the formal and informal relationships between the threatener and the person threatened... those who have equal or greater amount of social power than oneself can issue threats of relatively great magnitude” (Brehm, p. 7). If the source of persuasion has

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<sup>11</sup> Wicklund (2002, p. 5) gives “the freedom, for a *non-drinker*, to choose between 30 kinds of bourbon” as an example of an “irrelevant” freedom.

demonstrable expertise, this can also increase the perceived threat: “a communicator who is expert on the issue and who is trustworthy as well should have more power to threaten one’s freedom”. Two related factors Brehm expected to affect reactance were *justification* and *legitimacy*. If the threat to freedom is in some way seen as legitimate or well justified, this might moderate the reactance response. Yet Brehm argues forcefully that reactance responses do *not* require a perceived lack of legitimacy or justification to be potent, and that “a loss of freedom, no matter how well justified, should still create reactance” (p. 8).

One elicited, reactance can have several different effects. These may include: (1) increased desire for the threatened behavior (2) an increased feeling that this behavior is obtainable or accessible (3) a tendency to engage directly in the threatened behavior or to increase frequency of this behavior (4) a tendency to engage in any other behavior which “implies one could also engage in the threatened or eliminated behavior” and (5) a tendency to encourage any “equivalent” person<sup>12</sup> to engage in the threatened behavior (Brehm, p. 119). Reactance leads to an attempt to re-assert behavioral freedom, either directly or indirectly. Direct re-establishment means “engaging in that behavior which one has learned one cannot or should not engage in” (Brehm, p. 10) and resisting pressure to cease a behavior not just by continuing it but also by actively increasing the frequency with which one engages in it. This shift to an even more extreme position in response to attempts to change an attitude or behavior is known as a “boomerang” effect<sup>13</sup>. Indirect re-establishment of freedom means “performance of a behavior that is either more costly, dangerous, or taboo than the one threatened” and may also involve urging similar others to engage in the prohibited behavior even if one abstains one’s self (Brehm, p. 11).

Reactance, when elicited, is also believed to augment the social attraction of any threatened or eliminated freedom—giving it a “forbidden fruit” appeal, which “is not diminished by the comparative appeal of other freedoms” (Fogarty, p. 1279). Reactance to one

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<sup>12</sup> Equivalent in the eyes of the person reacting—e.g. a peer, a colleague, any one of equal status or similar position who may be seen to be susceptible to the same category of threat.

<sup>13</sup> Wicklund (1974) argues that although a “rational response [to resisting persuasion]... would be to maintain the original attitude—to show zero change”, reactance almost always produces a boomerang effect instead (4). In the psychology of young children, this effect is known more generally as ‘negativity’.



message can also influence response to other related behaviors: “the arousal of reactance may serve to sensitize an individual to additional threats, both to the same and other freedoms” (Fogarty, p. 1280).

## II. Empirical evidence about reactance

Numerous experimental studies have lent substantial empirical support to reactance theory, robustly reproducing the results that led Brehm to formulate his theory originally. Studies of public health communications and other attempts to change behavior or attitudes through persuasion also support the idea that reactance responses can undermine persuasive messages, and that the appeal of “forbidden fruit” can indeed perversely “help to sell the fruit” that is being warned against (see Atkin 2002, Burgoon et al. 2002, Fogarty 1997). As one group of researchers concluded in their study of reactance in teens: “if adolescents are told that they’re too young to perform a behavior, or simply warned not to do it, there’s always the chance that psychological reactance may lead to the opposite response” (Atkin, p. 39).

Empirical studies have also explored the link between reactance and several other known variables in individual psychology. The first of these is age. Perhaps not surprisingly, reactance responses appear to be especially likely (and strong) in adolescents, who as a group “are generally sensitive to issues of independence” (Burgoon et al. 2002). The relationship between levels of reactance and age is an inverse one (Hong et al. 1994)—as age increases, reactance responses tend to decline<sup>14</sup>.

Reactance has also been linked reliably with personality. Individuals differ from one another in the intensity of the value placed on freedoms of various sorts (Fogarty 1997), and a tendency for reactance responses appears to be “more of a characteristic of the person than of the situation” (Seeman 2003). Some research links standard psychological evaluative personality

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<sup>14</sup> Although reactance responses do not seem to differ across gender lines, there is evidence of a significant interaction effect between age and gender: reactance levels in women appear to decrease at a greater rate as age increased than did reactance levels in men (see Hong et al. 1994).

types to differing predictable degrees of reactance (see Dowd et al. 1994, Seeman 2003), making reactance especially likely in (for example) risk-takers. Other research argues that reactance itself is “an enduring personality trait” that can be measurable independently (Hong & Page 1989, for example develop a “Psychological Reactance Scale”), and may not be easily correlated with other personality metrics.

Reactance may also play a role in generating the “evaluative tension” that has recently been a focus of psychology research on decision-making and persuasion. In much research on persuasion (particularly in public health applications), successful changes in actual behavior are now thought to occur through a focus on *attitudes*, rather than simply *beliefs*.<sup>15</sup> However, empirical evidence suggests that attitudes can (and often do) contain both positive and negative elements simultaneously. In some cases, even very strong positive and negative elements can coexist (Priester 2002). Such attitudes are known as “ambivalence” (not the same as neutrality or disinterest), and lead to “evaluative tension” about the object of the attitude. This tension complicates the link between attitudinal change and behavioral change, and (in turn) makes policy assessment difficult since public health campaigns are often measured by success in changing attitudes. Thus “what looks like a successful outcome (an attitude changed in the desired direction) may not be associated [at all] with the desired behavioral changes” (Priester 2002, p. 151). Reactance can help increase “evaluative tension” by generating contrary responses to attitude shifts achieved through social persuasion, complicating attempts to assess the effectiveness of public health campaigns.

Reactance explanations have been applied convincingly in several empirical studies on public health. For example, there is substantial evidence of reactance effects in alcohol use among adolescents. The change in legal regulation of drinking that occurred in 1987 (when the federal government pressured states to raise drinking ages to 21) provided a particularly good test of the theory. Longitudinal studies done before and after the change show a substantial

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<sup>15</sup> Beliefs and attitudes are generally not considered to be equivalent concepts in psychology. Although many different formulations of these terms exist in psychology, ‘belief’ is often used to refer to information or knowledge that individuals take as simply true or false—e.g. facts. The term ‘attitude’, by contrast, generally carries a sense of positive or negative feelings about the object of the attitude. (see e.g. Cacioppo et al. 1997, Schwarz & Bohner 2001).

*increase* in abusive drinking behavior (especially in underage drinkers) *after* the law changed, and despite a background context of a gradual and continuous decline in drinking levels population-wide (see, for example, Engs et al. 1988, Allen et al. 1994). This is consistent with the idea of a “reactance” effect among the newly underage drinkers. Cross-cultural studies of drinking have also lent support to reactance theory. Cultures which consistently exhibit few drinking related problems tend to share certain legal and social characteristics: rather than forbidding underage drinking by law and promoting abstinence, alcohol “is seen neither as a poison nor a magic potent, there is little or no social pressure to drink, irresponsible behavior is never tolerated but young people learn at home from their parents and from other adults how to handle alcohol in a responsible manner” (Higgins & McCartney 1980). Drinking patterns and problems can often change through interaction with different cultural drinking norms (Engs & Hanson 1986; O'Brien & Chafetz 1982; Greeley, McCreedy, & Theisen, 1980). Although these conclusions are not without controversy (a recent widely publicized U.S. government study concluded that stricter U.S. alcohol laws had helped to reduce teen drinking), the scientific community has consistently reaffirmed conclusions consistent with reactance theory (see e.g. Hanson 2005).

Recent research in public health suggests that reactance can also play a role in drug use of other kinds, and especially in tobacco use among adolescents (see Burgoon et al. 2002). Designing public health strategies to reduce tobacco and drug use while minimizing reactance responses has been the focus of much of this recent work (see section IV below).

### **III. A model of social influence with networks and reactance**

A model developed by J. Epstein, R. Hammond, J.Parker and H.P. Young at the Center on Social and Economic Dynamics at the Brookings Institution<sup>16</sup> studies adolescent tobacco use

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<sup>16</sup> For a full description of the model and its results, see the technical power-point in *Social Influences and Smoking Behavior: Dynamics and Policy Implications* and the documentation provided in *User Manual: Agent-Based Smoking Policy Model*, both included in this report.

using a “social influence” model. This model can be used to help explore the implications of reactance theory for public health campaigns designed to curb smoking.

In the model, individuals make decisions about smoking based on a simple “utility” calculation. If smoking gives a positive utility to a particular individual  $i$  at time  $t$ , he/she will begin smoking (or continue smoking). If smoking gives negative or zero utility, he/she will not begin smoking (or will cease smoking). In other words, an individual’s decision in each time period  $t$  is:

If  $U_i(t) > 0$ , smoke. Otherwise, do not smoke.

The utility of smoking for each individual is based on three factors: physical, social, and psychological. *Physical* utility is based on a simple “addiction function”—the more cigarettes smoked recently, the greater the probability of addiction and of continuing to smoke. *Social* utility comes from “peer pressure”. The agents are connected to their peers in a weighted, directed social network (generated randomly according to parameters based on real data)<sup>17</sup>. Each connection signifies a flow of influence or “messages” from a friend, and the weight of the connection signifies the strength of influence. Individual decision-makers generate a weighted average of these social signals, which can be either pro-smoking or anti-smoking. Individuals also receive “messages” from outside their network (e.g. from public health campaigns, authorities, etc.) which are assumed to be anti-smoking. *Psychological* factors affect the impact of both types of message. Messages from friends are attenuated according to an individual’s susceptibility to peer pressure. Messages from outside the social network are influenced by individual characteristics including degree of skepticism about the message source, and *reactance level*.

Results from the model illustrate the particular role reactance can play in a networked social system. While overall network structure, starting conditions, and the distribution of other individual traits are held constant, anti-smoking “messages” are sent under three different “reactance” conditions:

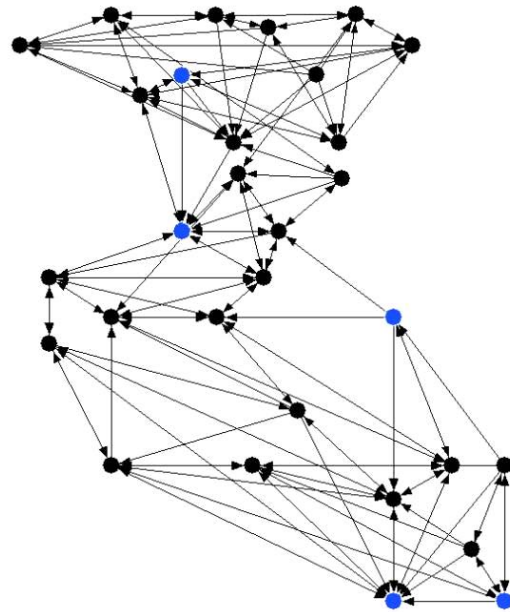
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<sup>17</sup> See “How to Form a Network of Junior High School Students” by Ben Klemens, included in this report.

### A. No reactance

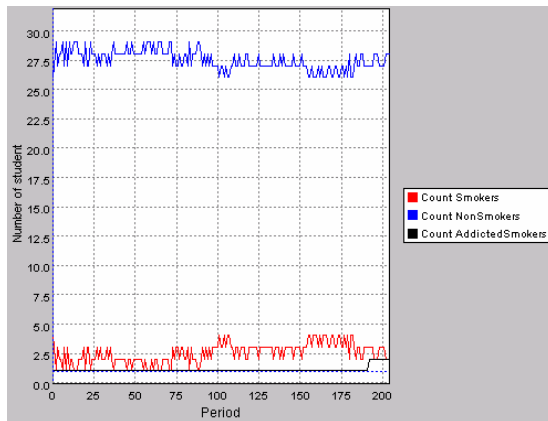
In this condition, strong, negative, and credible messages prove effective in reducing smoking. The extent and speed of the spread of non-smoking behavior through social networks is determined primarily by the structure of the “peer pressure” network. Weaker messages are less effective.

**Figure 1. A network with distributed reactance.** Dots represent individuals. Arrows represent social influence (more inward facing arrows means greater influence). Blue colored dots represent individuals with high reactance.



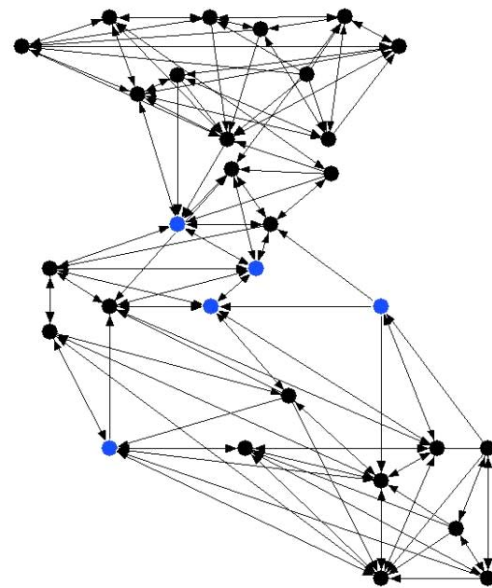
### B. Distributed reactance

When reactance is added to the model, strong negative messages (even highly credible ones) can become ineffective. If reactance is largely “distributed” among sparsely connected individuals in the network (as in Fig. 1 above), negative messages may simply have no effect. A sample simulation run illustrating this result is shown in Figure 2 below. At time period 100, a strong “anti-smoking” message is sent, but has little effect on the rate of smoking. Some individuals are swayed by the information in the message, but the message also activates a reactance response in others which offsets the effect.



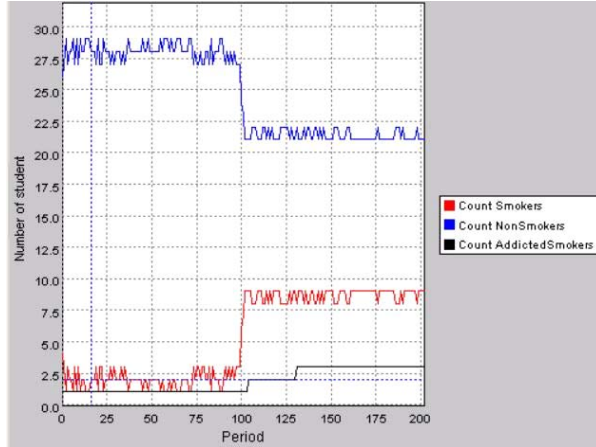
**Figure 2. A strong negative message has minimal effect with distributed reactance.** The x-axis represents time (each individual makes a decision about smoking in each time period). The y-axis shows the number of individuals (here, “students” in a classroom) who are smokers or non-smokers.

**Figure 3. A network with concentrated reactance.** As in Figure 1, dots represent individuals (blue color indicates high reactance) and arrows represent social influence (more inward facing arrows means greater influence).



### C. Concentrated reactance

If reactance is *concentrated* in the network (as in Fig. 3 above), strong negative messages can actually “backfire”. Figure 4 shows a simulation run comparable to the run in Figure 2. As before, a message is sent at time period 100. This time, however, the message does have a noticeable effect—in the opposite direction from the intended one.



**Figure 4. A strong negative message backfires with concentrated reactance.** The x-axis represents time (each individual makes a decision about smoking in each time period). The y-axis shows the number of individuals (here, “students” in a classroom) who are smokers or non-smokers.

Even if the credibility of the message is high, skepticism among recipients is low, and the starting condition is a very low number of smokers, concentrated reactance leads to a substantial *increase* in smoking after a strong anti-smoking message is received. Again, there are some individuals who might be persuaded by the message content and others who react against it. But this time, the reactant individuals are highly connected. Their social influence outweighs any persuasion the message might have on those less reactant than themselves. Thus, as reactance theory might predict, even individuals who (in isolation) might agree with the message content will end up responding in a manner contrary to that suggested by the message.

Although an extremely negative message can backfire when reactance is concentrated, the results of the simulations show that a more *moderate* message can be effective. Moderate negative messages can be strong enough to dissuade many potential smokers, while remaining weak enough to avoid triggering reactance responses. An important policy implication of taking reactance into account, then, is that the most extreme or vehement message may not be the most effective one.

#### IV. Reactance responses and message design

Empirical research on reactance in public health campaigns suggests that the *framing* and *content* of messages, in addition to their intensity, can be modified to reduce reactance responses. Reactance to persuasive or informative social messages can be minimized by carefully constructing the message. The empirical literature on this topic, largely driven by results from public health campaign experiments, has much to say about what types of messages to avoid (and what strategies have been successful) in changing undesirable behaviors or attitudes without eliciting reactance.

One experimental study is of particular relevance to the problem of cigarette use among adolescents. Burgoon et al. (2002) study the comparative impact of several different types of media messages on adolescent attitudes and behaviors regarding tobacco use. The goal of the research was to aid in “designing more effective tobacco prevention messages” by identifying “message design characteristics that avoid message failure... and boomerang effects” due to reactance. Informed by reactance theory, the experimenters tested “explicit” vs. “implicit” messages, and “controlling” vs. “non-controlling” message language. The study was conducted using a representative random sample of 924 students from 22 schools at three grade levels (4<sup>th</sup>, 7<sup>th</sup>, 10<sup>th</sup>), from a wide variety of socioeconomic and ethnic backgrounds. In addition to various message types, three message orientations were tested: messages for tobacco use, against tobacco use, and against tobacco use but with “restoration” at the end of the message. The “restoration” consisted of a separate clip designed to “restore a participant’s threatened freedom of choice by explicitly stating that any decision to be made in regard to tobacco use is solely up to the viewer” (76). Findings from the study included:

- *Explicit* messages (especially those that used “threatening or controlling language”) elicited cognitive processing that “resulted in higher levels of psychological reactance, and consequent message rejection” (77).



- Study participants who received explicit messages “felt controlled and manipulated regardless of the position being advocated” and explicit messages caused the message source to be perceived as less trustworthy.
- *Implicit* messages that “left conclusions up to message receivers” resulted more reliably in negative attitudes toward smoking than explicit messages did (again *regardless of the position being advocated*).
- Tobacco prevention campaigns that employ explicit messages can backfire and “actually elicit more *positive* attitudes toward tobacco use, particularly among high risk groups” (77).
- Because “a strong need for autonomy and self-expression” is a particularly important part of adolescence, persuasive messages targeting adolescents should be “complex and information rich” and “designed to emphasize choice rather than control”.

More general conclusions from a variety of literature suggest the following types of messages should be avoided:

1. ***Unconditional, overly threatening, or overly dogmatic messages***

Reactance theory predicts that the more absolute or unconditional any messages designed to discourage a behavior appear to be, the more likely such messages will trigger a strong reactance response. In studies of alcohol use, for example, evidence has shown that promoting abstinence can often be *less* effective than promoting moderation (e.g. policies known as “harm reduction”). Messages of unconditional abstinence may be especially likely to backfire when it is likely that recipients will have at least a small amount of exposure to the behavior anyway (Albarracin et al. 2003). Highly threatening or fear-inducing messages can also backfire by either seeming exaggerated

(undermining the perceived legitimacy of the source) or by desensitizing the audience to harmful outcomes (Atkin 2002).

2. *Message portraying behavior as “forbidden” or age-inappropriate*

Adolescents who are told they are too young for a behavior or that it is simply forbidden or off-limits are likely to experience a strong reactance response to such a perceived threat to their freedom. Portraying a proscribed behavior as “risky” can also increase its appeal to those in the audience who have risk-taker personalities (Atkin 2002).

3. *Messages containing statistics about or detailed portrayals of misbehavers*

When prevalence statistics (intended to be alarming) or portrayals of misbehavers such as celebrities are given in the message, these may create “inadvertent social norming” and thus normalize unhealthy behaviors (Atkin 2002).

4. *“Explicit” messages*

Explicit messages are those that directly and self-consciously attempt to influence, persuade, control, or threaten.

The following strategies, by contrast, have been found effective in successfully changing attitudes or behaviors with minimal reactance responses:

1. *Targeted audience selection*

Several studies suggest that individuals differ in their predisposition to reactance responses, in their response generally to various types of messages, and in their social connectivity (Atkin, 2002). For all of these reasons, selecting a particular audience to target with a given message can help make the message more effective.

## 2. *Indirect influence (via linked attitudes)*

As reactance theory demonstrates, a “frontal attack” on a given attitude is not always the best approach (Moscovici 1985). Indirect influence can help avoid reactance by targeting “attitudes related to the issue under persuasive attack, but not identical to it” (Atkin 2002, Perez & Mugny 1987). A substantial body of psychology literature suggests that attitudes can often turn out to be powerfully linked even when they don’t appear to be, and even when influence targets themselves are unaware of any relationship (see Schwarz & Bohner 2001). Such linkages can be well mapped through research, allowing persuasion to take an “indirect form”.

## 3. *“Implicit” messages*

Implicit messages often convey points by illustration rather than by assertion, and are structured to avoid seeming controlling (see Burgoon et al. 2002).

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II. RESEARCH PAPERS (3 OF 3)

## How to Form a Network of Junior High School Students

*Ben Klemens*

**January 2006**

### **Abstract**

This paper describes a method for producing artificial networks whose characteristics match those of the networks of friends observed in actual junior high school classrooms in L.A. The networks used to calibrate this network generation model are also used to compare various competing methods of describing link density in networks; the Gamma distribution has the highest likelihood among the density functions tested.























### **III. Agent Based Models (1 of 2)**



## *Smoking Model Users' Manual*

Jon Parker

### INTRODUCTION

The goal of this model is to study the spread of smoking among young teenagers. In this model we account for many factors that can influence a teenager's decision to smoke. Among them are:

- Peer Pressure
- Health concerns
- The "forbidden fruit" effect

Once teenagers have some smoking experience they also consider the pleasure that smoking brings (if any). The model simulates each teenager making this decision on a regular basis. Over time an epidemic of smoking may or may not spread depending on various factors.

If teenagers ever decide to stop smoking they may not be able to follow through with that decision. Once a teenager's ability to quit smoking is very small they are considered addicts. For more information on the addiction process, refer to "Addiction and Cessation Functions in the Agent-Based Smoking Model" by Zirui Song, included in this report.

### PREPARING TO RUN THE MODEL

This model is written in JAVA. It has been distributed as a .jar file. A .jar file is an executable package of JAVA files. Since JAVA is designed to be platform independent, you should be able to run this model on any computer. To determine if your computer is ready to run this model is to double-click on the "SmokingModel.jar" file. If the model does not start you will need to install a JAVA runtime engine. The runtime engine can be found at <http://www.java.com/en/download/manual.jsp>. Just download and install the appropriate engine for your operating system. Once the installation is complete you should be able to start the model by double-clicking on the "SmokingModel.jar" file.

## CREATING A MODEL

Models exist in two separate stages. The first stage is a “graph”. The second stage is a “classroom”. There is more information on both of these stages below. To create a new simulation, click on “Create New Graph”. You can find this option under the “Load and Create” menu. You will be asked to provide the number of teenagers that will be in the simulated classroom. This is the only step required to create a graph.

You may save a model at any time. One good place to save a model is right before you promote it to a classroom. This way you can create several classrooms with an identical network of friends.

## THE GRAPH STATE

When the model is in the graph state every person is a vertex in the graph. And every friendship is represented by a directed edge between two vertices. You are not given the opportunity to manually adjust the friendship network because the network is created using the method described in “How to Form a Network of Junior High School Students” by Ben Klemens included in this report. This method faithfully reproduces the survey data.

When a model is in the graph state it is your only opportunity to:

- Adjust the size of the vertices
- Determine if the vertices should be numbered
- Adjust the placement of the vertices

Three ways to arrange the vertices are given. You can manually arrange them. They can be automatically assigned to the perimeter of a circle. Or they can be arranged to reduce edge length. If you choose to use the last method a “redraw” button is provided because sometimes the vertices are clustered too close together, and other times the vertices separate into cliques that are not difficult to discern.

To promote the graph to a classroom click the “Confirm Promotion” button under the “Promote to class” menu.

## THE CLASSROOM STATE

Once a graph is promoted to a classroom the vertices cease to be vertices with edges, but are now virtual children with friendships. Now is the time to determine the “human” qualities of the agents. To set these qualities, pause the model, and check the “edit agents” box. Now, when you click on a child you can edit his properties. Here is a list of questions that the child’s properties are meant to answer:

- How strong is each of the teenager’s friendships?
- Has this teenager smoked in the past?
- Is this teenager currently a smoker?
- If the teenager has smoked in the past, how many days is it since his last cigarette?
- How much nicotine is in this teenager system?
- How strong does this teenager want to emulate his friends?
- How much does this teenager care about his health?
- Biologically speaking, how susceptible is this teenager to nicotine addiction?
- How skeptical is this teenager of any health message he’s received?
- How strong is this teenager’s will power when quitting?
- Will this teenager intentionally rebel against a health message?

As you can see, each agent has many facets. It is not necessary to set each property manually. Some of the properties can be set in batches by clicking the “edit all agents” button when the “edit agents” check box is checked.

You can also adjust agents by “intervening.” When you click on the “intervene” button you are presented with 3 choices. Each choice is meant to simulate an in-class anti-smoking program. You can increase students’ concern for their health. You can reduce students’ concern for smoking related peer-pressure. You can also change how strongly the “anti-smoking” message is presented. Remember that reactant agents will actually be influenced to smoke as the strength of the anti-smoking message increases.

### RUNNING THE MODEL

Once the agents have the properties you desire you are ready to run the model. Just click the “run” button. Be sure that the “edit agents” box is not checked. The model won’t run if that box is checked. Once the model starts running a chart will appear. This chart initially just shows the number of teenagers that are smoking. However, the chart can show the following variables:

- The number of smokers
- The number of non-smokers
- The number of social smokers (teenagers who receive more peer pressure to smoke than not smoke)
- The number of people that are “happy” (happy people are successfully doing what that want to. Consequently only addicted smokers can be unhappy)
- The number of addicted smokers. These are children that want to quit smoking, but can’t due to addiction.



### **III. AGENT BASED MODELS (2 OF 2)**



*An Agent-Based Computational Model of Smoking Behavior  
Among Adolescents due to Peer Effects:  
Coordination with Addiction in Social Networks*

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**Abstract**

Adolescent smoking behavior is described as a coordination game played by purposive individuals within social networks. Payoffs to coordinated plays of the game change over time to reflect addiction. This model is a variant of a more general stream of research involving coordination games played adaptively by agents in social networks. The overall population consists of such agents as well as agents who behave in other ways (e.g., fixed behavior or random behavior). Thus we have truly heterogeneous agents. The literature on this subject is well represented by the abstract models of Young (1999), the model of retirement behavior of Axtell and Epstein (1999), along with a model of crime due to Glaeser, Sacerdote and Scheinkman (1996).

JEL keywords: coordination game, social networks, addiction, smoking, peer effects

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## I. Introduction: Coordination with Addiction

There are long literatures on the adoption of smoking behavior among youth, from physiological, sociological and rational economic perspectives. In lieu of surveying these research streams here, we instead pick and choose those aspects of them that seem the most relevant for our purposes—building an agent-based computational model of peer effects mediated by social networks. From sociological work we adopt the notion that the actions of people in one's social network can have a significant influence on behavior. From physiological research we utilize the idea that the more one smokes the more difficult it is to quit smoking, and having quit, the easier it is to become a smoker anew. That is, the physiology of addiction is something we model explicitly. Finally, from the theory of rational choice we treat the concept of *purposiveness* explicitly, directing our agents to choose the behavior that yields for them the highest immediate reward.

The summary of all this is that we model the adoption of smoking behavior as a process of imitation in social networks, where the larger the number of peers an agent has who smoke, the more likely it is a person will take-up smoking. Furthermore, the more one smokes the more difficult it is for the agent to quit and the easier it is for an agent who has quit to recommence smoking.

In the next section the data we use to estimate the model is described in detail. It turns out that data exist from high school surveys concerning (a) attitudes about smoking and (b) self-reported social networks, of friends and of leaders. We utilize the latter in construction of the model and the former in estimating the parameters of the model, and each is the subject of its own section below. Once the model is built in this way we then exercise it to study the effects of various perturbations on its main output statistic—the proportion of agents who smoke at any instant in time.

It turns out that the model output is quite dynamic, with periods of rapid increases in smoking followed by periods of rapid decline, with few epochs of relative stasis. The intrinsic dynamism of the model seems to be characteristic of the range of parameters estimated for the

model, and suggests difficulties interpreting annual rises or declines in actual smoking data in terms of underlying fundamentals. That is, the reasons for annual changes may be intrinsically stochastic, without simple, causal, deterministic explanations, and therefore, not facilitating easy, explicit policy intervention. In this the model, output qualitatively resembles the fluctuations one sees in complex adaptive systems of other, various kinds (e.g, crime rates, traffic congestion, certain financial time series, and so on). The addictive property of smoking behavior appears to be largely responsible for the high variability in output.

## II. The Data

A unique dataset from Los Angeles schools serves as the basis for the empirical aspects of this model. Specifically these data are comprised of multiple waves of survey data in which adolescent-age students self-report many things, including social networks and attitudes toward smoking. Some of the statistical structure of these data are described in the next section, as input to the model, e.g., social network topology. Other features of these data are used to estimate the model, e.g., attitudes toward smoking.

Much of the empirical work done to date on youth smoking adoption has not considered peer effects (e.g., Jonathan Gruber, 2000), focusing instead on conventional economic variables like prices. Other studies do consider peer effects, but do not consider social networks. It appears that the data we have for this study gives us the possibility to include social networks for the first time.

## III. Model Structure

The principal components of the model are agents, their social networks, and their behavior. We describe each of these in turn.

### Agent Behavior

There is a population of agents arranged into classrooms. We model the adoption of smoking as follows. Each agent has a social network in which some fraction,  $f$ , of its



members are smokers;  $f \in [0, 1]$ . Each agent also has an internal *peer pressure* threshold,  $\theta$ , meaning that if  $f > \theta$  then the agent becomes a smoker. If at any subsequent time  $f$  falls to below  $\theta$  then the agent quits smoking. Agents with  $\theta \sim 0$  are early adopters of smoking, while those with  $\theta \sim 1$  will essentially never adopt. The distribution of  $\theta$  in the population determines the proportions of these early and late smokers.

We can formulate this notion of thresholds through the relative payoffs that accrue to the distinct behaviors of either smoking or not smoking. To see this, write payoffs associated with these activities as

	Not smoking	Smoking
Not smoking	$a, a$	$0, 0$
Smoking	$0, 0$	$b, b$

**Table 1:** Payoff interpretation of threshold behavior

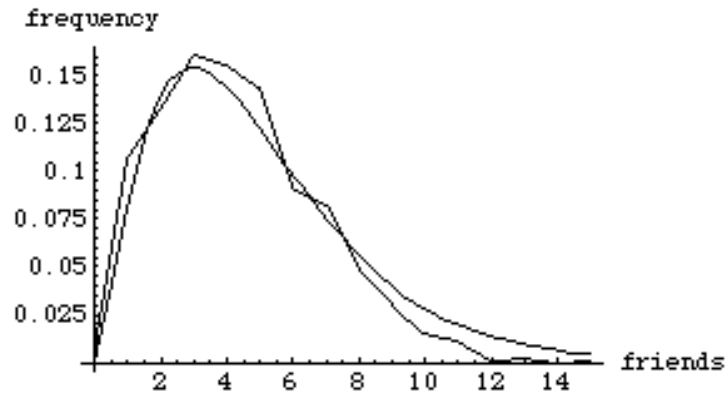
The payoffs to not smoking and smoking, respectively, to an agent having  $f$  of its friends smokers, is therefore  $b(1-f)$  and  $af$ . We treat agents as purposive, and therefore the behavior is chosen that has the highest payoff. Setting these payoffs to be equal and solving for  $f$  gives that agents will smoke whenever  $f > b/(a+b)$ . This means that  $\theta$  can be written as  $b/(a+b)$ . With some small probability  $p > 0$ , agents chose their behavior randomly.

Each agent updates its behavior once per period, asynchronously; eventually we estimate the duration of the period, e.g., monthly or annually, depending on data frequency. Within each period each agent is activated in random order.

**Agent Social Networks**

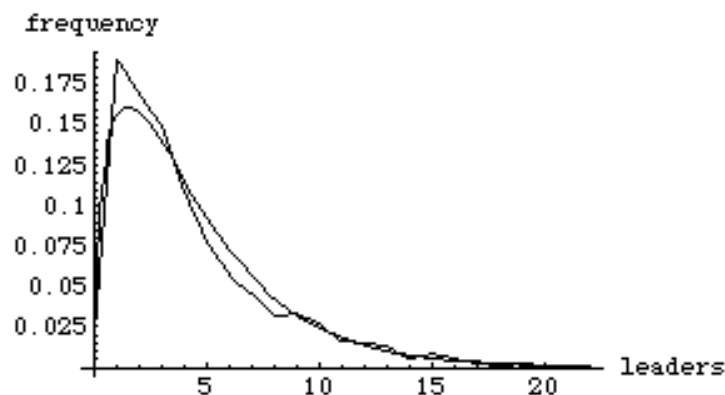
Agents are situated in classrooms of 20-30 students, and interact primarily with other students within their classrooms. Some interaction between agents in different

classrooms occurs, as does interaction across grades. Agent social networks are random graphs, based on analysis of data we have on Los Angeles classrooms. There are two kinds of social networks operational, one involving friends and another so-called leaders. The degree distribution of each of these is shown in the following two figures by irregular lines; moment-based fits using the gamma-Erlang distribution are shown as smooth lines.



**Figure 1:** Gamma-Erlang degree distribution of the *friends* network; mean is 4.0; variance is 2.5

The mean number of friends is just over 4, with a standard deviation of nearly 2.5. The leader network is somewhat more skew.



**Figure 2:** Gamma-Erlang degree distribution of the *leaders* network; mean is 4.5; variance is 3.6

Here the average leader is identified as such by about 4.5 students, with a standard deviation of 3.6. In our model either one or both of these networks are operational during any run. The CDF for the gamma-Erlang distribution is

$$\Pr[X \leq x] \equiv F_X(x) = 1 - k \exp\left(-\frac{x}{k}\right)$$

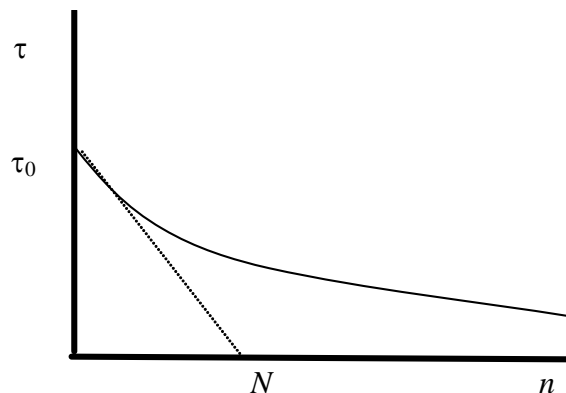
which is convenient for creating artificial social networks using the so-called ‘inverse method’ (Averill M. Law and W.D. Kelton, 2000).

### Agent Physiology

We model the physiology of addiction as the monotonic reduction of  $\tau$  with the amount of smoking,  $n$ . Once agents begin smoking they become progressively addicted—quitting is evermore difficult as a function of past smoking, while smokers who have quit find it difficult to refrain from smoking. This is modeled as an exponential decline of  $\tau$  with  $n$ :

$$\tau(n) = \tau_0 \exp\left(-\frac{n}{N}\right)$$

where  $\tau_0$  is the agent’s initial threshold of adoption and  $N$  is the amount smoking the agent has to do in order for its effective threshold to be reduced by a constant fraction. This is shown graphically in the following figure.



**Figure 3:** Exponential decline of smoking threshold with quantity smoked

In the limit of large  $n$ , i.e., a large amount of smoking, the agent's threshold falls to 0 and it becomes both very difficult to quit and, if the agent has quit, very easy to restart smoking.

#### IV. Typical Model Output Regimes

A typical run of the model unfolds with agents aging and moving through the school system. Older agents are removed while new agents are added. The main output from this model is a time series for the fraction of agents smoking. The following classes of social behavior have been observed, as a function of the model parameterization:

1. Low levels of smoking, isolated to low threshold agents, primarily when there are few of these kinds of agents and average thresholds are high with little variance; in such circumstances there exists a well-established social norm of little smoking;
2. High levels of smoking when there are either a relatively large number of low threshold agents or high variance in thresholds; such circumstances are characterized by social norms in which smoking predominates;
3. Intermediate levels of smoking that constantly change, typically produced by heterogeneous agent thresholds; this is usually accompanied by quasi-regular oscillations in the overall smoking rate, as shown in Figure 4 below, although for certain parameters the fluctuations are sufficiently erratic as to appear incoherent.

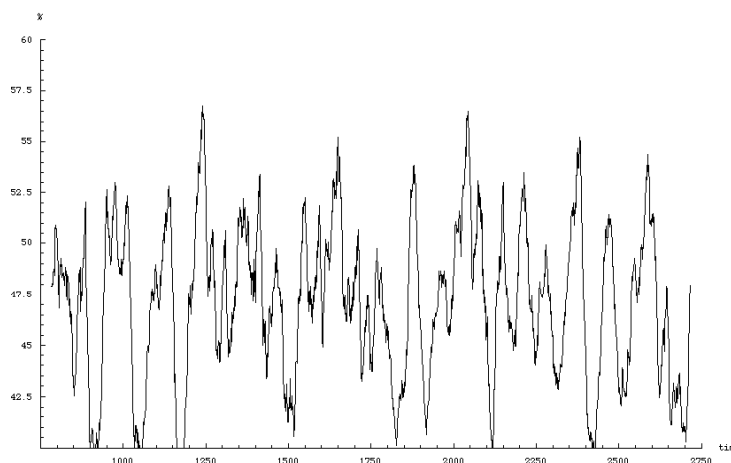
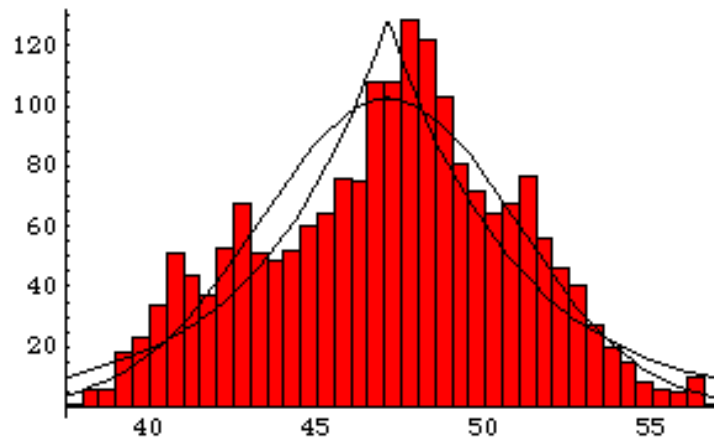


Figure 4: Typical smoking rate time series

By visualizing a large number of these models runs, for wide values of parameters, we have empirically observed certain regularities that obtain over a variety of these regimes:

1. In those cases involving a relative high level of smoking and the establishment of a quasi-social norm for smoking, there is a definite sense in which the younger grades are led into smoking by older kids;
2. When such social norms unravel it is less often that older kids give up smoking. Rather, a 'clean' grade of kids comes along who shun the older kids (stochastically), thus breaking the high smoking social norm and possibly establishing a low smoking norm, although this not a necessary consequence;
3. The transition from a low smoking norm to a high smoking one is due to the adoption of smoking at high rates by younger grades;
4. Increasing the rate of addiction makes the system behave in a more 'lumpy' way; alternatively, decreasing the rate of addiction makes the dynamics 'smoother' and reduces the magnitude of fluctuations (volatility);
5. The structure of the social network is crucial to the overall qualitative dynamics.

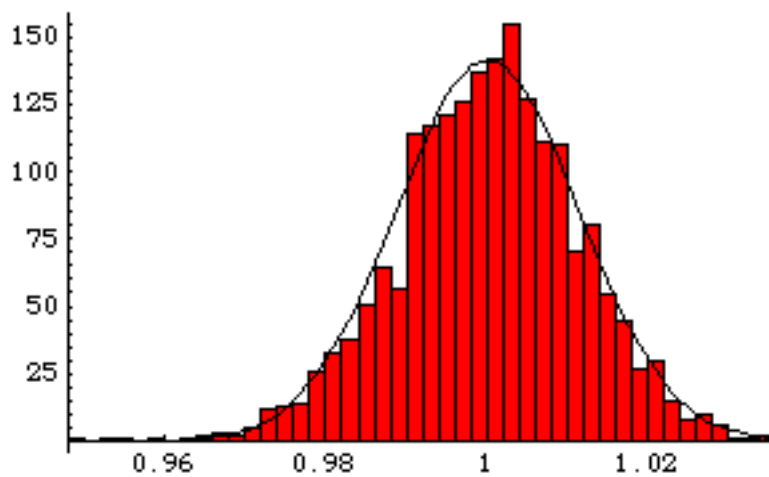
A different way to look at these time series data is as a histogram of smoking adoption rates. A typical distribution of this type is shown in Figure 5. These data are approximately unimodal. Fits of both the normal and Laplace distributions are superimposed.



**Figure 5:** Distribution of smoking adoption rates

Notice that this distribution has more mass at the mean, less in the ‘shoulders’ than the normal. Neither distribution well fits these data and we suspect that an intermediate form, e.g., the Subbotin distribution, would be a better way to describe these data.

A closely-related statistic is the distribution of *changes* in average smoking rate from period to period. This is a kind of growth rate in smoking adoption. For the data from Figure 5, its growth rate distribution is shown in Figure 6.



**Figure 6:** Near normal distribution of growth rates

This distribution is approximately normal, with mean of unity and relatively small variance. Interestingly, as agent populations are increased, the variance in smoking growth rates stabilizes at a non-zero level, meaning that fluctuations are persistent in this model.

Data on smoking adoption in school populations has a similar statistical structure, although data of sufficient frequency is not available to make a direct comparison of distributions.

## V. Model Estimation

There are a variety of ways we can go about estimating this model. Because microdata are available—at least attitudinal data on individual agents—we can perform ‘estimation by simulation’. That is, we can develop parameter estimates for our model on the basis of these data by running the model many times and finding the parameters that generate the best fit to the data.

Abstractly, our agent-based model can be written in the following mathematical form, despite the fact that we don’t actually have the functional form explicitly:

$$y = g(x; \theta)$$

where  $y$  is vector of agent attitudes toward smoking,  $x$  is a vector of known agent attributes, and  $\theta$  is a vector of parameters (e.g., thresholds, addiction rate, agent activation parameters, and so on). Since we don’t have  $g$  explicitly, but the model is an implicit version of it, the parameter vector  $\theta$  can be estimated by ‘simulating’ the model, i.e., running it many times. A similar estimation effort in the context of an anthropologically-significant model is due to (Robert L. Axtell et al., 2002).

We have done this systematically, expending over one CPU month on simulations in order to come up with a model that well-describes the data. This model is instantiated in the QuickTime movie that accompanies this paper. In that visualization, agents are colored by their type and behavior. Blue agents are those being influenced by peer pressure, magenta agents are

early adopters of smoking behavior, yellow agents are not susceptible to peer pressure but can adopt smoking. Agents turn red when they adopt smoking.

## VI. Summary and Conclusions

It has been demonstrated that a model of coordination in social networks with addiction can lead to time-varying adoption rates that can be calibrated to have much in common with empirical data on smoking among adolescents.

The notion of addiction we use here is apparently novel in the game theoretic literature and substantially different from more conventional rational choice formulations (e.g., Becker and co-authors (1992, 1994, 1991, 1988)). Its connection to a coordination game with state-dependent payoffs has been described. We feel that this model enriches significantly conventional framings of addiction, retaining the purposiveness of individual agents but adding in a social component. Such social or peer effects are widely believed to play important roles in the initial adoption of addictive behaviors (Paul Slovic, 2001). There are also significant econometric estimates of such peer effects.

Our model is richer than standard econometric specifications, and therefore we have had to resort to 'estimation by simulation' in order to make the model empirically-relevant. This is a new approach to bringing agent-based models to bear on data, and we hope represents less the final word on this subject than the first foray into what seems to us a pregnant confluence of methodologies for public policy modeling.



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## Appendix: Code Base

This model lives as ~2K lines (including terse comments) of C++ with graphics specific to the Macintosh operating system, with binaries compiled for OS 9 (Classic).

In this code, each individual person is an agent object, and the collection of agents that constitutes a social network is also an object. Each classroom is also a container of objects that is itself an object.

Agents have heterogeneous thresholds and social networks. Each agent's threshold is given initially and then evolves with the amount of smoking performed by the agent. Agents are activated randomly, with the annual frequency of such activations an estimated parameter. Agents typically start and stop smoking multiple times over a multi-year realization of the model.

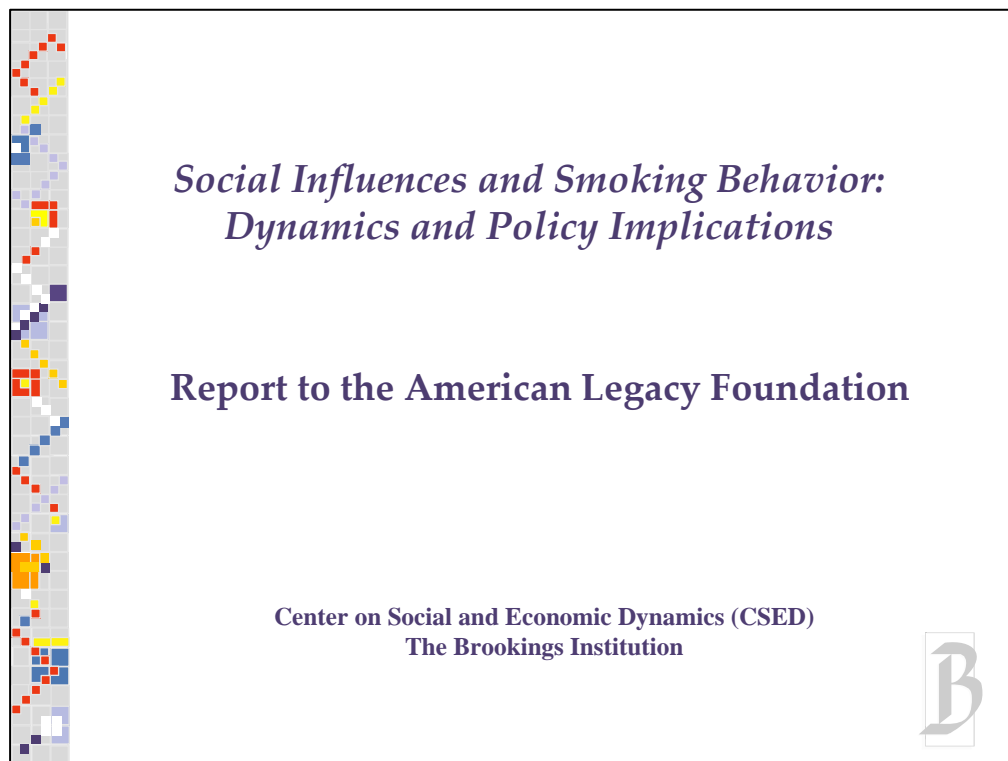
Agent social networks are fixed for the duration of a run of the model. They are random within a classroom, having gamma-Erlang distribution of degree as described above. Some agents have many more social connections than others. These networks are directed graphs, with social influence being exercised asymmetrically.


Agent classrooms have fixed membership for the duration of a realization. The classrooms have statistical sizes in accord with the data. Weak social influence extends beyond the classroom during a particular year— clearly a crude idealization— with classrooms are reconfigured randomly each year.

The model proceeds by initialization of all agents, social networks and classrooms. Then agents are activated for one period. Agents are then moved up one grade and the process repeats.

## VII. SOCIAL INFLUENCES AND SMOKING BEHAVIOR: DYNAMICS AND POLICY

**IMPLICATIONS** *Powerpoint Presentation to the American Legacy Foundation*







## Research Staff

**Robert Axtell**  
**Joshua Epstein**  
**Ross Hammond**  
**Ben Klemens**  
**Zirui Song**  
**Jon Parker**  
**Peyton Young**


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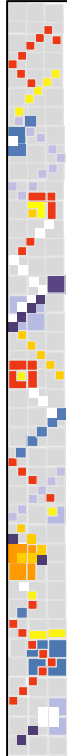


## CSED

- ❑ Pioneered agent-based models to analyze policy questions.
- ❑ Only group combining social, psychological, and economic effects into an integrated modeling framework
- ❑ Superior to static equilibrium approaches

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## Key features of the CSED approach

*Individuals are embedded in a dynamic framework where they influence each other and also respond differentially to information, advertising, and anti-smoking campaigns.*

- ❑ Models the position and connectedness of each agent in a social network
- ❑ Incorporates individual differences in predisposition to addiction
- ❑ Allows varied psychological responses to information and advertising

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## CSED approach (cont.)

- ❑ Other models focus on one or two factors in isolation (e.g., price effects)
- ❑ Our model highlights the interconnectedness of social, economic, and psychological effects.
- ❑ Allows for genuine heterogeneity in individual characteristics.


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## Outline of the presentation


- ❑ Social networks: friends and leaders  
*Ben Klemens*
- ❑ Physiology: factors leading to addiction  
*Zirui Song*
- ❑ Psychology: reactance theory  
*Josh Epstein and Ross Hammond*
- ❑ An empirical agent-based model of peer effects  
*Robert Axtell*
- ❑ An agent-based model for policy  
*Josh Epstein, Ross Hammond, and Jon Parker*
- ❑ Summary


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## Social networks: friends and leaders


- ❑ The USC data
  - Who are your five best friends?**
  - Who would be the best candidates to lead a class project?**
- ❑ Know network for each of 86 classrooms


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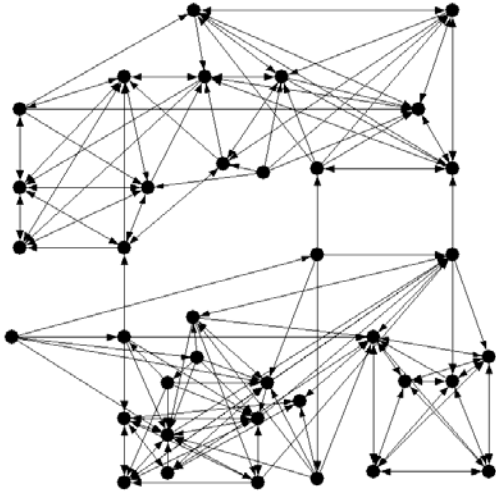
## Network characteristics


- ❑ Friend and leader networks have different structures
- ❑ Leader networks have a few superstars
- ❑ Friend networks are more homogeneous
- ❑ Both networks exhibit clustering of boys and girls

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## A network from the survey



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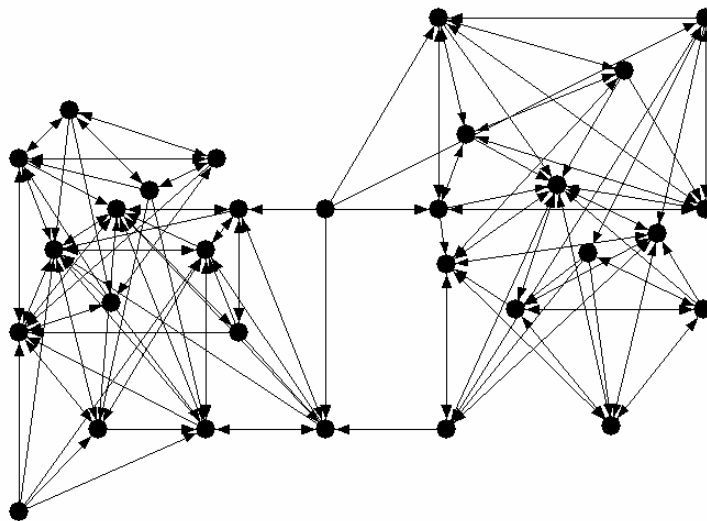
## The artificial network

- ❑ People with similar characteristics are more likely to be friends.
- ❑ Factor analysis determines the most relevant characteristics.
- ❑ Networks are generated by a probability model calibrated from the data.

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## An artificial network



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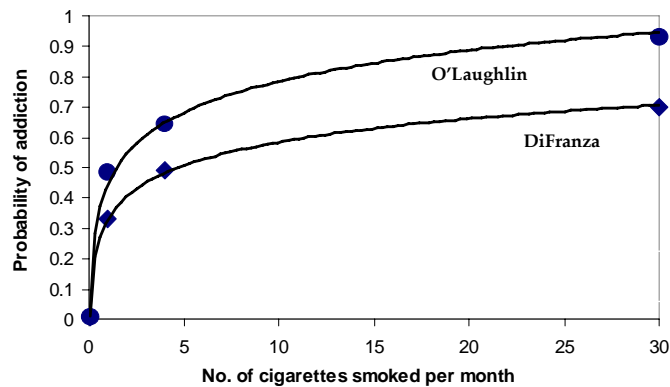


## Physiology: factors leading to addiction

$P(A)$ =probability of being addicted

- $P(A)$  depends on smoking rate, genetic predisposition, other factors
- Smoking is dynamic; genetic predisposition is fixed
- Data from two studies
  - DiFranza *et al.* (2002)
  - O’Laughlin *et al.* (2003)


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- Discrepancy likely due to male/female ratio: McGill 1:2, DANDY 1:1.
- Girls achieve symptoms of addiction in a median of 21 days.
- Boys achieve symptoms of addiction in a median of 183 days.

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## An Agent-Based Smoking Model

“As Simple as Possible, but no Simpler” *Einstein*

**We want a *simple but revealing* model of the decision to smoke or not.**

- **Simple**  
If  $U = \text{Utility}(\text{Smoking}) > 0$ , then Smoke;  
Otherwise Do Not Smoke.
- **Revealing**  
 $U = F(\text{Networks, Messages, Psychology, Biology})$


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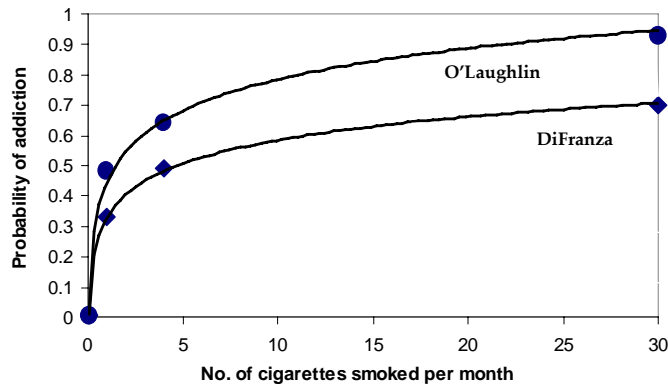
## Build Up decision function

- Individual Biology
  - Addiction Function
- Individual Psychology
  - Reactance
  - Skepticism
- Social Network(s)
  - Weighted
- Information
  - Messages

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## Individual Addiction Functions



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## Social networks

- What are my friends doing?

$$\text{social coefficient} * \sum_{i=1}^n \text{weight}_i * \text{friend}_i$$

- Previous model included ONLY this.

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## Messages and risk aversion

- ❑ **What message am I getting from “authorities?”**  
Normally  $[0,1]$ , no positive smoking message possible

$$message \in [-1,0]$$

- ❑ **To what degree do I believe it?**  
 $message * (1 - skepticism)$

- ❑ **How risk averse am I?**  
 $risk\ aversion * message * (1 - skepticism)$

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## Reactance


*“Assail my sense of personal control by telling me I cannot do something and I will want to do it all the more” (Phares, 1991)*

### Reactance generally causes:

- increased desire for proscribed behavior (“forbidden fruit”)
- increased tendency to try (or to increase frequency of) the behavior
- tendency to engage in even more extreme behavior
- tendency to persuade “peers” to engage in the behavior
- adoption of opposite/extreme view (“boomerang”)


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## Reactance: empirical evidence

- Studies confirm basic theory, and link reactance to:
  - age (adolescents maximally susceptible to reactance responses)
  - particular personality types; measurable personality trait itself
- Public health studies focus on persuasion & “forbidden fruit”
  - substantial evidence on reactance and teen alcohol use
  - (on smoking , see Burgoon *et al.*)

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## Messages and reactance

- What message am I getting from “authorities?”
- What is my reactance level?

***message\*reactance***

If *message* = -1 and *reactance* = 1, this term equals -1  
and *ceteris paribus*, I *gain* utility from smoking

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## Putting it all together

- $U = F$  (Networks, Messages, Psychology, Biology)
- Utility = (social coefficient)(weighted sum of network)  
- (message)[(1-skepticism)(reactance + risk aversion)] + pleasure.

If  $U > 0$ , agent decides to smoke;  
Otherwise, agent decides to not smoke.


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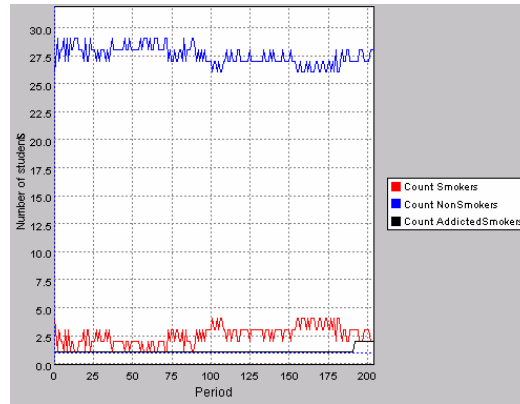
## Runs on network data

- We've collected a large body of school network data.
- Reactance distribution on that data has big impact on message effectiveness.

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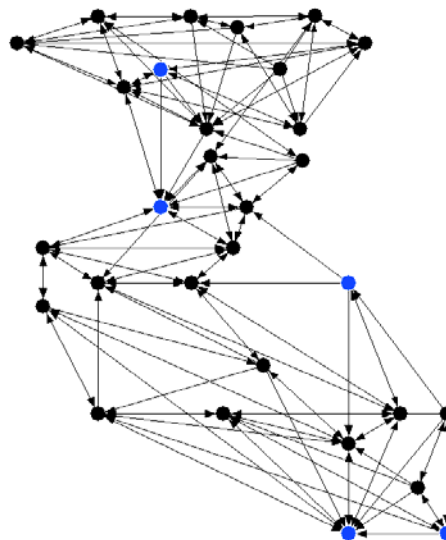
## Zero Reactance. Extreme Message (-1)Effective



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## Case 1: Dispersed reactance

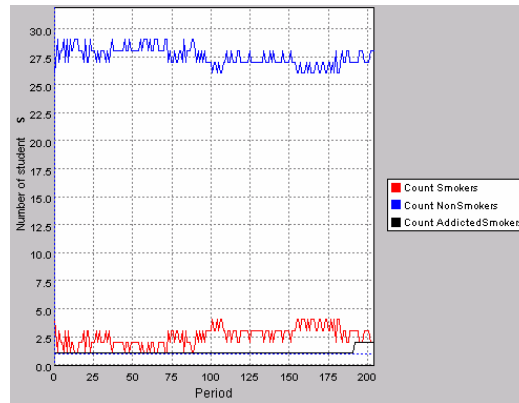


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## Case 1: Dispersed Reactance Extreme Message (-1) Neutral

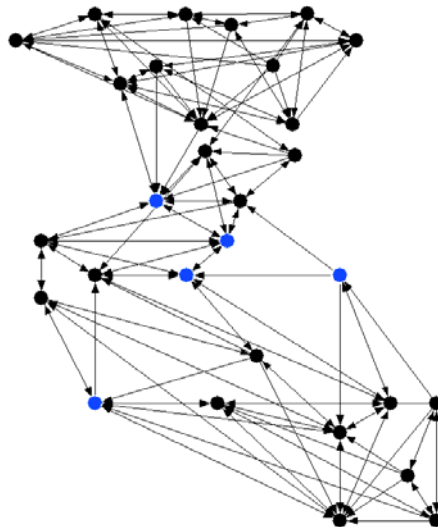
With reactant kids dispersed through the network (not concentrated in a clique), the extreme negative message  $m=-1$  neutral.



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## Case 2: Concentrated reactance



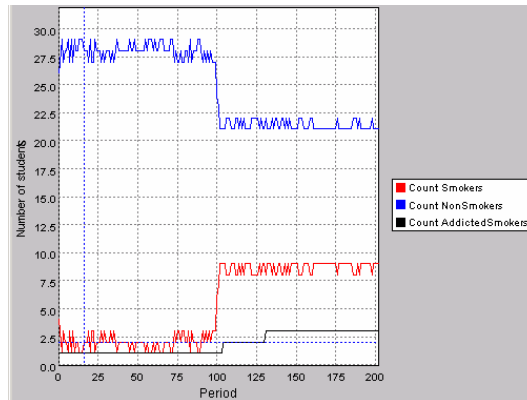
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## Case 2: Concentrated Reactance $M=-1$

However, the same extreme message backfires if reactant kids are concentrated in the network.

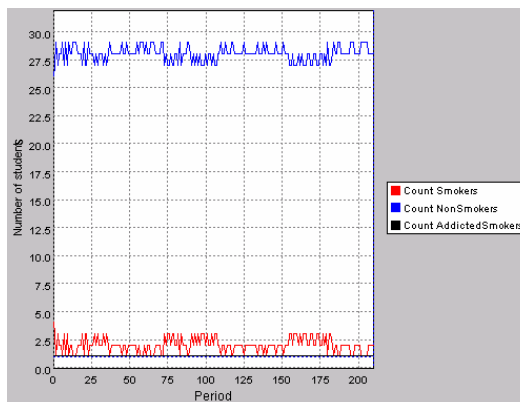


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## Case 3: Concentrated Reactance $M=0.25$

With concentrated reactance, a weaker message does NOT backfire...no epidemic.

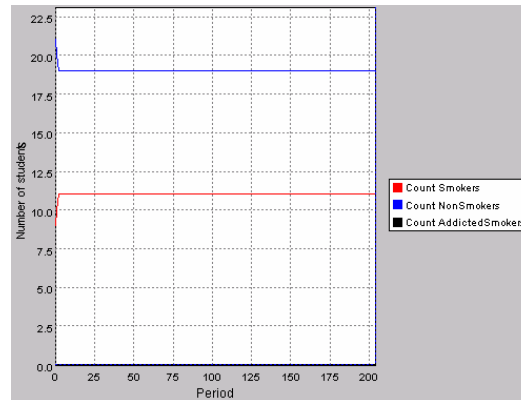


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## Case 4: Dispersed Reactance $M=-0.25$

With dispersed reactance (Case 1), this weaker message is as effective as the extreme one.



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## Extreme messages can backfire.

- In networks where high reactance kids have high weight and high degree, a message of -1 can *increase* smoking.
- In networks where low reactance predominates, or where high reactance kids are low weight and/or low degree, the same message of -1 will be far more effective.

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## Finding the “Sweet Spot”

Suppose message of  $-.25$  is strong enough to dissuade Tim, but that he cares about his peer network. Suppose this is dominated by high reactance kids. The  $-1$  message sends the reactant kids into smoking, and Tim goes along through network effects.

By contrast, a message of  $-.25$  is still strong enough to deter Tim, and weak enough to avert the reactance catastrophe.

### The Policy Goal

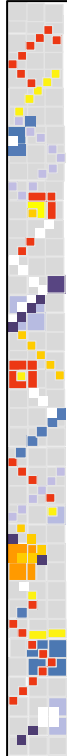
*Find the message strong enough to deter Tim and NOT strong enough to induce the reactance epidemic. This is the “sweet spot.”*



## Tailored interventions

- The sweet spot will vary among communities, and will depend on:
  - network structure (topology and weights),
  - psychological patterns (skepticism, reactance, risk attitudes)
  - biological patterns (addiction functions).
  
- Hence, optimal messages must be heterogeneous, tailored to specific communities, and adaptive over time.





## Summary

- ❑ Social network structure and heterogeneity are critical to understanding the dynamic impact of different forms of intervention.
- ❑ Intervention strategies must be **targeted** to be effective.
- ❑ More empirical studies are needed to determine which policies yield best results for particular groups of individuals.