AN AGENT BASED MODEL OF DISEASE DIFFUSION IN THE CONTEXT OF HETEROGENEOUS SEXUAL MOTIVATION

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Dedication

I dedicate this work to my parents, in lieu of payment.

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Abstract

This project focused on building and analyzing an agent-based model of disease diffusion in order to explore the hypothesis that the relative risk associated with an individual's "sexual motivation profile" (SMP) is influenced by the distribution of strategies represented in the population – that is, that sexual motivation functions as a frequency dependent trait. Sexual motivation is hypothesized to be composed of a sexual inhibition system (SIS) and a sexual excitation system (SES), following the Dual Control Model of Sexual Response. Results of the model show that the relative risk of a SMP does vary depending on the relative representation within a population, but that that variance is constrained by agents' absolute values of SIS and SES. The model produced several parallels with empirical data on humans, suggesting that the model accurately reproduced some aspects of human sexual behavior. For example, agents' SES was a better predictor than SIS of total number of partners, while SIS was a better predictor than SES of Age at Infection. Also, the more accurately the agent population matched the human population, the more the model produced human-like results. Future work should focus on increasing the verisimilitude of agents and their environments, in order to make models more practical for designing and testing intervention and policy strategies.

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Chapter 1

INTRODUCTION

Why do people engage in high risk sexual behavior (HRSB)? What does it mean for sexual behavior to be "high risk?" Most treatments of HRSB emphasize the cost of particular behaviors (e.g., infection, unwanted pregnancy) and the choices individuals make with regard to those behaviors (cf. Broder and Hahman, 2003; MMWR, 2004; Zierler and Krieger, 1997). Individual differences in sexual inhibition and excitation correlate with HRSB (Bancroft, Janssen, Carnes, et al, 2004; Bancroft, Janssen, Carnes, et al, 2003). However, another potential source of risk is the behaviors of other individuals. How the risk-taking of *others* influences an individual's risk is unknown. The goal of this project was to examine the idea that risk is determined by the proneness to risk-taking of others, as well as the existence of negative consequences and an individual's proneness to risk-taking. In order to explore this empirically, an agent based model was used to manipulate the proportionate representation of different sexual motivation profiles in a population and examine the influence of those manipulations on the diffusion of sexually transmitted disease through the system.

Statement of the Problem

The research focused on building and analyzing an agent-based model of disease diffusion in order to explore the hypothesis that the relative risk associated with an individual's "sexual motivation profile" (SMP) is influenced by the distribution of strategies represented in the population – that is, that sexual motivation functions as a frequency dependent trait. Using an agent based model, this project created an artificial environment where agents with different genders, mate values, and levels of sexual

excitation and inhibition made decisions about whether or not to engage in sexual activity. Experiments in this environment revealed how the SMPs of conspecifics can influence the risk associated with any individual profile. Analysis emphasized the influence of individual-level motivations on system-level organization.

Research Questions

Two research questions were addressed in this study:

1. Does the relative risk associated with any given sexual motivation strategy vary according to the representation of that strategy in the population?
2. Can agent based modeling generate global patterns of disease diffusion from local interactions?

Purpose of the Study

This study as designed to create and analyze an agent-based model of disease diffusion in the context of different sexual landscapes, in order to determine the plausibility of the hypothesis that sexual motivation functions as a frequency dependent trait.

Significance of the Study

The significance of this project is threefold. First, this model introduces a unique, new perspective in sex research, using agent based modeling and the idea of frequency dependence to investigate psychological, social and biological influences on sexual risk. Second, it advances an evolutionary approach to sexual motivation which may provide insights into the adaptive function of seemingly maladaptive sexual motivation profiles, such as those leading to sexual risk taking or sexual dysfunction. Finally, it has the potential to help inform intervention strategies, both individual and community-based, by

introducing an approach which accounts for the *consequences of interactions* between heterogeneous individuals, as well as biological, psychological, or social factors in sexual risk taking. Each of these is discussed below.

Perhaps the most fundamental contribution of this project is the introduction of agent based modeling to sexual health research. Interactions across biological, psychological, and social levels of influence are known to be important in shaping sexual behavior, but theoretical models of interaction are scarce (Bancroft, 2000). Other interactional modeling approaches, such as dynamical systems theory (see Fausto-Sterling, 2003, for an application to gender development) exist, but agent based modeling and other computational methods offer a powerful way to model highly complex interactions among multiple agents in a heterogeneous population.

On a more abstract level, the idea of sexual motivation as a frequency dependent trait that is shaped by natural and sexual selection offers two contributions. First it offers an explanatory mechanism for variations in sexual motivation, and second it acts as a theoretical frame for future empirical work in the evolution of human sexual motivation. As an explanatory mechanism, frequency dependence is one explanation for the variety observed in human sexual motivation. Since it is a trait which has social functions, it relies on other individuals for its success or failure (see Wallen 1990, 2001). But variety is also seen in traits which were not under selection pressure – i.e., traits which are not adaptations – and this poses an alternative hypothesis (Lloyd, 2005). A third hypothesis is that variety is the normal variability around the optimal state of an adaptation (Futuyama, 1986). This last hypothesis would assert that humans have evolved an optimal level of sexual motivation which balances the risk of sexually transmitted infection or unwanted

pregnancy against the reproductive disadvantage of never desiring sex. The results of the project suggest which of these three may be most relevant to human sexual motivation. If one SMP (or a subset of SMPs) is found to be universally higher or lower risk than the others, then that refutes the idea of frequency dependence, supporting instead the idea that sexual motivation is optimal. Alternatively, if the model produces results without a pattern supporting either an optimal SMP or frequency dependent SMP, that supports the hypothesis that SMP varies in a population because it has not been associated with reproductive success and therefore has not been under selection pressure.

As a theoretical frame for future research, understanding sexual motivation as an adaptation promotes two mindsets in the study of sexual motivation and sexual risk taking. First it frames the construct of "individual choice" within the context of both genetic and psychological predisposition. This helps to correct the widespread theoretical shortcut which assumes that humans are rational decision makers. Second, thinking of SMPs as frequency dependent traits gives a concrete description of risk. If risk must be assessed contextually, and not simply in terms of an individual's behavior, then understanding the evolutionary pressures that shaped the mechanism underlying that behavior can assist in describing the nature of the risk. A valuable methodology within the theoretical framework of frequency dependence, evolutionary game theory, including agent based modeling, provides a powerful way of exploring hypotheses related to frequency dependence.

Understanding and managing high risk sexual behavior is a crucial step toward controlling the spread of sexually transmitted infections (STIs), particularly human immunodeficiency virus (HIV). While researchers have identified critical risk behaviors

such as high numbers of partners, psychological determinants such as sexual compulsivity (Kalichman and Rompa, 1995; Reece, 2003), and social factors like HIV stigma (Burkholder et al., 1999), no comprehensive model for explaining the interaction of these elements exists. By modeling the system-level consequences of local interactions, complexity theory and methodological approaches which exploit it help to contextualize individual choice within a system of reciprocal determinism and illustrate the influence of system dynamics on individual behavior and its consequences (Bar-Yam, 2002). Accounting for the SMPs of an individual's partners or the SMP landscape of a given community focuses attention on the environment in which individuals behave, while traditional interventions focus on the choices and behaviors of individuals. When researchers understand how individual decisions and their consequences shape and are shaped by larger-scale, social patterns, they can adapt intervention strategies to maximize the benefit and minimize the drawbacks of such interactions, intervening on the environment in which individuals behave, rather than the individuals per se (see Bar-Yam, 2002).

Delimitations

This model serves as a launching platform for future agent-based models of sexual motivation, and as such it is, by design, highly simplified. As Axelrod (1997) puts it, "When a surprising result occurs [in an agent based model], it is very helpful to be confident that we can understand everything that went into the model. … The complexity of agent-based modeling should be in the simulated results, not in the assumptions of the model." (p. 5). Consequently, decisions such as number of agents and spatial constraints influence the outcome of the model as much as the behavior rules of each agent and the

infection properties of the disease. No attempt was made to replicate precisely the specific qualities of human sociosexual systems such as lifespan development, courtship, and long-term monogamy.

A key delimitation in the model was the exclusion of non-sexual transmission of the infection. Although the hypothetical disease (HD) is modeled after HIV, for simplicity its transmissibility is limited to heterosexual contact (see Table 1 for comparison of HIV to the HD). Further, the HD is modeled after the North American infection in its course and virulence.

The model presented here was designed to answer a specific question: does the risk associated with a particular SMP vary depending on the SMPs of an individual's conspecifics? It was intended to explore the utility of agent based modeling in HIV-related health research and to test the plausibility of the idea that sexual motivation is a frequency dependent trait. It was not, however, definitive proof of anything. A great deal more work in a variety of disciplines is necessary to establish the genetic basis and adaptive significance, if any, of the trait. The intention here in terms of evolutionary psychology was not to provide evidence that sexual motivation is a frequency dependent adaptation, but rather to demonstrate the plausibility of such a hypothesis.

Testing this hypothesis alone represented a substantial and intricate project. Because no other models of heterogeneous sexual motivation exist, the model must be generated *ab initio,* including its development, parameter setting, and validation. Beyond the model proper, no protocols exist for the analysis of this particular model, necessitating the development of a grounded analytical method, based on the output of the model. Another substantial challenge is the incorporation of theoretical and empirical

work from multiple disciplines including sexual health research, sexual psychophysiological research, agent based modeling, epidemiology, evolutionary biology, and evolutionary psychology. Due to its interdisciplinarity and novelty, testing the two fairly straightforward hypotheses of this study presents theoretical, methodological, and analytical challenges.

Limitations

This study possesses two primary limitations: lack of competing models and drastic simplification from real systems. The selection of a model from among competing models improves the researcher's modeling power (Shalizi, 2003). The model constructed for this study is a first; no competing models exist. Without a competing model, establishing external validity presents challenges and forces the model to rely on theoretical, parameter, distributional and other measures of validity in the absence of a comparative model.

The severe but necessary simplification of a model relative to the real system on which it is based is the primary limitation of most agent-based models. The present work is no exception. Several factors known or believed to be important in sexual motivation and mate selection were excluded from this model for simplicity:

- The nature of the hypothesized disease is unrealistic. It is transmitted with a high level of probability, infects permanently, has no detectable markers, and does not change agents' behavior. The transmissibility of real sexually transmitted infections (STIs) is influenced by such factors as the individual's sex and preexisting infections. The simplicity of the disease and its transmissibility are intended to decrease distortion of transmissibility on the overall influence of the

sexual motivation landscape on risk. Because the model's variable of interest is how an agent's risk of infection is influenced by the sexual motivation landscape, not the disease itself, a simple disease is adequate.

- Resource abundance or scarcity (Hrdy, 1999; Jones, 1996; Sanderson, 1999; Low, 1999) is not addressed. For simplicity, agents have no intake or expenditure of energy and no dependence on resources in the environment.
- Additionally, this model substantially simplifies the notion of "mate value" or "attractiveness," replacing it with a simple, objective, universally recognized attractiveness score. Many better, more complex models are available in many other models (cf. Simão and Todd, 2002). However, this model relies on a simplification of mate value, since the variable of direct interest is not mate value or the recognition of mate value. The variable of direct interest is sexual motivation and its interaction with incentive value, however it may be instantiated.
- Reputation, an important element in human sexual decision making (Platek, Burch, and Gallup, 2001; Graham et al, 2004) and other social constraints are absent. Agents are amoral relative to sex, and have no social norms imposed to restrict their sexual choice. The only constraints on their decision-making are their own motivation and the motivation of those around them.
- Agents exist in a social vacuum. Social hierarchy is proposed to be a factor particularly in sexual inhibition (Bancroft, 1999; Wallen and Zehr, 2004), and without such a social structure in the model, this factor is not accounted for.

- Agents in this model experience no courtship. The decision to mate or not is based entirely on sexual motivation and the mate value of the partner. Models of courtship exist (Simao and Todd, 2002; 2003), and mediating mating with courtship in this model would likely shift the results substantially.
- Agents never marry pair off in a permanent, exclusive dyad (e.g., marry) which allows for greater freedom to pursue sexual partners than most humans experience.
- Another major factor in human sexual behavior is parenting behavior, particularly in women. While pregnant and breastfeeding women continue to be both proceptive and receptive, in all likelihood their sexual behavior changes in ways which are not accounted for in this model. For simplicity, parenting behavior is excluded here.

These simplifications restrict the generalizability of the data, cautioning a conservative approach in interpreting the results. Future elaborations of this model may add these elements for additional complexity and it is certainly possible that more elements will substantially change the results of the model.

Assumptions

1. Human social processes are complex, with individual, micro-motivation giving rise to system-level, macro-organization (Bar-Yam, 1997; Axelrod, 1997).
2. Agent based models can provide useful, though simplified, representations of dynamics observed in real social systems (Axelrod, 1997; Goldstone and Janssen, 2005).

3. Natural and sexual selection have shaped human psychological mechanisms, including motivational systems, to evolutionarily stable strategies, adapted to the human environment of evolutionary adaptedness (Miller, 2001; cf. Lloyd, 2005, Barkow, Cosmides and Tooby, 1992). No assumption is made with regard to the adaptability of these mechanisms in the context of humans' present environment.

Hypotheses

Null Hypothesis 1: The relative risk associated with any given sexual motivation profile will not vary according to the representation of that profile in the population

Null Hypothesis 2: An agent based model of sexual motivation can not generate global patterns of disease diffusion observed in real human social systems.

Definition of Terms

Adaptation. An adaptation is a trait which has been under pressure by either natural or sexual selection (Futuyama, 1986, p. 215).

Agent-based modeling (AMB) uses a computer program to create a simulation of group social interactions where many individual units ("agents") are given a set of rules and allowed to interact with each other according to those rules. ABMs account for local and global effects, and allows for agents as complex as necessary, as well as communication between agents (Goldstone and Janssen, 2005).

Conspecifics. A member of the same species (Miriam-Webster, 2005).

Complexity theory is an approach to the study of physical, biological, and social systems which have characteristics not immediately explicable from their components (Bar-Yam, 1997). Particular characteristics of complex systems include sensitivity to initial conditions (Lorenz, 1963), a tendency toward equilibrium (Clark, 1998), and

nonlinear (Lorenz, 1963), bifurcated (Thelen and Smith, 1994), or periodic (Lester and Brazelton, 1985; Neda et al, 2000; Li and Savit, 2004) intrinsic dynamics. Intrinsic dynamics refers to changes that occur within the system in the absence of external influence (Clark, 1998). These systems may be physical (Lorenz, 1963), biological (Thelen and Smith, 1994), or social (Lester, 1998). A system's complexity is not measured by the number of components, nor by the complexity of the components per se. Complex systems are characterized instead by many parts interacting such that understanding of a part of the system does not give one understanding of the system as a whole, and systems that are complex must be analyzed at the scale of the whole system (Bar-Yam, 1997). That is, understanding the characteristic of parts of the system will not generate understanding of the whole of the system because the parts do not exhibit qualities found in the whole. Instead, the structure of the whole emerges from the interaction of the interdependent parts. Additionally, while the system may appear to have a cohesive agenda, there is no central controlling agent and the system's parts are interdependent so that a change in one part will initiate a change elsewhere, in ways that are not always predictable (Bar-Yam, 1997).

Diffusion is the movement of a construct through a population (see Rogers, 1962). In the case of the present model, the construct is the hypothetical disease, which will enter the system at a random location and diffuse via sexual contact through the population. Unlike many epidemiological models of disease diffusion, this model allows the diffusion to occur as a consequence of local interactions between heterogeneous agents, rather than homogeneous interactions in an imposed network structure.

Emergence, is one primary principle of dynamical systems. Emergent properties of a system *cannot be altered directly*; rather one must change an element within the system and measure whether that change results in the change in emergence one sought. The self-organization of the swarm or flock is emergence, a pattern of behavior that both arises from and influences the internal functioning from the system (Clark, 2001). To put it another way, according to Nowak and Vallacher (1998):

> Rather than being imposed on the system from above or from outside the system altogether, the higher order structures emerge from the internal workings of the system itself. In this process, the system loses degrees of freedom, and the state of the system may be described by a small number of variables. Ironically, then, complex systems can sometimes be described by fewer variables than can relatively simple systems. (p. 53)

Frequency dependence is a term which describes traits whose reproductive benefit is influenced by the strategies of that trait exhibited by an individual's conspecifics (Maynard-Smith, 1982; Cronin, 1991). Unlike optimal traits, frequency dependent traits will exhibit variety across a population. Unlike traits which were not under selection pressure, variations on the trait within the context of a social system will influence the reproductive fitness of an individual.

High risk sexual behavior is that sexual contact which puts an individual at risk for sexually transmitted infection (Bancroft, Janssen et al, 2003; Reece, 2003). In real systems this also includes social and reproductive consequences, but for the purposes of the model risk is limited to infection. Risk varies depending on the existence of an infection in the system and the individual's decision to have sexual contact. The model

will test the hypothesis that risk also varies with the proportionate representation of different sexual motivation profiles.

Parental investment. Parental investment is expenditure of resources on offspring for their survival at the expense of other offspring; such expenditure ranges from metabolic energy producing gametes to complex caretaking behavior (Trivers, 1972).

Sexual landscape. In the context of the model, the sexual landscape is the proportionate representation of SMPs. The model tests whether variation in the landscape affects the risk associated with any given SMP.

Sexual motivation constitutes an individual's sexual inhibitory and excitatory mechanisms which motivate sexual behavior. It bears the characteristics of inhibition and excitation outlined by the dual control model of sexual response (Bancroft and Janssen, 2000), plus these characteristics: abstinence does not produce aversive affect, but does increase the palatability of incentives (Singer and Toates, 1987); it is conditioned through innate hedonic systems, which have higher thresholds in response to stimuli that present greater risk (Toates, 1986); and females exhibit overall higher levels of inhibition and lower levels of excitation than men (Graham et al, 2004; Carpenter, 2002; Bjorklund and Kipp, 1996).

Sexual motivation profile or *phenotype.* In the context of the model, this is an agent's combination of sexual inhibition and excitation. Inhibition refers to the agent's sexual behavioral damping, or "brakes," while excitation is the agent's sensitivity to sexually relevant stimuli. Agents of high excitation and low inhibition are likely to pursue sex more actively than agents of low excitation and high inhibition.

Systems are components interacting in an organized way (Nowak and Vallacher, 1998). Closed systems consist of components interacting in the absence of external influence. Open systems consist of components interacting both internally as well as in response to the "suprasystem," or to external influences. All living systems are open, including social systems. Complex adaptive systems are those which reorganize themselves in response to changes in the suprasystems (Bar-Yam, 1997).

Trait. This is defined as "a correlated set of features caused by a single developmental or ecological process" (Fristrup, 1992, p. 43). Strictly speaking, this project treats sexual motivation as two traits – a sexual excitation system and a sexual inhibition system. For brevity this is often collapsed into the singular trait of sexual motivation.

Chapter 2

REVIEW OF RELATED LITERATURE

In order to locate this project within existing work, a review of related literature must encompass theoretical, empirical, and methodological elements. This chapter first elaborates the concept of frequency dependence, emphasizing the potential utility of this concept in thinking about sexual motivation and sexual risk taking. Next it describes agent based modeling as a method of examining frequency dependence. This is followed by a review of extant models of agent based models, including models of frequency dependence, disease diffusion, and sexual behavior. Then the major theories of sexual motivation are described and a working definition is generated. Since the concept of frequency dependence ultimately assumes that the trait in question is an adaptation, this includes a discussion of whether or not sexual motivation is an adaptation, and what evidence might prove or discount this possibility. This is followed by is a description of HIV's transmissibility and course. The chapter concludes with a discussion of high risk sexual behavior, and its behavioral, psychological and environmental determinants. The ultimate aim of this review is to build the case for agent based modeling as the optimal methodology for exploring disease diffusion in the context of heterogeneous sexual motivation.

Frequency Dependence

A species holds traits in common, yet variability in some traits is observed within a species. It may be that the variability results from a lack of selection pressure – that is, the trait does not influence reproductive fitness, nor is it directly tied to a trait which does, and therefore variability may exist without affecting an individual's reproductive

success (see Lloyd, 2005 for a discussion related to female orgasm). It may also be that the variability is simply the normal variability around an optimal solution (Futuyama, 1986). But a third possibility is that the trait exhibits variability within a species because it is frequency dependent. A trait is frequency dependent when "the success of [an organism's] behavior may well depend critically on the relative frequency of its own behavioral type in the population to which it belongs (its species, say, or sex, foraging party or nest): if success depends on being the rarer of two types, then selection will automatically maintain variability" (Cronin, 1992, p. 75). As contrasted with simple optimization, where an adaptation is successful or not without reference to the behavior of others, frequency dependence results in multiple outcomes in the population, rather than one uniform outcome. Anisogamy – sexual dimorphism of gametes – is an example of this: larger gametes may be selected for because they produce organisms which survive better. As females begin to invest more energy in producing a small number of larger gametes, males are favored when they produce a large number of the smallest gametes which are still physically capable of traveling to the female gamete. The stable solution resolves in the smallest number of the largest eggs a female can afford and the largest number of the smallest gametes the male can afford (Smith, 1984; Wade and Shuster, 2002).

Jones (1996, p. 20) lists five potential outcomes unique to frequency dependence:

1. Evolutionarily Stable Strategy (ESS) emerges when, rather than achieving an optimum solution, a species may reach an equilibrium, where no one benefits from adopting a different strategy, though the whole would benefit if *everyone* adopted a different strategy. Importantly, the optimum rule for any individual to

follow is different when it depends on others than when it does not matter what others choose to do. In the second case, likely all successful conspecifics will adopt virtually identical strategies, given virtually identical environments. Maynard Smith specified (1982) that ESS's are characterized by "uninvadability" (p. 10), that is, the quality of the system which makes it impossible for mutations in strategies to generate reproductive benefit.

2. In an arms race, all try to get ahead with no one making any substantial gain. The quintessential example of arms race is the constant co-evolution of an organism and its parasites. The moving target or "red queen" hypothesis promotes this arms race as the very origin of sexual reproduction; the combination of two individuals' immune systems, rather than random variation in a clone, provides much faster adaptation against infections; evolution then favors parasites with faster mutation, which spurs adaptations in immune systems, and so on (Hamilton, Axelrod, and Tanese, 1990).
3. Frequency dependence may favor reproductive success of individuals but reduce viability of the group. For example, if individuals in a species produce many offspring, most of whom fail to survive to reproductive age, females who reproduce more have a selective advantage over those who reproduce less. Yet if the females had fewer offspring and thus did not allocate environmental resources to offspring who would not themselves reproduce, the species as a whole would benefit.
4. Handicapping generates traits that exhibit waste and extravagance. The quintessential example is the peacock's tail, a metabolism hog which serves no

survival function except as a signal to peahens about a peacock's health. Though it may pose a threat to a male's longevity, if he is attractive to females, he will be reproductively successful.

5. Runaway selection is a co-evolutionary positive feedback loop which amplifies arbitrary traits. Again, sexual displays like the peacock's tail exemplify this process. Even though an individual male's fitness may be decreased by a large tail, if females prefer it then he is more likely to produce offspring. Females, in turn, who prefer small tails may have sons who survive longer, but unless that small tail is attractive to females, his longevity is inconsequential. Her short-tail preferring daughters will face the same difficulty with their own sons, and the preference will be extinguished, while the preference for long tails, along with the tails themselves, will grow.

In each case, frequency dependence gives rise to traits which may seem wasteful, meaningless, or even counter-productive, *prima fascia*. Thus individual interest cannot account for these traits; instead, these traits are a product of the system in which they evolve.

Evidence indicates that that human sexual motivation could be frequency dependent. Variability is observed in sexual responsiveness (Carpenter, 2002) and sexual contact with others is not strictly a product of an individual's motivation to engage in such behavior; it is also mediated by the motivation of one's potential partners, through consent or initiation, and the motivation of other members of the same sex, through intrasexual competition. Not yet established is whether or not reproductive success

"depend[s] critically on the relative frequency of its own behavioral type in the population to which it belongs" (Cronin, 1992, p. 75).

Fitness effects of sexual behavior are not limited to reproductive benefits. Individuals also risk infection with a sexually transmitted infection, which has the potential to lead to reduced fertility or sterility, transmission to the infant, and social stigmatization (Kurzban and Leary, 2001). Do the risks and benefits associated with sexual behavior change if the population is more or less populated with individuals who are more prone to risk-taking behavior? In short, could sexual motivation function as a frequency dependent trait?

Agent Based Modeling

A classic way of examining frequency dependence is evolutionary game theory (Maynard-Smith, 1982). Agent based modeling, an extension of evolutionary game theory, allows a large number of heterogeneous agents to interact according to clearly defined rules, in order to observe the relationship between local interactions and the system-level organization of the population, such as the scale-free distribution of number of sex partners (Liljeros, et al 2001).

Distributed artificial intelligence, or agent based modeling (ABM) uses a computer program to simulate group interactions where distinct units ("agents") are given a set of individual characteristics or traits and are then allowed to interact with each other (Gilbert and Troitzsch, 1999). The object of such a model is to understand how individual level motivation iterated across a population may give rise to higher level organization. ABM is a social science simulation technique which can account for local and global effects, and allow for agents of any degree of complexity, including the ability to

communicate. ABM provides a strictly controlled environment for examining the higher level consequences of individual level decisions. It is an ideal method for studying "spatially distributed systems of heterogeneous autonomous actors with bounded information and computing capacity" (Epstein, 1999, p.56), of which human social systems are a prime example.

As with all methods, ABM has a number of strengths and limitations. While ABM forces a massive simplification of the system, thus limiting its generalizability, it also gives a controlled method for manipulating the parameters of the system. Such control allows exploration of the interaction between agents and the environment. Additionally, ABM is absolutely theory dependent – the program is only as good as the model, and the model is only as good as the theory. Thus a valid model demands well-reasoned parameters and cautious interpretation. The theory underlying ABM is complexity theory.

Complexity

Complex systems are characterized by emergent properties, qualities of the system which are "not readily understood from the behavior of the parts" (Bar-Yam, 1997, p. 10). These systems consist of "a large number of interdependent components" (Shalizi, 2003, p. 6) such that a change in one part of the system may give rise to changes in another part. An example of a complex system is a flock of birds: one might describe everything about an individual bird, and yet still not have described flocking. A description of the flock itself is a description of the *interaction* of birds and is observable only at the interactional level (see Reynolds, 2001). Flocking is an emergent property of the system.

Complexity theory is a useful way for public health professionals and policy-makers to think about social systems, because emergent properties are resistant to the usual interventions. Emergent properties are not available for "twiddling" (Clark 2001, p. 99); that is, they can only be accessed at the interactional level, not the individual level. Consequently, these characteristics are extraordinarily stable and difficult to change. Metaphorically, a health intervention might persuade individual birds not to flock. Yet even if some birds behave differently, the rest of the system is intact. The flock still emerges. To change emergent characteristics requires understanding the environmental conditions which generate them and then directing interventions toward those conditions, rather than toward the individuals in the system.

Health behavior and health behavior interventions have historically been viewed from a wide variety of theoretical and methodological approaches. What does agent-based modeling uniquely contribute? Five distinct but related reasons are suggested: first, individual theories such as the Health Belief Model fail to account for the social nature of sex (Schroeder and Rojas, 2002), whereas ABM specifically treats the individual in the context of a social environment. Second, the trend in health research is toward multi-level approaches, which acknowledge the reciprocal influence between individuals, their social networks, and the policies and institutions in which they function (Glanz, Rimer, and Lewis, 2002). ABM specifically models the *interaction* of the levels, illustrating how motivation on the local level shapes the observed global patterns. Third, ABM offers more rigor than natural language description of systems, and more adaptability than mathematical models (Bonabeau, 2002; Macy and Willer, 2002). The ability to control parameters in simulations gives researchers more experimental power than ethnographic

observation or even intervention. The trade off is that the models are simplified, a problem also associated with some mathematical models, which lack the flexibility of agent based models. Fourth, ABM offers a different way of studying social behavior, examining not simply how a static limitation on the field of possible choices affect choice, but how social systems behave, interacting reciprocally with individuals. When an individual's behavior is stochastic, highly complex, evolving, or happening in the context of a heterogeneous environment, ABM is the method that can best explore that behavior (Bonabeau, 2002; Epstein, 1999). Finally, ABM can produce visual models which are appealing and intuitive to understand, making such models ripe for diffusion among public health professionals and clients. The very intuitiveness and persuasiveness of the models is a potential danger, since it may lead to overdrawn conclusions, under-determined theory, or sloppy application. But used rationally and systematically to think about how environmental and individual factors interact to shape the population's health patterns, ABMs can help generate interventions which are specifically adapted to the peculiar characteristics of complex systems (see Bar-Yam and Kuras, 2003).

Certain kinds of complex social phenomena lend themselves to ABM. These phenomena can be categorized as cooperation, patterns and organizations, and disease diffusion (or, more generally, contagion) (Goldstone and Janssen, 2005). The current project is an example of disease diffusion, and other examples of this group are described below. More generally, simulation is valuable, and perhaps preferable as a methodology in six situations identified by Marney and Tarbert (2000):

1. Where there are complex emergent global processes and dynamics from simple local behaviour.

2. Where coordinated global outcomes are generated by the heterogeneous local decision rules.
3. Where the representation of the unfolding of the dynamic process is an important part of the overall modeling process.
4. Where, on the grounds of realism, it is desired to improve mapping iso-morphism in either the input or output of a simulation.
5. Where it is desired that the characteristics of the behaviour being modeled encompasses holism (section 6.2).

Building the Model

Gilbert and Triotzsch (1999, p. 18) outline four stages in social science simulation: design, build, verify and validate, and publish. Designing the model is the process of determining which elements of the target system will be included or excluded from the model. Models which are primarily theoretical, like the present project, lean toward simplicity, while predictive models tend toward a larger number of more precisely determined variables. To build the model, researchers select a programming language based on its efficiency, to handle the many runs; its graphics library, in order to manage and illustrate a large amount of data; its freedom to make incremental changes, since models are generally exploratory; and its acceptability to the modeling community. Verification consist of running test cases to find run time errors, and validation, elaborated on below, is the researcher's comparison of the model's data to data from the real system.

Data Analysis

There is no uniform practice for data analysis for agent based models, though there are certain standards of practice (Gilbert and Troitszch, 2004). As a starting point, three issues distinguish ABM data analysis from standard social science data analysis: (1) agent based models have a different purpose; (2) there is not a standard or typical way to report data; and (3) inferential statistics are of either dubious or varying value.

To begin with, the models have a different purpose from that of surveys or other typical forms of social research, and thus approach the data differently. They "provide computational demonstrations that a given microspecification is in fact *sufficient to generate* a macrostructure of interest" (Epstein, 1999, p. 42, emphasis original). For example, the simulations discussed below are used to "check the consistency of models and derive their implications" (Caldas and Coelho, 1999, section 10.5), explore what logically possible outcomes of a system are stable (Smith and Stevens, 1999), and look at redistribution of cost in a heterogeneous system (Castelfranchi et al., 1998). These three purposes share the theme of looking at the system itself, the structure of interactions, and the movement of resources through the system, outcomes which are not observable in any individual. The standard purpose is to establish a correlation between two constructs or measure the change in a construct over time. The different purposes give rise to different management of the data.

The second way that data analysis varies from standard social science is the lack of standard reporting method. For example, three different reporting methods are natural language descriptions of the system's dynamics (Smith and Stevens, 1999; Caldas and Coelho, 1999), function plots (graphs) of behavior of individuals or groups across time

(Caldas and Coelho, 1999) and descriptive statistics (means and standard deviations – Castelfranchi, Conte, and Paolucci, 1998; Saam and Harrer, 1999). Since models are built for specific purposes, a standard or generalized reporting method would constrain reports inappropriately. Instead, model results are reported in whatever way is specifically relevant to that model.

Thirdly, agent based models tend not to report inferential statistics. Instead, they describe interaction patterns between individuals or system-level behavior over time, which is qualitatively different depending on variations in agent characteristics. The overall newness of the method in modeling human health behavior, the relative lack of control had by the experimenter, and the large number of data points make data analysis more an art than a science (Bonabeau, 2002). Statistical differences found in artificial societies may have no meaning, since statistics may be explicitly built into the model or be substantially influenced by minute changes in the parameters. Further, differences between individuals are typically not of interest; instead the interactions between them or the behavior of the system is the target of analysis. Descriptions of dynamics, rather than statistics, are more relevant. While it is certainly possible to perform statistical analysis of the results of agent based models, it is unknown what value these analyses hold.

Validity

Empirical research is subject to tests of validity – do the results of the study reflect what is known to be true about the system? The standards and techniques for assessing validity of simulation models vary somewhat from those for statistical models. For these models, internal validity consists largely of analysis of the output of the model to determine whether its results are consistent with the models intended goals and

purposes. Evaluating the model's "internal validity" consists of debugging – do the numbers make sense in the context of the model? Is the program free of logical, implementation, and run-time errors? Essentially, internal validity is limited to establishing whether a model runs as it is intended, without reference to the model's relationship to any other existing models, theories, or data (Gilbert and Troitzsch, 1999; Boero and Squazzoni, 2005).

External validity, on the other hand, compares the model with existing data, models, and theories. Küppers and Lenhard (2005) argue that a social science simulation may be considered valid when "some of the characteristics of the social dynamics known from experience with the social world are reproduced by the simulation" (1.3). Often results of physical system simulations are judged on the basis of model results rather than precise replication of the real system – i.e., the equations describing the system are known, but so complex as to render a simulation model unstable; therefore the mathematics of the models are adapted for long term stability in the computer simulation, and this adjustment does not influence the "validity" of the model; indeed since the pattern of results may be superior to more mathematically realistic models, they are considered more valid (Küppers and Lenhard, 2005). Social science simulation validity parallels this – the simulation is considered valid when it reproduces the structural, rather than mechanistic, qualities of the real system (for an example of this method of validation applied to an epidemiological model, see Bagni, Berchi, and Cariello, 2002).

Ahrweiler and Gilbert (2005) discuss the problem of validity in terms of social construction – one does not compare the real system to the model; instead the objects of comparison are "*what you observe as the real world* and what you observe as the output"

(section 3.1, emphasis original). The authors resolve the epistemological problems inherent in a constructivist view by relying on expert and end-user evaluation: the validity of a simulation model derives in part from the very process of generating a model (section 4.1). This is, more generally, the position of naturalized epistemology, that knowledge is assessed in terms of the process by which it is generated, as well as more traditional criteria (see Quine, 1977). This is of particular importance for predictive models, an issue Shalizi (2003) confronts when he notes that models of complex systems are inherently biased because they are designed to be a close match to the available data – i.e., over-fitting. But for theoretical models, too, such as this project, over-fitting can result in under-determined theories, without enough data to distinguish between two equally plausible explanations. Shalizi offers four practices for improving the validity, internal and external, of any model of a complex system: "(1) Replication is essential. (2) It is a good idea to share not just data but programs. (3) Always test the robustness of your model to changes in its parameters. (This is fairly common.) (4) *Always* test your model for robustness to small changes in qualitative assumptions" (pp. 27-8). Based on the issues discussed above, it also seems important to recognize the influence of the process of modeling on the results, and to interpret them cautiously.

After reviewing the epistemological issues inherent in agent based modeling of complex systems, more straightforward categories of types of validity make useful heuristics. Carley (1996) categorizes types of external validation as face, point, value parameter, process, pattern, distributional, and theoretical. Face, point, and value validity deal with what might be considered superficial analysis. Face validity, where the model appears to match the real system, is not particularly informative. Point validity (where the

means of independent variables have the same mean as other data) and value validity (where "the specific results from the computational model match on a point by point basis the real data," (Carley, 1996, p. 12) are valuable when the goal of the model is to reproduce the exact behavior of another model. Process validity may result in wildly different means, while still generating the same dynamic. Distribution validity, having a broader scope than point validity, falls somewhere in between. Process validity is determined by how closely "the computational model corresponds to real processes" (Carley, 1996, p. 12). Distributional validity is determined by how closely "the distribution of results generated by the computational model has the same distributional characteristics as the real data; e.g., means, standard deviations, and shape of results are the same" (Carley, 1996, p. 12). Theoretical validity, perhaps the most powerful measure, is high when "the underlying theoretical constructs in the computational model provide a better predictive indicator of real data than does a linear model" (Carley, 1996 p. 12). Parameter validity is shown when the model values match "values observed for parameters in field, survey, archival or experimental settings" (Carley, 1996 p. 12). Pattern validity is shown when "results generated by the computational model matches real patterns of results" (Carley, 1996 p. 12).

Process validity is often paramount, though perhaps the most difficult to assess. Typically the statistical equivalent to process validity is distribution validity – because the results fall into the theoretically predicted distribution, they are assumed to match any hypothesized underlying mechanism (Carley, 1996). However, for an agent-based model, the goal is to reproduce not merely a matching end-state, but also a matching through-state. Due to the profound complexity of real social systems, it is unlikely that a

simplified model of a social process will produce a perfect match with the real system; instead one attempts to reproduce a fragment of that process, so that this fragment can be incorporated with other fragments of social process developed in other models. So while process validity is crucial, direct knowledge of the model's match to the real-world process can only be indirectly known.

Extant Agent Based Models

Agent based modeling (ABM) has been utilized to ask similar questions about frequency dependence, disease motivation, and sexual behavior. This section reviews relevant existing models of each of these three constructs. First is a presentation of relevant ABMs of frequency dependence. This is followed by a review of models of disease diffusion. Because the many relevant epidemiological models are based on static networks of homogeneous agents, this discussion emphasizes the particular benefits of ABM compared to network models. Finally, models of sexual behavior are reviewed and discussed in relation to the present model.

Models of Frequency Dependence

ABM facilitates the investigation of frequency dependence by allowing heterogeneous agents to function autonomously. As such, ABM is a valuable and frequently used methodology in computational evolutionary biology. ABMs of frequency dependence have two general structures, the classic structure, and evolutionary game theory (cf. Axelrod, 1984, Axelrod, 1991, ch. 1; Maynard Smith 1982). In classical game theory agent based models, agents interact according to preset strategies, the success of which is dependent on the strategies of other agents. Models in evolutionary game theory, in contrast, incorporate genetic algorithms such that agents "reproduce" with mutated,

sometimes sexually integrated (i.e., half of the strategy of one agent, half of the strategy of another) strategies. Agents reproduce in accordance to their success within the game – unsuccessful agents do not reproduce, and highly successful agents reproduce multiple times. The next generation is thus "adapted," with novel strategies that are hybrid recombinations of successful strategies from the previous generation. A classic example of evolutionary game theory is the "red queen" theory, reviewed by Hamilton, Axelrod and Tanese (1990), which proposes that sexual reproduction is an adaptation which evolved in an arms race against parasites. In such models, asexually reproducing parasites compete with sexually reproducing hosts to test under what conditions sexual reproduction (far more costly than asexual reproduction) can be maintained.

In comparison, Jager, Popping, and van de Sande (2001) built a classic agent based model, exploring the influence of different proportions of different types of agents on crowd behavior. This type of model and its hypothesis are similar to the present model of sexual motivation. The authors hypothesize that crowd size, group size, and proportion of "hardcore" members will influence whether, when, and where a riot occurs in the simulated environment. Agents can be hardcore, hangers-on, or bystanders, the distinction being the frequency with which the agent scans the environment. Hardcore members scan half as often as hangers on, who scan one fourth as frequently as bystanders. Agents are further tagged with a group identity ("party") and 15 "acquaintances." All agents have limited vision and "aggression motivation," whereby their tendency to approach members of their own party increases as the representation of their party drops, and vice versa. Agents out of contact with other agents move in one of three ways, with differing probability. When the agent perceives another agent, it

approaches it if it is a member of the same party, with a preference for acquaintances, or fight with non-party members. The authors conclude that large groups cluster more than smaller groups, given the same space, indicating that density is a determinant of clustering. Also, asymmetry promotes fighting behavior. Proportion of hardcore members appears less important a determinant of fighting and clustering than symmetry and group size.

This model provides a comparable framework to the present project, and its results are relevant to interpretation of the present model's results. In both, agents are assigned to one of two groups, assigned characteristics that vary systematically and which are expected to influence the results. The proportionate representation of "high risk" agents (hardcore agents) is varied. Jager and colleagues find that size and asymmetry in groups is more influential than proportion of hardcore agents in determining crowd behavior. The present model keeps both group size and symmetry constant, thus measuring only the influence of changing proportions of high risk agents. This model of crowd behavior indicates that, in a model of sexual motivation, operational sex ratio and population density will like influence results. This result should be taken into account when interpreting the results of the present model, as well as in future elaborations of this model.

Models of Disease Diffusion

Two general kinds of disease diffusion models are ABMs and network models. Epidemiological network models have been used for more than 60 years for tracking sexually transmitted infections (Doherty, Padian, Marlow, and Aral, 2005). Advances in computational power of computers has allowed for the analysis of theoretical network

structures, using a static structure to examine how a disease spreads through different types of connectivity, and what types of interventions stop an epidemic (Dezső and Barabási, 2002).

Agent based modeling offers multiple advantages over network modeling for disease diffusion, primarily the ability to generate system-level patterns from the independent interactions of heterogeneous individuals. AB disease diffusion models traditionally follow a standard Susceptible-Exposed-Infected-Recovered (SEIR) framework (see Rahmandad, 2004, for a review and comparison with mathematical models). For example, a model of foot-and-mouth disease uses agents to represent individual animals and follows Susceptible-Latent-Infectious-Removed (SLIR) (Chen, 2001). A model of bovine leukemia follows a Susceptible-Infected-Detectable (SID) model (Bagni, Berchi, and Cariello, 2002).

The present model will follow an adapted, S-I-D model – Susceptible, Infected, Deceased, following the trajectory of HIV infection. Since newly infected HIV+ individuals are infectious, the Exposed phase is eliminated. Functionally, the model's lifecycle, whereby infected agents die after a number of timesteps, to be replaced by an infection-free clone, is similar to Recovery insofar as an identical agent exists, but is infection-free. However, this device of the model does not reflect the real trajectory of HIV, and serves only to remove infected agents from the system while also maintaining the proportionate representation of different SMPs.

Existing ABMs of HIV transmission are few, and those which exist are quite different from one another. Shiwu and Jiming (2005), for example, developed a massively multi-agent system (MMAS) of HIV-immune interaction. Rather than

modeling the spread of HIV through a population of humans, their model focuses on the interaction between the virus and an individual immune system. Though less immediately relevant to the present project, the HIV-immune model is an example of a biological, rather than social, application of ABM to the problem of HIV.

Teweldemedhin, Marwala, and Mueller (2004) present a model of HIV transmission in a population of agents which vary in gender, HIV status, personality type, safer sex practices, addiction, and relationship status. Unlike the most standard epidemiological models, it represents only infection rate, without progression of infection. Based on epidemiological and census data for South Africa, the model assumes that males are 15% less likely to be infected than females. Researchers varied total number of agents in the model, and found that in all variations between 20.7-21.97% of the population was infected after four simulations, representing one year. They further found that "there is a strong relationship among outcomes of the simulation steps for a particuler (*sic*) group of agents" (p. 158), though they do not specify which group. Unlike the present model, Teweldemedhin and colleagues incorporate higher level functions such as safer sex and monogamous relationships to produce infection rates similar to those observed in the real population.

Models of Sexual Behavior

Existing agent based models of sexual behavior are limited to those related to mate choice – i.e., mate selection and mate preference. These models differ from the present model insofar as, in existing mate choice models, all agents are equally likely to pursue an agent of a given mate value relative to their own. That is, agents may have heterogeneous mate value, heterogeneous mate search strategies, or heterogeneous mate

preference, but they have homogeneous motivation to pursue a mate. The first section below distinguishes between mate selection, mate preference, and mate choice, and contrasts these to sexual motivation. The next section discusses the existing literature on agent based models of mate selection, the nearest construct to sexual motivation. Included is a justification for approaching mating behavior in terms of sexual motivation, as the present model does, rather than in terms of mate preference.

A few definitions will serve to clarify the distinctions between related but separate concepts: *Mate preference* is the psychological machinery underlying choice (Buss, 1989). *Mate selection* is about pursuit strategies and tactics (Jaffe, 2002). *Mate choice* is the interaction of mate preference machinery and mate selection strategies with the real ecology of the system – available mates, one's own attractiveness, the operational sex ratio, and the spacing and timing of mating (Miller and Todd, 1995; Jones, 1996; Geary et al, 2004), the result, so to speak of mate preference and mate selection psychological mechanisms acting in a constrained social and ecological environment.

Sexual motivation is defined in the context of this project as an individual's sexual inhibitory and excitatory mechanisms, which motivate proceptivity and receptivity. This might be paralleled to mate selection, since both primarily concern an individual's internal machinery which generates affect and behavior, whereas mate preference concerns primarily an individual's machinery which makes judgments about the quality of an incentive (in this case, a potential mate). Mate preference is relevant to sexual motivation insofar as sexual motivation is an appetite, rather than a drive, and the appeal of external incentives can generate behavior even when an organism is relatively sated. A large amount has been written on the subject of mate preference in humans;

however, a thorough discussion of mate preference (and by extension mate choice) is outside the scope of the current work. Suffice it to say that humans have evolved propensities for being attracted to other humans who possess certain qualities hypothesized to be innately appealing, such as gender-appropriate waist-to-hip ratio, low facial fluctuating asymmetry, high social status, compatible genes, and a different immune system (Buss, 1989; Miller, 1997, 2001; Paul, 2002; Geary et al, 2004; Jaffe, 2004; Jones, 1996; Thornhill and Gangestad, 1996). The present model simplifies mate preference with a simple, objective, numerical score for mate value.

The study of mate preference in evolutionary psychology is the search for characteristics of one sex that are correlated with ratings of attractiveness by the opposite sex. Agent-based modeling has been used to model mate choice, the connection of mate preference with mate selection strategies. Miller and Todd (1998) outlined three functions of computational models of mate choice – perception of sex cues, judgment of attractiveness, and search strategies – and the present project is none of those. Instead, this is a model of sexual motivation, examining male and female sexual inhibition and excitation, whittling away the cognitive elements emphasized in those authors' work (Simao and Todd, 2003; Simao and Todd, 2002; Werner and Todd, 1997; Todd, 1996; Miller and Todd, 1995, 1998). The exclusion of cognition in the present project is not intended to deny the potential importance of cognition and mate selection processes, but rather to add a sexual psychophysiological, motivational component to the explanation of human sexual proceptivity and receptivity. Given the essentialness of sexual behavior to human existence, it seems plausible that a great deal of "decisions" and "judgment" related to mate choice are less cognitive than they are appetitive.

A difficulty with existing models is that many assume that "mating" means a single, life-long, monogamous pairing (Simao and Todd, 2002). Ethnographic data from contemporary pre-literate cultures indicates that neither monogamy nor life-long coupling are typical in human societies (Hrdy, 1999). Indeed most humans living in the modern industrialized west report having had more than one sex partner in their lifetime (Laumann et al, 1992) and 15-17% of heterosexual men and women report having had an extra-marital affair (NORC, 1998), despite widespread social norms condemning extra-dyadic sexual contact. Instead, while human sociosexual systems appear to be influenced by such environmental factors as resource abundance and parasite density, anthropological evidence asserts that we are on the whole a polygynous species (Low, 1999; Sanderson, 2001). The assumption of monogamy substantially alters the kinds of mate search strategies which are effective in the model, decreasing verisimillitude (Todd, 2005). To manage this issue, for the purposes of this model "to mate" is synonymous with "to engage in sexual contact," without references to social relationship.

A second difficulty is the assumption in standard mate selection models of homogeneous motivation to pursue mating, though human societies exhibit heterogeneity in sexual motivation (see Carpenter, 2002). The mate choice models allow for differences in mate preference, as well as different mate values and different mate selection strategies, but agents in these models are equally motivated to mate. A third difficulty is that they do not account for negative health consequences of mating. While sexual contact has many benefits for humans, from reproduction to social bonding, it also has potential risks, such as infection, negative social consequences, and unwanted pregnancy. Mate choice models focus exclusively on strategies for selecting mates rather than

balancing the costs and benefits of mating. Models of insect and other animal systems have incorporated the risk of sexually transmitted organisms into mating models, but of course these systems are based on sociosexual systems such as female choice with male intrasexual competition, much simpler than human sociosexual systems (Seeman and Nahrung, 2005; Nunn et al, 2003).

A complex systems approach to social behavior can generate more parsimonious explanations than those which rely on descriptions of individual mechanisms (Hemelrijk, 2002). Evolutionary game theory has is a fundamental method for studying frequency dependence (Maynard Smith, 1977, 1982; Soltis and McElreath, 2002). Agent-based modeling, an extension of evolutionary game theory, allows researchers to create a large number of virtual organisms and allow them to interact according to hypothesized rules. Thus it provides a useful empirical frame for studying the impact of heterogeneous sexual motivation on a population's health risk.

Sexual Motivation

Rather than reviewing empirical work on sexual motivation, this section is chiefly intended to review theories of sexual motivation and, from those theories, generate a working definition and operationalization of the construct. To do this, this section begins with a history of theories of modern motivation research, beginning with Freud. The subsequent section outlines critical differences observed in women's and men's sexual motivation. The concluding section establishes a working definition and operationalization of sexual motivation in the context of this agent based model.

History of Theories

Modern theories of human sexual motivation emerged from the work of Freud. Freud's theory was a theory of drive motivation, where sexual impulses are generated by negative internal sensations, though most of the subsequent theories of sexual motivation are incentive motivation theories. Four characteristics distinguish incentive motivation theory from drive motivation theory. First, and most apparently, incentive motivation derives from external sexually salient stimuli, whereas drive motivation derives from an internal state. Second, most incentive approaches generally held that arousal generates desires, whereas the drive approach typically describes desire as preceding arousal (cf. Both, Everaerd, and Laan, in press, Kaplan, 1996). Third, incentive motivation assumes that the basic function of sex is some combination or of pleasure and social bonding, whereas drive motivation assumes the basic function of sex is reproduction (cf. Åmgo, 1999, Kaplan, 1996). Finally, incentive motivation dominates the empirical literature and the animal literature, while drive motivation dominates the clinical literature (cf. Pfaus, 1999; Beach, 1956, 1976; Kaplan 1996; Basson, 2002, 2005).

Researchers cite two reasons for using an incentive approach rather than a drive approach. First, and famously, no tissue damage has ever been observed as a consequence of abstinence from sex (Beach, 1956), and, further, sexual behavior is also not necessarily associated with the promotion of an individual's health (Pfaus, 1999). Therefore viewing sexual motivation as a homeostatic process is not useful. Second, women's sexual behavior is, by and large, unrelated to their fertility, further reducing the utility of a "drive" view of sexual motivation. Although there is evidence that women's sexual behavior changes predictably around ovulation (Graham, Janssen, and Sanders, 2000;

Krug et al, 2000; Slob et al, 1996), women, unlike most female animals, can engage in and even pursue sex without reference to their fertility. One might argue that motivation need not be biological (i.e., reproductive) to be a drive, but might be social (i.e., one of the many hypothesized social functions of sex for women [Wallen and Zehr, 2004; Hrdy, 1999; Hill and Preston, 1996; Symons, 1981; Trivers, 1972; see Wolff and MacDonald, 2004 for a review]) and still be a drive. However, no connection has been observed between socially motivated behavior and an aversive internal state. For these reasons, the present project treats sexual motivation as an incentive process.

Freud's theory of psychosexual development had two components of primary relevance here. First, the triumvirate of id, ego, and superego represented different levels of motivation. Id is pure want, an infant's primary process, or what we might loosely describe now as "drive." The superego, conversely, consists of acquired, rather than innate, motivations, typically social. The two are mediated by the ego, which explores the environment for stimuli to satisfy the organism's needs – i.e., secondary process. The second relevant aspect of Freud's work is psychosexual stage theory. He proposes that humans pass through a series of stages – the oral, anal, phallic sage, latent, and genital stages – intercourse being the most mature, best adjusted expression of sexuality.

Empirical and theoretical responses to Freud were many and varied. John Bowlby's attachment theory (1969) proposed that emotional bonding, and not sex per se, was at the heart of infant behavior. He distinguishes attachment, sex, and parenting behaviors from those related to hunger and thirst insofar as the first group is social behaviors, whereas the second is individual. He presents this distinction to make the case for more "primary drives" than the purely physiological. Thus he includes sex, parenting,

and infant attachment as innate *social* motivational systems. Maslow developed the hierarchy of needs, a motivational theory which contrasted "deficit motivation" with "growth motivation." Maslow (1943) includes sex in both the basic needs (along with hunger and thirst) and in the love needs (pp. 372-2 and 381), but no other basic needs are included at the higher levels of need. Skinner found the very notion of inferred motivation to be "bankrupt" (Nader, Bechara, and van der Kooy, 1997, p. 86), and developed an empirical approach which ignored an organism's internal state. Looser interpretations of behaviorism generate a great deal of animal research on motivation, as different internal states in the presence of identical external stimuli give rise to different behaviors (see Toates, 1986, for a review of motivation research on rats, and a systems theory interpretation of those studies).

Beach's model of animal sexual motivation divided male sexual response into two mechanisms, the sexual arousal mechanism (SAM) and intromission and ejaculatory mechanism (IEM) (Beach, 1956). SAM was characterized by great flexibility and influence by learning, whereas IEM tended to be stereotyped. We might loosely parallel IEM with Freud's "primary drive" and SAM with "secondary drive," as a set of behaviors which emerge from learning. His later work (1976) divided female sexuality into three parts: attractivity, proceptivity, and receptivity. The first is the female's non-behavioral signals of reproductive value or fertility. Receptivity is whether or not she consents to the sexual advances of others. Proceptivity is the female's behavior of seeking sexual contact, and it was an important innovation in models of female sexual behavior. Importantly, Beach specified that these described female mammals in *estrus*, and the status of human female estrus is an area of contention (Dixson, 1998, pp. 93-4).

While females of many species exhibit clear signs of fertility, such as swollen, red genitals, human females have no such obvious visual cue. Subtler changes in female physiology and morphology have been observed, such as an increase in the symmetry of soft tissue (Scutt and Manning, 1996; Johnston, Miles, et al, 2005) and different conditioning of sexual response (Hrdy, 1999; Slob, Bax, Hop, Rowland, and van der Werff, 1996), along with changes in behavior, such as increased risky social behavior (Broder and Hahman, 2003), wearing more revealing clothing (Grammer, 1996) and more frequently engaging in extra-dyadic sexual behaviors (Gangestad and Thornhill, 1998; Penton-Voak et al., 1999). Whether or not such changes constitute "estrus" is unclear (see Tarín and Gómez-Piquer, 2002). Beach's model of male sexual response was followed by many new and different models of male sexual response (see Pfaus, 1999). The female model has remained more or less intact (e.g., Avitsur and Yirmiya, 1999) and turns up in alternative clinical models of women's sexual response (Basson, 2002).

In the mid-1960s, both Hardy (1964) and Whalen (1966) proposed theories of human sexual motivation. Hardy's (1964) "appetitional theory" of sexual motivation was developed as an alternative to drive theories. He proposed that motives derive from learned expectation that the individuals actions result in hedonic change. That expectation is reinforced through repetition. The more certain, positive, and immediate the reward, the stronger the motivation. The activation of a motivation depends on the presence of incentive cues, combined with the strength of the affective change.

Whalen's (1966) "energetic" model consisted of two components: arousal and arousability. It is more or less a trait-state theory, with arousal conditionable by sexual gratification – i.e., "the reinforcement, reward or 'pleasure' associated with or caused by

sexual activities" (p. 152). Arousability is defined as "the threshold for erotic stimulation, regardless of whether the threshold in question is one of peripheral tissue sensitivity or of central neural sensitivity" (p. 157). It is mediated by hormones and by feedback of sexual activity, such as post-ejaculatory inhibition. Both of these are incentive motivation theories, since they propose that it is an external cue, not an aversive internal state, which generates motivated behavior.

Toates (1986) also describes motivation as a process of the interaction of the organism with the external incentives. Describing primarily empirical studies with rats, he outlinesr a systems approach to motivation which emphasizes the interaction between the internal mechanisms of the individual and external factors. Singer and Toates (1987) further elaborate an incentive theory of sexual motivation. They propose that the hedonic quality of an incentive may be diminished by consummation or increased by abstinence; sexually relevant stimuli are more motivating after deprivation (abstinence) than after satiation and novel stimuli will be more "palatable" than stimuli to which an individual is habituated. An individual's deprivation state interacts with the hedonic quality of an incentive to generate the ultimate motivational state. Characteristic behavior mediates the individual's motivational state and the environmental incentive.

Basson (2000, 2002, 2005) discusses a women's sexual motivation as their reasons for having sex. "Women's sexual motivation is far more complex than simply the presence or absence of sexual desire (defined as thinking or fantasizing about sex and yearning for sex between actual sexual encounters)" (2005, p. 1327). Elsewhere she specifies such reasons as enhancing emotional intimacy and commitment, attractiveness and attraction (Basson 2002, p. 18). In this model, sexual behavior begins with *nonsexual*

desires which may be met through sexual contact. From there, the women make a "deliberate choice to experience stimulation" (p. 53), which generates arousal, and the arousal in turn generates sexual desire, a craving for sexual sensations for their own sake. Arousal is more mental than physical and may be followed by an orgasm, multiple orgasms, or no orgasms at all, depending on the type of stimulation (2000, p. 51). Interestingly, she divides women's sexual behavior along the same lines as Beach – initiation and receptivity – but suggests that women, whose sexuality is less straightforwardly reproductive then men's (due to women's sporadic fertility), are also motivated by desire for intimacy and shared pleasure, and not strictly by a desire for sex.

The dual control model of sexual response offers an alternative framework for thinking about sexual motivation. The theory runs this way: humans exhibit excitation and inhibition impulses in the central nervous system in response to sexually relevant stimuli (Bancroft, 1999; Bancroft and Janssen, 2000). While the actual inhibition of anatomical arousal happens peripherally, in the sympathetic nervous system, the mechanism implicated in the management of this inhibition is the central nervous system, with emphasis on the limbic system, though precisely how this mechanism works is far from straightforward. Conceptually, this central control mechanism is organized in terms of a sexual inhibition system (SIS) and a sexual excitation system (SES). Individuals posses an inhibitory "trait," (Bancroft, 1999, p. 779) and an excitatory trait, and may be expected to vary in this trait, exhibiting different propensities for inhibition or excitation. The individual's propensity for inhibition or excitation may give rise to sexual dysfunction or sexual risk taking (Bancroft, 1999; Bancroft, Janssen, Carnes, Strong, Goodrich, and Long, 2004; Bancroft, Janssen, Strong, Carnes, Vucadinovic, and Long,

2003; Janssen et al., 2002a; Janssen et al, 2002b). Bancroft (1999) describes four types of inhibitory response patterns:

1. In response to a perceived non-sexual threat. This will activate the BIS [General Behavioral Inhibition System, see (Gray, 1987) …] and increase general arousal and at the same time activate the SIS to inhibit sexual arousal and genital response (as well as activating inhibition of other unwanted response patterns such as feeding).
2. In response to a perceived sexual threat. The principal difference from (i) is that the sexual threat will be derived from or be associated with a sexual stimulus which will also activate the SES. Whether a sexual response then occurs will depend on the balance between SIS and SES. In the presence of a weak SIS, sexual response may not only occur but also be augmented by the effects of the threat-induced general arousal increase (i.e. excitation transfer). A further possible difference from (i) is that there may be direct activation of SIS rather than activation via BIS.
3. Chronic stress (and possibly depression) will enhance SIS (in vulnerable individuals) and possibly impair SES. General arousal may or may not be increased.
4. Ejaculation will enhance SIS resulting in the postejaculatory refractory period (or in the case of repeated ejaculations in a short time period, sexual 'satiation') (Bancroft, 1999, p. 780).

A questionnaire developed on the basis of the dual control model of sexual response differentiated between two distinct inhibitory mechanism: SIS1, inhibition due to fear of performance failure, and SIS2, inhibition due to fear of performance consequences (Janssen et al, 2002a). These consequences include unwanted pregnancy, disease transmission, and negative social consequences (Janssen et al, 2002a). Lower SIS2 in men has been positively correlated with a greater number of anonymous sex partners and a greater number of partners with whom one has not used a condom (Bancroft, et al, 2004). SIS2 is proposed to be a "'context' or 'stimulus' specific" function (Bancroft and Janssen, 2000, p. 572), which parallels the environmental dependence exhibited in mammal motivational systems like hunger and thirst (cf. Toates, 1986).

The nature of the SES is less explored, but is taken to be a trait which is sensitive to sexual stimuli in the environment, depending in part upon hormonal factors. Low SES appears to be implicated in erectile dysfunction, but not premature ejaculation in men (Bancroft, Herbenick, Barnes, Hallam-Jones, Wylie, Janssen, BASRT, 2005).

Gender Differences

The dual control model was initially built on evidence from men. How does female sexuality vary from males'? Men's and women's sexual response appear to differ in two ways relevant to this project: oscillation with the menstrual cycle, accompanied by the capacity to have sex without reference to hormonal state; and conditionally higher inhibition or lower excitation. One proposed cause of these differences is differential parental investment. This section describes differences in human parental investment, and then discusses differences in men's and women's sexual motivation, and outlines a theoretical framework for understanding these differences as the result of parental investment in shaping the sexual excitation systems and sexual inhibition systems.

Parental Investment. The classic theoretical account is that parental investment is expenditure of resources on offspring for their survival at the expense of other offspring, beginning with metabolic expenditure in producing gametes and extending to caretaking behavior (Trivers, 1972). If males and females invest differently in offspring, then the operation of sexual selection will be shaped by that difference, due to differential cost/benefit profiles between males and females (Trivers, 1972; Maynard Smith, 1977). In the case of humans, differential parental investment is *prima fascia* plausible, given the female's 40 weeks of pregnancy, potentially fatal childbirth, and possibly years of breastfeeding and caretaking, compared to the male's biological investment of a single

ejaculation. Additionally, since a man does not have the biological evidence of fatherhood that a woman has of motherhood, men face the possibility of being cuckolded. This is the tradeoff men face: putting energy into existing offspring of whom one has no guarantee of paternity, or putting energy into attempting to mate with additional females (Trivers, 1972; Gangestad and Simpson, 2000). Since men have both less biological investment and less parental certainty, they benefit more than women from a strategy of promiscuity, whereas women, with high parental investment and high parental certainty, benefit more from a more selective mating strategy.

Trivers' proposition that parental investment changes an organism's cost/benefit profile for sexual behavior has influenced a great deal of evolutionary psychological theory on the subject of the human female mate choice and sexual behavior (Buss, 1989; Hrdy, 1999; Miller, 2001; Symons, 1982; Gangestad and Simpson, 2000). However, the classic theory is not a description of human parenting behavior. Trivers proposes a clear distinction between parental and non-parental reproductive effort, and for women this is a false dichotomy. During the many years of immaturity of her offspring, a woman may incorporate sexual behavior as a mechanism for protecting her young and herself. Non-conceiving sexual contact can have benefits for a woman and her offspring. Other possible functions of sex include reinforcing her social network (Wallen and Zehr, 2004; Hrdy, 1999; Hill and Preston, 1996), creating a gateway to a new partner (Buss, 1994), and obfuscating paternity (Symons, 1972; Trivers, 1972), among others (see Wolff and MacDonald, 2004). In these cases, a woman's "mating" (i.e., sexual) behavior may function more as parental behavior. These social functions of sex are paralleled in

women's biology by the decoupling of sexual receptivity and proceptivity from ovulation. Sex for women is not driven by gametes alone.

Thus what might traditionally be construed as reproductive behavior – i.e., promoting conception of new offspring – often functions more as parenting behavior – i.e., promoting the welfare of existing offspring. This has important implications for sexual motivation, since we can hypothesize that female sexual motivation systems were shaped evolutionarily not only by the role that sex plays in the *production* of children, but also by its role in the *protection* of children (Hrdy, 1999; Soltis and McElreath, 2002; Geary, Vigil, and Byrn-Craven, 2004). The following two differences between men's and women's sexuality are interpreted in terms of women's incorporation of sexual behavior into parenting behavior, along with their higher parental investment and parental certainty.

Female sexuality oscillates with hormone levels, but is not dependent on hormones. While it is not clear what distinct role any particular hormone plays in increasing sexual desire (Levin, 2002), evidence from humans and other primates indicates that female sexual desire is influenced by hormones, which oscillate with the menstrual cycle (Wallen, 2001). Primate females including humans appear to be somewhat more sexually motivated around their fertile phase (Wallen, 1990; Wallen and Zehr 2004). It also seems that cycle phase influences conditioning of sexual response (Graham, Janssen, and Sanders, 2000; Hrdy, 1999; Slob, et al, 1996), and possibly a variety of other behaviors, including clothing selection (Grammer, 1996), risky social behavior (Broder and Hahman, 2003), extra-dyadic sexual behaviors (Gangestad and Thornhill, 1998; Penton-Voak et al., 1999), and a change in preference for masculine

facial features (Penton-Voak et al., 1999) and major histocompatibility (Gangestad and Thornhill, 1998). Wallen's (2001) review of the data also included some evidence of increased initiation by women at ovulation, as well as increased masturbatory behavior. It should be noted that other research has found no difference in desire, either genital or subjective, in women across the menstrual cycle (for example, Meuwissen and Over, 1992).

Yet it is also the case that female sexual motivation is not dictated by hormones, a crucial factor in the function of sex for other purposes than conception (Wallen, 2004, 1990; Dixson, 1998). In fact women's sexual motivation appears to be *less* influenced by hormones than men's (Bancroft, 2002). In women, as in most female primates, sexual behavior is not governed strictly by hormones, but rather appears to have a substantial element of social contingency (Wallen, 2001; Dixson, 1998), a critical factor in liberating female sexual behavior from purely reproductive functions.

The parental investment explanation for primate females' oscillation with the menstrual cycle, along with their freedom from hormonal regulation of their capacity to have sex, has been explored in some depth by Wallen (1990; 2001; Wallen and Zehr, 2004):

> Sex when the female is nonfertile would have little evolutionary consequence if sex also occurred during fertility. Thus, the couple of increased sexual motivation with peak fertility through changes in the same hormones increases reproductive success and still allows the occurrence of sexual behavior in nonreproductive contexts. A reliance upon sexual motivation as the mechanism coordinating fertility with sexual behavior produces a less tight coupling between hormonal and behavioral change in primates than that seen in nonprimate species…The maintenance of nonreproductive mating requires some selective advantage to offset its small increased cost…. What this selective advantage might be is unclear, but one possibility is that it enhance social affiliation between males and females. (Wallen and Zehr, 2004, p. 104).

They go on to describe perpetual sexual potential as "social cement" (p. 104). It is also plausible that this perpetual affiliation has significant impact on reproductive success in terms of parental investment, either (or both) by facilitating increased paternal investment or in serving as maternal investment, as described earlier. The oscillation promotes sex when a woman is fertile, while the freedom from strict hormonal governance allows for social (including maternal) functions of sex.

A sexual motivation mechanism influenced but not determined by hormones would parallel biological shifts such as the changes in cervical mucus which promote the passage of sperm and the change of direction in the contractions of the uterus (Lloyd, 2005) and behavioral changes such as wearing less clothing (Grammer, 1996), being more active (Broder and Hahman, 2003), and having more extra-dyadic sex partners (Gangestead and Thornhill, 1998; Penton-Voak et al., 1999).

In terms of the dual control model, this would suggest an oscillation of excitation or inhibition systems. For example, it may be that women experience a periovulatory drop in SIS, increasing their engagement with sexual stimuli in the environment and decreasing behavioral inhibition. Such a shift may be obfuscated in naturally cycling women by other factors such as stress or relationship status. Psychophysiological experiments can explore this possibility.

Female sexual response is more inhibitory. To begin with, women test with lower SES scores and higher SIS scores on a SIS/SES survey instrument than men do (Carpenter, 2002; Graham et al., 2004). Recent brain imaging research has found that women exhibit markedly less limbic activity when viewing sexually explicit images than do men (Hamann, Herman, Nolan, and Wallen, 2004). Research on women's sexual

desire indicates that they are less likely to experience "spontaneous sexual desire" (Levin, 2002, p. 406). Bjorklund and Kipp (1996) argued for a wide array of greater inhibitory tendencies in human females, including cognitive, emotional, and kinetic. Yet females do not have domain general inhibition – for example they have a lower threshold in brain activation in response to non-crisis infant cries than do males (Hrdy, 1999). Moreover, women's physical arousal can be elicited with more general, less specific sexual information (Chivers and Bailey, in press), which indicates lesser, rather than greater, inhibition. It is possible that the primary difference in inhibition lies not in physiological arousal, but in the affective and cognitive mechanisms which organize the meaning of the arousal (Everaerd, Laan, Both, and van der Velde, 2000; Laan, Everaerd, van der Velde, and Geer, 1995).

Thus human sexual motivation is different across genders insofar as the function and *consequences* of sexual behavior are different across genders, in accordance with parental investment. Bancroft's (1999) four types of inhibitory response patterns serve as the theoretical framework for this proposition, since the differences between human males and females influence at least two of these responses in a predictable and categorical way. Namely, perceived sexual threat (Bancroft's inhibition response 2) and the refractory period (Bancroft's inhibition response 4) are necessarily different in women, given their differing parental investment and sexual anatomy and physiology. Regarding the latter, females by and large do not ejaculate, and it is ejaculation *per se*, rather than orgasm, which triggers the refractory period.

Regarding the former, "perceived sexual threat" includes threats "derived from or … associated with a sexual stimulus, which will also activate the SES" (Bancroft, 1999,

p. 780). This has been operationalized in men as SIS2, inhibition of sexual arousal in the face of risk or threats, such as unwanted pregnancy, disease transmission, and negative social consequences (Janssen et al, 2002a). If SIS2 represents, at least in part, an individual's perception of the risk of unwanted pregnancy, then human females, according to parental investment theory, should have a reliably higher SIS2 than do men, *ceteris paribus*, since the biological risk associated with conception is, evolutionarily speaking, substantially higher for them.

Herein lies the link between the dual control model of sexual response and parental investment: with greater risk may come greater inhibition, and while we observe variation across individuals, we will also observe variation across genders. Women are more at risk for sexually transmitted infection due to biological factors (UNFPA, 2002). Pregnancy also is a greater risk for women, given their larger biological investment, and so, the hypothesis runs, they have evolved a greater SIS2 – proneness to inhibition of sexual response in the face of negative consequences. Thus it appears women's sexual response in terms of their inhibition and excitation has been shaped by parental investment to have overall lower excitation and higher inhibition.

Working Definition and Operationalization

Definition. The preponderance of theory supports the position that sexual motivation is derived from the interaction between an organism's internal mechanisms, both excitatory and inhibitory, and stimuli in the environment. These stimuli may be broadly defined and include both reproductive cues and social cues. This notion of *interaction* is crucial to modeling sexual motivation (Savage, 2003).

Operationalization. For the purposes of this project, "sexual motivation" constitutes an agent's sexual inhibitory (SIS) and excitatory (SES) mechanisms which motivate sexual behavior, SIS functioning as behavioral inhibition and SES mediating an individual's sensitivity to sexually relevant stimuli. Abstinence does not produce aversive affect, but does increase the palatability of incentives (Singer and Toates, 1987); and women will exhibit overall higher levels of inhibition and lower levels of excitation than men (Graham et al, 2004; Bjorklund and Kipp, 1996). Some individuals have high trait levels of sexual excitation or inhibition, and others have low trait levels of either. The term "sexual motivation profile" or phenotype (SMP) refers to the particular combination of inhibition and excitation that an individual possesses. High sexual excitation with low sexual inhibition appears to be associated with HRSB (Bancroft et al., 2003, 2004).

Is It an Adaptation?

While social norms and biological and hormonal factors influence sexual motivation, findings from the dual control model of sexual response suggest that sexual motivation is a trait, more or less stable across an individual's lifespan. If this is the case, the question of heritability arises. Inherent in the idea of a genetic basis to sexual motivation are two difficult propositions: first, that "sexual motivation" is a *trait*, per se, and second that it is an *adaptation*. (Of course not all traits are adaptations, but theorists of sexual response and sexual motivation often couch their arguments in terms of evolutionary adaptiveness; Beach, 1956; Pfaus, 1999; Bancroft, 1999; Kaplan, 1996.) As to the first, no sexual science attempts to establish whether or not risk taking is a trait or "character" – i.e., "a correlated set of features caused by a single developmental or

ecological process" (Fristrup, 1992, p. 43) – or even a clearly established definition for what that trait is (Bancroft and Vukadinovic, 2004).

As to the second, establishing that a trait is an adaptation requires four different types of evidence: (1) variation in the trait has a genetic basis; (2) the trait influences reproductive success; (3) researchers can outline how the trait influences reproductive success; and (4) experimental manipulation of the trait or the environment should generate predictable variations in behavior and reproductive success (Sinerva and Basolo, 1996). The case for each for each of these type evidence follows.

Variation in the trait has a genetic basis. There is evidence that elements of human sexual response are heritable; two studies on women's orgasms show that between 30-45% of a woman's orgasmic capacity may be attributed to genetic factors (Dunn, Cherkas, and Spector, 2005; Dawood, Kirk, Bailey, Andrew, and Martin, 2005). Further research suggests that some elements of emotional systems are in part genetically heritable, including depression and suicidality (Glowinski, Bucholz, Nelson, Fu, Madden, Reich, and Health, 2001). Tying together these two bodies of evidence is the research establishing a genetic basis for sensation seeking and impulsivity (Hur and Bouchard, 1991; Depue and Collins, 1999; Hollander, Rosen, 2000; Isles, Humby, Walters, and Wilkinson, 2004), which appears to be closely associated with sexual compulsivity (Bancroft and Vukadinovic, 2004; Gaither and Sellborm, 2003; Reece, Plate, and Daughtry, 2001; Janssen et al., 2002a; Janssen et al, 2002b). However, importantly, there is currently no direct evidence that sexual compulsivity per se is genetically heritable, and not all those who propose individual proneness argue explicitly for a genetic basis to individual variability. The dual control model of sexual response (Bancroft, 1999;

Bancroft and Janssen, 2000) posits that individuals posses an inhibitory "trait," (Bancroft, 1999, p. 779), but these authors do not directly assert a genetic foundation.

The trait influences reproductive success. Evidence regarding the influence of sexual motivation on reproductive success has not been established, and only a theoretical argument can be made, based on the concept of frequency dependence, discussed above. Sexual motivation, hypothesized as being a trade off between risk avoidance and reproductive success, may be a frequency dependent trait, where the relative risk or reproductive benefit of a particular sexual motivation strategy (i.e., high-risk/sensation seeking or low-risk) depends on the representation of an individual's strategy in the local population.

Researchers can outline how the trait influences reproductive success and experimental manipulation of the trait or the environment should generate predictable variations in behavior and reproductive success. No evidence exists to establish a correlation between high versus low sexual motivation, so researchers have not outlined how the trait would influence reproductive success.

In summary, a case can be made for the genetic heritability of sexual motivation, but the evidence is incomplete. Further research on the genetic basis and reproductive significance of sexual motivation will help clarify the issue. The present project may contribute a substantiation of the plausibility of the heritability of sexual motivation, particularly as a frequency dependent trait.

HIV

Human Immunodeficiency Virus (HIV) is the virus which causes Acquired Immune Deficiency Syndrome (AIDS). It is carried in an individual's blood, semen, pre-ejaculate, vaginal secretions, breast milk, and, to a lesser degree, saliva and tears (Centers for Disease Control [CDC], 2003). It is transmitted by contact with an infected person's blood, semen, pre-ejaculate, vaginal secretions, or breast milk. HIV does not survive outside the human body, and therefore it is not transmitted by environmental surfaces, insect bites, or casual contact (CDC, 2003a). Risk of infection per heterosexual contact is estimated between 0.0003 to 0.0014 in the United States (see for Royce, Seña, Cates, and Cohen, 1997 a review). Biological factors which increase the risk of transmission include pre-existing infection, which weakens immune functioning and potentially causes abrasions or tears in mucus membranes (CDC, 2003c) and sex – women are estimated to be twice as likely to contract HIV via heterosexual intercourse, compared to men (UNFPA, 2002).

Once an individual is infected with HIV, he or she is infectious almost immediately, though antibodies are not detectable until approximately a month following infection. Soon after infection, the individual experiences a flu-like illness, and then enters an incubation period. Without treatment, HIV degrades the immune system over a period of, on average, 10 years, until immune functioning no longer resists infectiousagents (CDC, 2003b).

High Risk Sexual Behavior

The discussion of sexual risk will localize around HIV risk in particular, since that is ultimately the risk assessed in the model. HRSB is characterized by the interaction of an individual's biology with the environment, mediated by that individual's behavior and

the behavior of elements of the environment, particularly the individual's conspecifics. As such, behavioral, psychological, and environmental factors shape the risk associated with an individual's sexual motivation. The goal of this section is to elaborate on known behavioral, psychological, and system risk factors, with the aim of highlighting the utility of the concept of frequency dependence as an influence on sexual risk.

Behavioral Factors

For the purposes of the present model, the discussion of high-risk contact is sexual behavior will be limited to those which pose a heightened risk of transmitting HIV. Other factors – e.g., other STIs, unwanted pregnancy, and negative social consequences – certainly are potential risks of sexual behavior, but for simplicity the present model focuses on HIV-like infection, since this is among the most pressing issues in sexual health. Rather than a global focus on risk of infection or risk of unwanted pregnancy, HIV risk behaviors narrow the range of target behaviors, eliminating irrelevant or superfluous variables. Several criteria increase a behavior's risk of HIV transmission: (1) one or both partners is HIV+ or does not know his or her HIV status, (2) partners do not know each others' status; (3) partners engage in unprotected penile-anal or penile-vaginal intercourse (including intercourse with a condom used incorrectly); (4) one or both partners has other partners with whom they have unprotected intercourse – the greater the number of partners, the greater the risk (MMWR, 2004; Anderson and May, 1988); (5) partner concurrency, as opposed to serial monogamy (Doherty et al, 2005); and (6) the seronegative partner has a preexisting sexually transmitted infection (CDC, 2003c).

Sexual Compulsivity

For present purposes, sexual compulsivity is defined as disruptive or uncontrollable sexual desire, which, when acted upon, serves to manage negative emotional states such as anxiety or depression (Kalichman & Rompa, 1995; see Bancroft & Vukadinovic, 2004 for discussion). Sexual compulsivity is associated with the risk behaviors identified above. For example, HIV+ MSM with higher sexual compulsivity are more likely to engage in the higher risk sexual behavior of penetrative intercourse (whether receiving or inserting) (Reece, 2003). This group reports that their penetrative intercourse is most often with partners of unknown serostatus. Additionally there is a correlation in HIV+ MSM between sexual compulsivity and a low perceived sense of responsibility for reporting HIV serostatus (Reece, 2003). Among heterosexual college students, those scoring high on measures of sexual compulsivity reported higher levels of involvement in non-exclusive sexual relationships (Dodge, Reece, Cole, and Sanfort, 2004). In one study, all of the heterosexual men who score highest on measures of sexual compulsivity reported having been the insertive partner in unprotected penetrative intercourse, often with a partner of unknown serostatus, while only 40% of heterosexual men overall reported having been the insertive partner in unprotected intercourse (Reece, et al, 2001). Measures of sexual compulsivity also correlated in heterosexuals with number of one-night stands, number of partners in the prior three months, and, among women, number of unprotected sexual encounters, number of unprotected one-night stands, and receptive anal intercourse (Gaither and Sellbom, 2003). Gaither and Sellbom (2003) found strong correlations between scores on standard measures of sexual compulsivity and the dual control model's sexual excitation and sexual inhibition related to performance consequences.

In the dual control model of sexual response, sexual compulsivity is associated with high excitation combined with low inhibition. In men, this sexual motivation profile has been positively correlated with a greater number of anonymous sex partners and a greater number of partners where a condom was not used (Bancroft, et al 2004).

Interestingly, the dual control model, which proposes that individual high on SES and low on SIS2 are most prone to risk-taking, due to sensitivity to environmental cues and a lack of behavioral inhibition, maps well onto Depue and Collins's (1999, p. 496) plotting of personality traits in which high "extroversion" (excitation) and low "constraint" (inhibition) interacted to generate the "Impulsivity-Sensation Seeking cluster." Again, this model appeals to genetic roots of personality (pp 505-6). Indeed, the frequent recurrence of inhibitory and excitatory dual control theories (Bancroft and Janssen, 2000; Kaplan, 1996, p. 17; Toates, 1987), along with its basic importance in the neurobiology of the nervous system (Rashevsky, 1971) suggests that it is a particularly valuable framework for understanding sexual motivation.

Preferential Attachment

Risk pools among high risk individuals, such as those who score high on scales of sexual compulsivity, as described above. Preferential attachment, a system process where nodes of a network preferentially connect with nodes which already have a greater number of existing connections, can generate pooling or "hubs" (Liljeros, Edling, Amaral, Stanley, and Aberg, 2001). Preferential attachment systems generate scale-free distribution of number of sex partners found in real human systems.

What is the nature of the preferential attachment which might be at work in human sociosexual systems? In non-human animal systems, a small number of males

have a large number of female partners, while females have a less variability in number of partners (e.g., Seeman and Nahrung, 2004). This may be seen as a rather straightforward instantiation of preferential attachment: the highly successful males are those with the highest perceived mate value, and thus are favored in both intrasexual competition and intersexual choice. While humans have greater variability in number of partners among men compared to women, evidence does not exist to suggest that this is related to any measure of mate value. This is further complicated by the high representation of men who have sex with men among those with the most partners; mate value in same sex sexual attraction is an ill-studied area. More evidence would be required to substantiate claims about mate value's relationship to number of sex partners.

However, since there is a relationship between a high number of partners and sexual motivation (i.e., SIS and especially SES; Janssen et al, 2003), this suggests that preferential attachment may be an interaction between sexual motivation and the presence of appetitive stimuli, which is mediated not by the attractiveness of mates, but rather the individual's ability to find sufficiently attractive individuals who find that individual sufficiently attractive. Since particular physical spaces in human ecological systems do act as hubs of sexual contact (such as those identified at websites like cruisingforsex.com), it may be that preferential attachment in human sexual networks is a function of landscape interacting with an individual's SES.

Sexual Risk Taking and Evolution

Theoretical evolutionary biology has examined the influence of parasite transmission on the evolution and maintenance of sexual reproduction as an adaptation, including the hypothesis that sex is an adaptation to resist parasites (Hamilton, Axelrod and Tanese, 1990), the risk associated with mating, given the risk of infection (Kokko, Ranta, Ruxton, and Lundberg, 2002), and how STIs have exerted selection pressure on mating systems (Thrall, Antonovics, and Beyer, 2000; Thrall and Antonovics, 1997). Boots and Knell (2002) note that the results of these models point toward the evolutionary benefit of monogamy, and yet truly monogamous species are rare. Their hypothesis is that the non-reproductive benefits are sufficient to outweigh the risk of promiscuity, as discussed for females above. In a mathematical model, they compare the reproductive fitness of a "risky" versus "safer" mating strategies, to find that "only when the increased likelihood of infection from risky behaviour is minimal and the benefits are equivalently small… will we get the exclusion of the risky strain" (p. 587). A difficulty with this study is that it assumes that more frequent mating behavior amounts to more frequent reproduction, which in humans is not the case (Lloyd, 2005). However, the cost-benefit approach to risky versus safe mate choice behavior establishes that risky and safe strategies can co-exist when the benefit of reproduction is sufficiently high to outweigh the cost associated with risk of infection.

Experimental evolutionary biology has also looked at the influence of sexually transmitted organisms (STOs) on mating, but generalization of comparative data is not always reliable, since humans and other animals, even other primates, vary in sociosexual systems. For example, in the Chapius beetle females are more likely to be infected with a

STO, but females also have more uniform mating success (i.e., number of partners), while males have greatly varied mating success (Seeman and Nahrung, 2004). Such sex differences are found in many species, but not necessarily in humans. While, as in many species, human females have reliably fewer partners, the distribution of number of partners follows the same pattern in men and women (Liljeros, Edling, Amaral, Stanley, and Aberg, 2001); yet it is arguable that the smaller scale of the distribution among women equates to "more uniform" behavior. Inarguable is the mutual mate choice structure of humans, as compared with the majority of species which exhibit female choice with male intrasexual competition (Cronin, 1991). Furthermore, male homosexual contact is not a significant feature in many non-human mating systems, but this is among the primary modes of transmission of HIV. Thus again, the distribution of sexually transmitted disease in humans will not necessarily parallel that of other species. Further, while population density is a known correlate of parasite density, regardless of species (Nunn, Altizer, Jones, and Sechrest, 2003; Low, 1999), comparative studies of parasite-host relationships do not account for such highly human constructs as social norms, which are known to correlate with STI transmission (see Bandura, 1994).

Poverty and other forms of social oppression, such as gender disparity and racial prejudice, also influence risk (Bates et al., 2004; Zierler & Krieger, 1997; Zierler et al., 2000; CDC, 2004). Furthermore, higher-level self-regulation functions do not develop until the mid-twenties (Steinberg, 2004), though humans reach sexual maturity more than ten years sooner. This discrepancy generates a window of vulnerability, where adolescents are more prone to risk taking. Such issues are beyond the scope of the present project, since the model is designed only to determine whether sexual landscape, not

resource scarcity, social oppression, or other factors affect risk. “Risk behavior” is assessed in terms of probability of infection. Agents lack preventive measures such as communication with their partners, awareness of their infection status, or condom use. Consequently the model addresses only the influence of SMP on risk, rather than any higher level cognitive capacities or larger social constructs.

Summary

In describing the literature related to the present model, theoretical, empirical, and methodological issues have been reviewed. Sexual motivation has been defined for this project as an individual’s sexual inhibitory (SIS) and excitatory (SIS) mechanisms which motivate sexual behavior. These mechanisms do not co-vary, and an individual’s combination of SIS and SES constitutes their sexual motivation profile (SMP). In humans, the traits – SIS and SES – vary across genders. Substantial evidence must be generated to establish that SIS and SES are adaptations, an assumption fundamental to the present project. Unlike drive mechanisms, sexual motivation increases not with deprivation but with stimulation by a sexually relevant incentive.

High risk sexual behavior appears to be, in part, a product of an SMP of exceptionally high sexual excitation and low inhibition, but it is also influenced by biological and social factors. Women are more vulnerable than men, making an otherwise identical choice (e.g., to engage in unprotected penile-vaginal intercourse) more risky for a woman than for a man. Further, those without access to health care, education, or within the context of oppressive sexual norms are at greater risk than those who are not.

The goal of the present project is to establish the plausibility of the idea that risk may also be a product of the SMPs of others – i.e., that it is frequency dependent.

Frequency dependence is a quality of a trait where its adaptiveness is dependent on the instantiation of that trait in conspecifics. ABM provides a valuable method for modeling frequency dependence, insofar as it allows for the modeling of autonomous interaction of heterogeneous agents. Models of mating behavior and disease diffusion have established a body of work that can inform the methodology of the present project.

Chapter 3

METHODOLOGY

Restatement of the research problem

The research focused on building and analyzing an agent-based model of disease diffusion in order to explore the hypothesis that the relative risk associated with an individual's "sexual motivation profile" (SMP) is affected by the distribution of strategies represented in the population – that is, that sexual motivation functions as a frequency dependent trait. Using an agent based model, this project created an artificial environment where agents with different genders, mate values, and levels of sexual excitation and inhibition made decisions about whether or not to engage in sexual activity. Experiments in this environment revealed how the distribution of SMPs can influence the risk associated with any individual profile. Analysis emphasized the influence of system-level factors on individual risk.

Agent Based Modeling

Agent based modeling is useful for this model of sexual motivation, given this project's emphasis on the interaction between incentive properties and internal mechanism. Savage (2003) identifies this interactionist perspective as not only the only tenable model of motivation, but also a key quality which makes ABM useful as a mode of testing theories of motivation. Because it allows for heterogeneous, independent agents which have both "internal" motivational mechanisms and incentive qualities which influence their ability to obtain incentive objects, ABMs can generate highly complex, realistic models of motivation.

The two main questions in this project were:

1. Does the relative risk associated with any given sexual motivation profile vary according to the representation of that strategy in the population?
2. Can agent based modeling generate global patterns of disease diffusion from local interactions?

The first asks how a characteristic of the system influences the behavior of individuals – viz., the proportionate representation of SMPs effect on an individual's risk of infection. The second asks how characteristics of the individuals influence the structure of the system – viz., the effect of individual decision making on the pattern of disease diffusion. A primary benefit of ABM is its ability to capture system-level patterns generated from individual-level behavior (Goldstone and Janssen, 2005). In other words, ABM generates "true bridging explanations that link two distinct levels of analysis: the properties of individual agents (e.g. their attributes and interactions), and the emergent group-level behavior" (Goldstone and Janssen, 2005, p. 424). Given the assumption that human social systems are complex, adaptive systems, such explanations are a valuable asset in understanding the relationship between individual risk proneness and disease diffusion.

Model Language

The model was built in C# 2.0, a modern object-oriented, strongly-typed language. C# is closely related both to Java and, to a lesser extent, C++. C# has a sophisticated garbage collector to simplify the burden of memory management for the programmer. The type safety of the language provides compiler support to make sure variables are used in valid contexts. C# 2.0 augments type safety with support for generic types, an improvement to the antiquated template system of C++. C# also has support for

tail-recursion, which allows more efficient implementation of functions that call themselves recursively.

In addition to these core language features, C# is a member of the .NET family of languages. The .NET framework provides the runtime language support to interpret C# bytecode in a manner similar to the Java runtime. It also includes the rich Framework Class Library (FCL) to provide a large amount of built-in functionality on topics ranging from regular expression parsing to user interface components. Finally, the Visual Studio 2005 development environment for C# facilitates programming with an integrated debugger, graphical class designer, visual user interface designer, context-sensitive command completion, and automated refactoring tools.

Model Parameters

Independent Variables

Gender. Agents were assigned to one of two categories: bois and goils (following Craig's flock of "boids," 2001). Agents were only attracted to agents of the other gender. The average SIS of the goils was higher than the bois', while the average SES of the bois was higher than the goils', per Carpenter (2002) and other dual control model research (Graham et al, 2004) and the predictions of parental investment theory (Bjorklund and Kipp, 1999). Operational sex ratio did not vary across landscapes; a 1:1 ratio of 5,000 bois and 5,000 goils was maintained. The genders further varied in the behavior of their SIS.

SIS. Each agent had a sexual inhibition system (SIS), an inhibition mechanism which damped sexual motivation. Bois' SIS remained constant except immediately following mating, when, for two timesteps, it increased exponentially. This spike, along

with a behavioral directive to move away from the mate, functioned as a refractory period. Two timesteps was selected as the shortest practical refractory period. It had to be short for verisimilitude, but it had to be long enough to allow the two agents to separate, or else the two agents would simply mate with each other continuously. This lock-in effect is interesting in itself with possible implications for pair bonding, but since it was not the target behavior in this model, it was avoided with this refraction function.

In goils, SIS oscillated over 100 timesteps (roughly 25 days), representing the hypothesized oscillation of female sexual interest over the menstrual cycle. It is SIS and not SES which was hypothesized to oscillate partly for simplicity – since SIS behaved in an unambiguously male-only way for bois (i.e., refraction), it was sensible for it to behave in a female-only way for goils – and partly based on interpretation of data on changes in women's sexual motivation over the menstrual cycle. The changes at ovulation enumerated in Chapter 2, (increased risky social behavior; Broder and Hahman, 2003; wearing revealing clothing; Grammer, 1996; and more frequently engaging in extra-dyadic sexual behaviors; Gangestad and Thornhill, 1998; Penton-Voak et al., 1999) can be generalized as behaviors associated with decreased inhibition – a lack of brakes – rather than increased excitation – an extra dose of fuel. The increased masturbatory behavior reported by women around ovulation (Wallen, 2001) might be an argument against this, if masturbation is viewed as a pregnancy-avoiding behavior to manage increased SES, but it might also be viewed as evidence of abandonment of social norms stigmatizing women's masturbation. The assumption that it is SIS and not SES which oscillates is thus a reasonable but not certain assumption. Exact mean values and

variability of SIS for Bois and Goils (Tables 2 and 3) were drawn directly from Carpenter (2002, p. 42).

SES. Agents' sexual excitation systems (SES) functioned as their sensitivity to sexually relevant stimuli – i.e., opposite gendered agents' mate value. In both bois and goils, SES increased incrementally across timesteps of abstinence. Upon mating, SES returned to baseline. Exact mean values and variability of SIS for Bois and Goils (Tables 2 and 3) were drawn directly from Carpenter (2002, p. 42).

Sexual Motivation Profile (SMP). Every agent possessed an assigned level of SIS and SES. Each could be set at High (H), Medium (M), or Low (L), based on the values from Carpenter (2002). SMP did not directly influence an agent's mate value, nor its susceptibility to infection. The probability of being assigned a given SMP varied depending on the sexual "landscape," which consisted of systematically varied proportionate representation of each of the SMPs.

Landscapes. Five sexual motivation profile landscapes – that is, five variations on the proportionate representation of the combinations of SIS and SES – were compared in order to assess the influence of the proportionate representation of the SMPs on individual risk (Table 4). The Risky (RISKY) landscape consisted of a high representation of higher risk SMPS. The Dysfunctional (DYSF) landscape was the inverse of RISKY. The Linear Risky (LIN-RISK) landscape exhibited a linear distribution with more high risk agents. The Linear Dysfunctional (LIN-DYS) landscape was inverse of LIN-RISK. Finally, the Normal (NORMAL) landscape consisted of a normal distribution of profiles, with the mid-risk SMP as the central profile and the high and low risk profiles in the tails.

Agent Mate Value. Agents were randomly assigned a numerical mate value (MV) on a scale from 1-10, with equal representations of every mate value and a mean population mate value of 5. Because the model treated sexual motivation as an incentive motivation system, this variable generated variety in appetitiveness of stimuli. Thus sexual decision making is influenced by the interaction of an agent's SES with the MV of the conspecific.

Agent Vision. Agents were capable of detecting stimuli in their immediate environment and also stimuli in the more distal environment, with sensitivity that degraded with distance. This feature allowed agents to search for and pursue appetitive stimuli.

Timesteps. The model advanced in timesteps, which were scaled to reflect, but not mimic, real time. One time step was approximately 6 hours. Thus, refraction, at two time steps, lasts about 12 hours, and mating, at three time steps, lasts 18. The 100 timestep SIS cycle of Goils is roughly 25 days.

Hypothetical Disease (HD). The HD was put into the system at a random location at time t1. Probability of infection (PI) was lower for boi agents than for goil agents, mirroring the differential biological infection risk of men and women (see Table 1). Rather than generating a PI based empirical data, PIs was set at .60 for Goils and .30 for Bois, proportionately realistic, but far higher than realistic probabilities of infection (Royce, Seña, Cates, and Cohen, 1997). This allowed changes in the model to happen faster.

As with HIV, there were no detectable markers for the disease; thus agents had no way to avoid infection. The infection is permanent, again like HIV, but after infection

agents continued to behave identically to those agents who were not infected. Agents died after 1,500 time steps, the rough equivalent of one year of real time. Upon agent death, another identical agent appeared, infection-free, randomly in the model. This served to maintain the proportionate representation of the SMPs while also allowing for the removal of infected agents from the system.

Tracer. The model assessed the role of a "tracer" in sexual decision making, to examine the idea of preferential attachment as a function of landscape. This functioned like a hormone tracer in ant foraging models, such as Netlogo's. Specifically, agents left a "trace" on the landscape where they engaged in sexual contact. Other agents perceived the trace, which influenced their decision making about where to go. When an agent's SES was sufficiently greater than its SIS, it incorporated the tracer into its decision process about where to find a suitable mate. It served multiple purposes in the model. First, it gave SIS something to respond to. SIS in humans functions as a "brakes" mechanism – either a handbrake, in the case of SIS-1, which responds to threat of performance failure, or a footbrake, as in the case of SIS-II, which response to threat of performance consequences. Agents without the adequate SES to SIS ratio were effectively allowing their SIS level to avoid high-likelihood sex locations. Data was generated both with and without the tracer to assess the influence of this variable on agent behavior.

Dependent Variables

Total Partners. A measured variable is the total number of partners. This was used in three ways: number of partners of each agent at the end of a run, number of partners at an agent's time of infection, and rate of accumulation of partners relative to

time of infection. Evidence from human data would predict that the greater the number of partners, the more likely the individual is to be infected.

Age at Infection. Whether and when an agent became infected with the HD was dependent on whether or not an agent has contact with an infected agent. As such it was a measure of risk, and was assessed in the context of both agent SMP, agent gender, agent mate value, and time.

Percent Infected. At the group level was assessed in terms of the proportion of infected agents in a given group over a simulation.

Model Functions

Initiation and Consent. Agents' motivation was ultimately the result of the ratio of excitation to inhibition. Without the tracer engaged, the ratio was

$$(SES_a * MV_b)/\text{ExcitationDivisor} :: SIS_a$$

where SES_a was the SES of a given agent, MV_b was the mate value of the potential partner, and SIS_a was the SIS of the given agent. Thus SES was a measure of the agent's sensitivity to the incentive value of the stimulus (viz., the conspecific), and SIS was a measure of behavioral inhibition.

Mating. If an agent initiated and the other agent consented, then the two became unavailable for behavior with other agents for three timesteps. Each added the other to its tally of partners. Then the boi agent's SIS increased exponentially for two timesteps (replicating the male refractory period), which generated a motivation differential sufficient to detach the two from each other, making them available for contact with other

agents. Mating was assumed to be equivalent to unprotected penile-vaginal intercourse. Future variations on the model may incorporate anal, oral, and protected sexual encounters, varying the relative risk associated with each behavior, but for simplicity sex was limited to one behavior.

Search. Agent vision, which allowed agents to perceive stimuli in their environment, degraded over distance. Thus agents were most likely to pursue conspecifics in their immediate vicinity, but could choose to pursue higher mate value conspecifics which are farther away. If agents had no conspecifics within their visual range, they wandered in an annealing function, which efficiently moved the agent into a populated area.

Human Subjects Procedure

Because no human subjects are used in this project, no Human Subjects protocol was observed.

Calibration of Parameters

Max Time

Max time was the total number of time steps for which the model is run. An agent lifespan after infection is 1,500. 14,600 time steps represented approximately 10 years, 7300 about 5 years, and 3550 about two and a half years, 1825 about 15 months. In addition, simulations in the General Neuter NORM landscape were run which calculated Total Partners and Age at Infection every 200 timesteps in order to determine the max time for experimental simulations. The results compared SMPs and genders on Age at Infection and Total Partners. Percent Infected was not calculated in these simulations for pragmatic reasons – it proved in the pair runs to be the least reliable risk measure and the

file in which the data were stored allowed for a limited number of columns, so Percent Infected was not calculated for the simulations which assessed time steps.

Changing max time affected the absolute values of the two risk measures, but it did not affect the relationships between SMPs or genders on either Age at Infection or Total Partners. Mean Age at Infection went down as the max time increased, but it went down at the same rate for all SMPs and both genders for 7,300 and 14,600 timesteps (Figure 2). Mean Age at Infection increased and decreased in waves following the lifecycle, (Figure 3). All lifecycles subsequent to the first exhibited lower average Ages of Infection, as a byproduct of the concentration of first infections in the first lifecycle, but since the pattern of the growth and decline followed the other lifecycles, in both boys and goils, this was taken as an indication that each of the lifecycles was essentially equivalent. Total Partners (i.e., ratio of partner to time steps) went down also, but again, it did so at about the same rate for all SMPs, though the variability across SMPs decreased for both sexes at 3,650, 7,300, and 14,600 timesteps (Figure 4).

Number of Simulations

In order to establish how many simulations were necessary for each landscape, the model was run for one, five, 10, 25, and 50 simulations at 14,600 timesteps per simulation. The goal was to find the smallest reliable number of simulations. Varying number of simulations changed the scores of the Age at Infection and Total Partners, but it did not affect relative scores across gender and SMP (Figures 5 and 6). Analysis revealed no significant difference between 10, 25, and 50 simulations in the difference between the SMPs on Age at Infection. Age at Infection was marginally influenced by number of simulations for bois, but primarily at one and five simulations (Figure 7).

Goils exhibit more variability, but the pattern of differences among SMPs stabilized at 25 simulations (Figure 7). Therefore it was established that the model would run 25 simulations per landscape.

Tracer Excitation Ratio

The tracer excitation ratio variable set the threshold for decision making in agents when the tracer was activated. In order to set this variable, the minimum, maximum, and average SIS and SES were assessed in General Neuter BASE, RISKY, DYSF, LINRISK, LINDYS, and NORMAL landscapes. The average ratios across landscapes were fairly similar, but the ratios for bois and goils were so sufficiently and reliably different that it was determined to set different ratios for the two genders. The ratio was set at a variety of thresholds in order to explore the influence of more or less selective responsiveness to the tracer. Specifically, it was set at the 50^{th} percentile – the mean ratio for each gender of 1.4 and 3.0, as well as at the 90^{th} and 95^{th} percentiles (see Table 5).

The overall robustness of model output across all these different parameter settings suggests that the model did not suffer from the fragility which afflicts some models (Gilbert and Troitzsch, 2005).

Development of Risk Rankings

In order to determine the relative representation of each profile in each landscape, three sets of preliminary runs produced results which defined the SMP landscapes of the ultimate model. First, a set of runs examined the influence of SES alone, where all agents were given the same SIS, and one third given high SES, one third medium SES and one third low SES. This was run three times – once with all agents at high SIS, once with all at medium SIS, and once with all at low SIS. Thus in each run, SIS was held constant and

SES was varied. Second, the reverse model was run, with all agents set to the same SES, but one third with each of the three levels of SIS. As with the SES-only runs, this was run three times, once each with all agents at each of the three levels of SES. SES was held constant for each run, while SIS was varied within each run. Finally, every pair combination of SMPs was run to find which was more risky relative to which. Each set of preliminary runs was examined in terms of agents' number of partners, time of infection, and Percent Infected.

The SIS-only and SES-only runs provided an initial background for establishing risk rankings. Three risk measures were assessed: Age at Infection, which measured how early in an agent's lifespan it was infected; Total Partners, which assessed the agent's total number of partners relative to lifespan; and Percent Infected, which assessed the percent of any given group which was infected. For bois, Age at Infection and Total Partners were influenced by low SES and high SIS. Medium and high SES were about equally risky, and low and medium SES were about equally risky. For goils, the relationship between these two risk measures and SIS and SES was more linear – as SES increased, so did risk, and as SIS increased, risk increased. However, there appeared to be no important relationship between either SIS or SES and Percent Infected, indicating that differences in Percent Infected was primarily a function of gender. Indeed the correlation between gender and Percent Infected was -.998 ($p < .001$). Bois were infected in higher numbers than goils (bois 95%, goils 87% overall). In the pair-comparison data described below, the correlation between gender and Percent Infected was -.732 ($p < .001$). Percent Infected thus appeared to be predominantly determined by gender. However, it was maintained as a measure of risk, since it had a .235 correlation ($p \leq .003$) with SES,

compared to a -.238 correlation ($p \leq .002$) between Total Partners and SIS and a -.224 correlation ($p \leq .004$) between Age at Infection and SES. The only substantially stronger correlation was SIS and Age at Infection, at .498 ($p < .001$).

Each SMP was run against every other SMP individually, and also against itself (to look for gender differences within the SMP). There were a total of 18 groups, since there are nine SMPs and two genders, and there were 36 pairings, not including the simulations of a single SMP against itself (see Tables 6-8). The purpose of these simulations was to generate the risk rankings for the sexual motivation landscapes. Since the main question of this project was whether or not changing the proportionate representation in the population of high risk and low risk SMPs changes the risk associated with any SMP, it was crucial to develop a landscape which accurately represented the relative risk of the SMPs.

Risk rankings were generated by calculating the "risk ratio" of each SMP according to the three risk measures within gender (Table 9). The risk ratio is the number of pairings where the SMP is higher risk than its partner, thus generating a probability that this SMP, paired with any random other SMP, will be riskier. For example, assessing Age at Infection risk, HH was riskier than LH, LM, HL, and MH, and less risky than HL, HM, ML, MM, and LL for the bois. For the goils, it was riskier than LL, LH, LM, MM, and MH, and less risky than ML, HM, and HL. This gives a risk ratio of 5:8 for goils, 4:8 for bois (see Table 10 for rankings, Table 11 for Risk Ranking codes). Assessing the same SMP for a different risk measure, Total Partners, HH has a risk ratio of 2:8 for bois, being riskier than LL and MM only. For goils, HH has a risk ratio of 6:8 for Total Partners, being less risky than HM and HL only.

Average risk ratios were also generated by calculating the average risk ratio of, say, all bois scores across the three risk measures, generating a General Bois risk ranking. The same was done to generate a General Goils ranking, Age Neuter, Partners Neuter, Percent Infected Neuter, and General Neuter. With these, a total of 12 rankings were generated. From these, 8 overall rank landscapes were generated. Age Bois and Age Goils were collapsed into Gender Dimorphic Age, and the same occurred with Dimorphic Partners and Dimorphic Percent Infected, to be contrasted with their Neuter counterparts. Each of these rank landscapes was run in the model in each of the 5 risk landscapes (see Tables 12-23.)

Validity Testing

For the purposes of this project, pattern validity provided the most valuable assessment of external validity. Pattern validity is shown when "results generated by the computational model matches real patterns of results" (Carley, 1996 p. 12). Pattern validity was assessed by system-level verification of patterns of behavior. Specifically, high SES, low SIS agents (HL) were expected to have higher overall risk, higher Total Partners, higher Percent Infected, earlier Age at Infection, and have contact with each other. In addition, low SES, high SIS (LH) agents were expected to have the reverse – the lowest overall risk, the lowest Total Partners, the lowest Percent Infected, and the latest Age at Infection. Another validity measure was the relationship among risk measures – to match empirical data, the model was expected to generate reliable relationships among the risk measures. The system was also assessed to search for the scale free distribution of number of partners seen in the empirical data. A scale free distribution is recognizable when it appears as a straight line on a log-log plot (see Liljeros et al, 2002; Dezső and

Barabási, 2002). Some controversy exists over the precise nature of the distribution of average number of partners in a year (e.g., Handcock and Jones, 2003; Jones and Handcock, 2003), but the consensus is that the distribution of number of sex partners exhibits "extreme skewness," (Jones and Handcock, 2003, p. 1123) with the vast majority of individuals reporting a very small number of partners (none or one) and a small fraction of the population reporting considerably higher numbers, up to hundreds or thousands of partners. This kind of distribution – a power law distribution in which each degree is ten times the previous degree (i.e., 1, 10, 100, 1000…) is what one finds as a straight line on a log-log plot. The mechanism which is ascribed to this distribution is preferential attachment, the process of nodes preferentially connecting to nodes with a greater number of existing connections (Dezső and Barabási, 2002).

HL highest risk, LH lowest risk. HL highest risk, LH lowest risk. Overall, was the case with HL was the overall riskiest SMP (Table 24, Figures 8-13). Also, as in the empirical data, SES was more important than SIS in predicting Total Partners, while SIS was more important for predicting other risk factors. However, the detailed picture was more complex.

For goils, HL scored as the second overall riskiest SMP, and the fifth riskiest for Total Partners. Indeed, HL did not score as the highest risk SMP for goils on any measure, with HM scoring highest for Age at Infection and HH scoring highest for Percent Infected. HL's average rank over the three risk measures for the goils was third. LL ranked as the riskiest SMP for Total Partners for goils, followed by HH, LM, and HM. The result of HM as the riskiest for Age at Infection is particularly important, since

Age at Infection is the most direct measure of the diffusion of the disease through the population.

The story was somewhat more straightforward for the bois. For Total Partners and Percent Infected, HL was the highest risk SMP, and it was second riskiest for Age at Infection, following HM. LH was the least risky for Age at Infection, and seventh for Total Partners and Percent Infected. Again, it is HM and not HL which is riskiest for Age at Infection, the most direct measure of disease diffusion.

Relationships among risk measures. In the model, as in the empirical data, risk measures were correlated (Tables 25 and 26). But in the NORM landscape, which in other ways produced results more similar to the empirical data than the other landscapes, Age at Infection was not significantly correlated with Total Partners or Percent Infected for any SMP for either gender.

Distribution of Total Partners. The model failed to reproduce the scale free distribution, instead generating a reliable normal distribution, even with the tracer (intended to function as preferential attachment) on at various thresholds (Figures 14 and 15). Thus the model did not account for the pattern of preferential attachment (as described in Chapter 2) observed in real human sociosexual systems.

Procedure for Collecting Data

After pre-experimental runs determined the representation of SMPs in each landscape, the model was varied systematically to explore the system-level consequences of various landscapes. For each landscape, the model was run 25 times for 7,300 time steps, representing five years**.** The model reports mean scores of 18 groups, defined by gender and SMP – HH Bois, HM Bois, HL Bois, MH Bois, MM Bois, ML Bois, LH

Bois, LM Bois, and LL Bois, along with the Goil equivalents. For each of these groups, the model reports mean time of infection, mean values of SIS and SES at time of infection, Age at Infection, number infected, Percent Infected, mean number of partners at time of infection, mean total number of partners, number of partners subsequent to infection, SMPs of partners, number of initiations, number of consents, and number of declines. Gustatory

Treatment of Data

While inferential statistics are unlikely to provide meaningful information, as described in chapter 2, descriptive statistics, including the means, distributions of various scores, and correlations between variables described the relationship between variables, which could then be compared to relationship observed in real systems. R^2 was used as a descriptive measure of association, to assess the variability accounted for by an effect across more than two groups (Keppel and Wickens, 2004). These comparisons were made in terms of probability rather than absolute value.

Analysis

Null Hypothesis 1: The relative risk associated with any given sexual motivation profile will not vary according to the representation of that profile in the population

In order to assess whether or not agents with higher risk sexual motivation strategies are more at risk for infection, the mean time of infection in agents of each SMP were compared separately with number of partners. If the null hypothesis were to be rejected, number of partners must be negatively correlated with Age at Infection, regardless of landscape, while SMP will have varying correlation with Age at Infection, depending on landscape.

Null Hypothesis 2: An agent based model of sexual motivation can not generate realistic global patterns of disease diffusion from local interactions.

In order to assess whether or not the model generates realistic global patterns of disease diffusion, the measures of external validity were expanded. Matches to scale-free distributions of number of partners, high risk agents having contact with other high risk agents, and whether and when agents are infected will illustrate whether or not the model generated system-level patterns consistent with human data. In particular, the NORMAL landscape, which appears to be the most similar to human populations in terms of the shape of the distribution of SMPs (Carpenter, 2002), would be expected to give rise to patterns like those observed in real systems. Again, these were measured not in terms of absolute value but rather relative value and probability.

Summary

An agent based model of disease diffusion assessed the influence of heterogeneous sexual motivation on disease diffusion in two ways. First, the model varied the sexual motivation landscape – i.e. the proportionate representation of different SMPs – to examine the effect of the structure of the system on individual risk. If risk varied systematically with variations in sexual motivation landscape, then it is plausible that sexual motivation may function as a frequency dependent trait. Second, the model assessed whether or not system-level patterns of disease diffusion can be generated from individual-level decision making. If individual level decision making generated, in particular, the scale-free distribution of number of partners, as observed in real systems, this supports the potential utility of agent based modeling to generate realistic models of

human sociosexual systems. With additional work, agent based modeling may become a valuable tool in the assessment, prediction, and prevention of disease diffusion.

Chapter 4

Analysis of Data

This agent based model was designed to test the hypothesis that risk of infection with a sexually transmitted disease depends on the sexual motivations of an individual's conspecifics. This chapter includes an analysis of that model. Varying the model's sexual motivation landscape measured the effect of the structure of the system on individual risk. Analysis assessed whether or not system-level patterns of disease diffusion could be generated from individual-level decision making

Procedure for Collecting Data

After pre-experimental runs determined the representation of SMPs in each landscape, the model was varied systematically to explore the system-level consequences of various landscapes. For each landscape, the model was run 25 times for 7,300 time steps, representing five years**.** The model reports mean scores of 18 groups, defined by gender and SMP – HH Bois, HM Bois, HL Bois, MH Bois, MM Bois, ML Bois, LH Bois, LM Bois, and LL Bois, along with the Goil equivalents. For each of these groups, the model reports mean time of infection, mean values of SIS and SES at time of infection, Age at Infection, number infected, Percent Infected, mean number of partners at time of infection, mean total number of partners, number of partners subsequent to infection, SMPs of partners, number of initiations, number of consents, and number of declines.

Results

Null Hypothesis 1: The relative risk associated with any given sexual motivation profile will not vary according to the representation of that profile in the population.

In order to assess whether or not agents with higher risk sexual motivation strategies are more at risk for infection, the mean Age at Infection in agents of each SMP were compared with number of partners. If the null hypothesis were to be rejected, Total Partners must be negatively correlated with Age at Infection, regardless of landscape, while SMP would be expected to have varying correlation with Age at Infection, depending on landscape.

Total Partners and Age at Infection. It was determined that genders did not differ in the way they varied across risk rankings and therefore the genders were not assessed separately. Since different risk rankings affected the comparison of results across the landscapes, the different risk rankings were assessed separately within each landscape.

Analysis revealed a strong negative overall correlation between Total Partners and Age at Infection for all landscapes ($r = -.451$, $p < .001$), along with a consistent strong negative correlation between Total Partners and Age at Infection within risk ranking and landscapes, overall (Table 27). That is, agents with a greater number of partners were infected earlier. Analysis also found a varying correlation between Age at Infection and SMP across different landscapes, from essentially no relationship ($R^2 = .000$) to a very large effect ($R^2 = .454$, $p < .005$) (Table 27). Importantly, the strongest, most consistent correlation was the NORM landscape, the landscape most like that found in real human populations. This was the only landscape in which all risk rankings generated an effect size over .06 (per Keppel and Wickens, 2004). There was no correlation between the absolute percentage of a particular SMP and any risk measure. Thus the correlation between landscape and risk was not a measure of absolute

representation in the population, but rather relative representation of a given SMP in the population. The first null hypothesis was thus rejected.

Since it appears that SMP varies in risk relative to other SMPs depending on its relative representation in the population, the questions remains: "in what way does it vary?" To determine this, a variable called "outcome rank" – the average outcome rank of a variable – was compared to "assigned rank" – the assigned rank of an SMP for any given risk ranking. Assessing rank change across landscapes, rather than assessing absolute value of a risk measure across landscapes, was important since some landscapes had higher overall scores on each risk measure. For example, the mean Bois' Age at Infection for the RISKY landscape was 114, while the mean Bois' Age at Infection for LIN-DYS landscape was 137 (Table 28). Age at Infection exhibited a .378 correlation ($p < .001$) with Landscape. Score-based assessments thus would compare landscapes' risk as much as SMPs' risks. Assessing rank change instead effectively standardized score differences across landscapes. Exploratory analysis examined the differences between assigned rank and mean outcome rank. Only those SMPs with more than one risk ranking in a particular assigned rank had sufficient N to calculate the relationship, and only those are reported. Results revealed that the change from assigned rank to outcome rank could be assessed most effectively by considering only groups defined by gender, SMP, and assigned rank, within each risk measure.

Overall, rank change was strongly associated with landscape relative to Age at Infection ($R^2 = .194$, $p < .001$ for bois; .175, $p < .001$ for goils, see Tables 29 and 30, Figure 16) and moderately associated with landscape relative to Total Partners (R^2 .086 for bois; .094 for goils see Tables 31 and 32, Figure 17). However, the correlations for

Total Partners did not reach significance. Rank change was not well associated with Percent Infected (Tables 33 and 34, Figure 18) and not at all associated with absolute percentage of agents of a particular SMP. These changes in rank across landscape were not significantly different across the various risk rankings.

SMPs were analyzed to explore the relationship between gender and Age at Infection, controlling for Total Partners. Partial correlation controlling for Total Partners revealed that only in the HL and LH profiles were genders significantly different in Age at Infection (HL $r = .271$ $p < .05$, LH $r = -.312$ $p < .005$), indicating that while bois had more partners than goils, the two groups were roughly equal in infection rates. Considering that goils had twice the infection susceptibility, this is an important result.

The SMPs which were influenced least by landscape (that is, the landscapes with the fewest large R^2) were HL, LH, and MM. These profiles, then, have the most stable risk, least influenced by landscape. MM had the middle average rank overall (mean outcome rank 8.64) while HL had the highest risk overall (mean outcome rank 6.73) and LH by far the lowest overall rank (mean outcome rank 13.63).

It is important to note that landscape had a more reliable effect on bois than on goils. This is most likely due to the greater variability in SIS in goils, as a result of the oscillation (Table 35). Analysis was also performed to measure the effect of SIS and SES separately on rank change within landscape. Overall SMP was more closely correlated with rank change than either SIS or SES (Table 36-39)

It is also important to note that SIS and SES also had main effects on risk. Across the landscapes, in general, the higher an agent's SES, the higher its risk on all three risk measures (Age at Infection, Total Partners, and Percent Infected), and across all

landscapes, in general, the lower an agent's SIS, the higher its risk on all three measures. Moreover, a SMPs outcome rank was closely related to its initial, assigned rank. As noted previously, risk measures were closely associated with gender, as well, and gender was largely defined by the behavior and values of SIS and SES. While it appears that landscape does influence a SMP's risk, that influence is bounded by and agent's gender and by the SMP's inherent risk. It is important, therefore, not to overdraw conclusions from the results related to changes in SMP rank across landscapes.

Null Hypothesis 2: An agent based model of sexual motivation can not generate realistic global patterns of disease diffusion from local interactions.

In order to assess whether or not the model generated realistic global patterns of disease diffusion, the measures of external validity were expanded. Matches to scale-free distributions of number of partners, high risk agents having contact with other high risk agents, and if and when agents are infected were assessed to illustrate whether or not the model generated system-level patterns consistent with human data. These assessments were performed only in the NORM landscape, which appeared to be the most similar to human populations (Carpenter, 2002). The three measures were assessed for relative value and probability rather than absolute value but rather.

Scale free distribution of number of partners. The distribution of Total Partners remained a roughly normal distribution without the tracer (Figure 19, compare Figures 14 and 15). The model did not reproduce this characteristic of real human sexual networks.

High risk agent partners. There was some support for the hypothesis that high SES, low SIS agents have more contact with other agents of high SES and low SIS. As delineated in Table 40, the tracer increased the correlation between an agent's SIS and

SES and those of its partners. SES in particular was associated with the partner's SES. The model assessed the density of tracer on the landscape where an agent mated. Analysis revealed a strong negative correlation between SIS and tracer density for both bois and goils ($r = -.734$, $p < .001$ for bois, $r = -.450$, $p < .001$ for goils). No relationship was found between SES and tracer density. Thus the tracer may have served primarily to deter lower risk agents, rather than attracting higher risk agents.

The model did not record the absolute SMP of agents' partners, but it did record the absolute value of both SIS and SES of partners and reported the average SIS and SES of agent groups' partners. In order to determine whether or not this was a good predictor of SMP, the model also reported the average SIS and SES of SMP/gender groups themselves. For goils, mean SIS and SES at infection was closely associated with SMP (SIS $R^2 = .520$, SES $R^2 = .852$). For bois, SES at infection was closely associated with SMP ($R^2 = .796$) but SIS was not ($R^2 = .007$). It is speculated that this lack of correlation is influenced by the effect of the bois' refractory SIS spike on the mean SIS score. However, because of this, and because goils SES had a higher R^2, only SES was used to predict SMP. The values compared thus appear to be adequate measures of the intended variables.

Age at Infection and SMP. In the NORM landscape, SMP had a moderate overall effect on Age at Infection ($R^2 = .073$, $p \leq .001$). The different risk rankings revealed effects between .079 and .200 (Table 41).

Overall, the second null hypothesis is rejected, with qualification. While the model did reproduce some of the dynamics observed in real social systems, it did not reproduce others. Specifically, the distribution of Total Partners did not follow the same

distribution as that observed in human systems, but the model did generate realistic relationships between age at infection and sexual motivation. To some degree, the model also found that high risk agents were more likely to mate with other high risk agents, particularly in the presence of the tracer.

Summary

Both of the primary hypotheses were partially supported. In the model, the risk associated with any given sexual motivation profile varied according to the representation of that profile in the population. This was not a function of absolute percentage of the population with a given SMP, but was instead more closely associated with proportion of the population, i.e., percent within a particular landscape. However, rank change of an SMP was constrained by the absolute risk of SIS and SES separately. SES was a better predictor than SIS of Total Partners (i.e., higher SES predicted a greater number of partners), while SIS was a better predictor than SES of Age at Infection (i.e., lower SIS predicted later Age at Infection). The NORMAL landscape generated the strongest relationship between SMP and Age at Infection, adding support to the idea that the model represented risk as it is enacted in real human sociosexual systems. The model failed to reproduce the scale free distribution of number of partners, though the tracer promoted same-SES mating.

Chapter 5

Summary, Discussion, Conclusions, Implementations, and Recommendations

Summary

This study focused on building and analyzing an agent-based model of disease diffusion in order to explore the hypothesis that the relative risk associated with an individual's "sexual motivation profile" (SMP) is influenced by the distribution of strategies represented in the population – that is, that sexual motivation functions as a frequency dependent trait. It also explored the utility of agent based modeling as a tool for understanding human sexual risk behavior. Using an agent based model, this project created an artificial environment where agents with different genders, mate values, and levels of sexual excitation and inhibition made decisions about whether or not to engage in sexual activity. Experiments in this environment revealed how the SMPs of conspecifics can influence the risk associated with any individual profile. Analysis emphasized the influence of individual-level motivations on system-level organization.

Discussion of Findings

Compelling similarities and important differences emerged between the model's results and human data. These similarities and differences are discussed below. Then the idea of frequency dependence of sexual motivation is revisited in light of the model's results. Next, the implications of the model for disease diffusion are delineated. Finally, the benefits and drawbacks of ABM as a method are discussed.

Model-Reality Similarities

Empirical data from real human systems describes human sexual systems as scale-free networks of individuals with heterogeneous levels of sexual motivation.

Variability in sexual motivation follows a normal distribution. Those with higher SES and lower SIS are more prone to sexual risk taking than individuals with lower SES and higher SIS. SES in particular appears to be associated with behaviors that increase risk for contracting and disseminating disease, specifically a higher number of partners. All else being equal, individuals with a greater number of partners are more likely to be infected, and tend to be infected sooner. These are the aspects of real systems which were assessed in the model.

The overall picture followed predictions of the dual control model of sexual response, the composition of which requires further detailed investigation. High SES, low SIS was overall the riskiest SMP, while low SES, high SIS was overall the least risky. This relationship seems to be directly related to the absolute values of SIS and SES. Additionally, SES was associated with Total Partners, while SIS was more associated with other risk measures, such as Age at Infection. In real human systems, the association between SES and number of partners would be expected to be related to protective behaviors such as condom use, which is associated with higher levels of SIS. Though a high SES individual may have more partners, he or she may also have adequate SIS to incorporate lower risk practices. In the model, no such protective behaviors are available. Instead, the stronger relationship between SES and Total Partners compared to SIS and Total Partners was likely due to the fact that SES responded to mate value. Specifically, an agent with higher SES required less mate value to generate a high level of incentive, while an agent with lower SES required a higher level of mate value to generate sufficient incentive to motivate behavior. In contrast SIS functioned as a generic, environment-independent brake. This was an effect of the incentive motivational system.

A number of other matches between the model results and empirical data were established. MM was the middle risk SMP, particularly in the NORM landscape. Empirical data indicates that most individuals cluster around the MM SMP in real human systems. The NORM landscape also produced the strongest correlations between number of partners and Age at Infection (between -.522, $p < .05$ and -.84 $p < .001$, depending on the risk ranking). This finding is consistent with human data which suggests that a greater number of partners increases risk of infection.

Model-Reality Differences

First and foremost, the model failed to reproduce the distribution of number of partners found in real human sociosexual systems. This highlights the difference between frequency of intercourse reported in the empirical data, which follows a normal distribution, compared to number of partners, which follows a scale free distribution. In the empirical data, the scale free distribution is based on number of unique partners over the course of one year (Figure 20), but the model generated something like a normal distribution, which is more characteristic of frequency of intercourse in humans (Figure 21 and 22) (Kopp, 1934; Laumann et al 1994). Humans with hundreds or thousands of partners in a year must by definition have a high frequency of intercourse. For example, as illustrated in Figure 23, if a total number of episodes of intercourse over a year are counted for a population, it is likely that the distribution of frequency of intercourse follows a normal curve, while the number of partners follows a scale free distribution. Thus, for the vast majority of individuals, the number of partners one has has no impact on frequency of intercourse, while a small minority with exponentially more partners must engage in the most frequent sex. Whatever mediates this difference between the

probability of having frequent intercourse and having a high number of partners is not accounted for in the model. The tracer was an attempt to generate preferential attachment, but it did not change the shape of the distribution of Total Partners. That is, the tracer did not significantly change the likelihood that any given individual would have a new partner.

One prime candidate for that mediating variable which might account for the difference in the distribution of frequency of intercourse compared to the distribution of number of partners is attachment, a competing motivation at the core of human social structures. Attachment is the mechanism which bonds infant and adult caregiver. In species with high levels of parental investment, such as humans, it protects the infant's survival by increasing the likelihood that an adult caregiver will attend to its needs. Bowlby (1969) originally proposed it as a social motivation, and subsequent work has illustrated that the attachment mechanism is at work in human adult relationships (e.g., Pietromonaco and Feldman Barrett, 1997). Since the function of the attachment mechanism is to bond individuals together, at least temporarily, to the exclusion of others, it is conceivable that individual differences in this competing motivation might give rise to the scale free distribution in number of partners. Specifically, if individuals with high SES and low SIS also have "high attachment," they might have a high frequency of intercourse, but a low number of partners. Conversely, an individual with high SES and low SIS with "low attachment" might have both a high frequency of intercourse and a high number of partners. A great many questions remain related to the interaction between sexual motivation and attachment, including the role of learning and environmental sensitivity. Research on sexual compulsivity has found a relationship

between childhood trauma and attachment problems with sexually compulsive behavior, though this literature has emphasized sexually abusive behavior (Kreeden, 2004).

Certainly many other factors influence an individual's decision to engage in sexual contact with a new partner – adherence to social norms, lack of access to partners, and lack of time for pursing sex, among many other factors, conspire to prevent individuals from having new partners. Since the scale free distribution represents a massive shift from the normal distribution of frequency of intercourse, it could be that a confluence of many factors is required in order for an individual to be one of the few with the exponentially higher number of partners compared to the rest of the population.

The model generated a complex relationship between number of partners, Age at Infection, and SMP, which did not appear to be similar to that found in the empirical data. One possible explanation for this might be the emergence of protective "pockets." If an HL agent commandeers all its neighbors, thus keeping the other agents busy with mating and also maintaining for itself a high frequency of intercourse without a high number of new partners, and if that agent is uninfected, it might provide a local protective influence. With its high sensitivity to appetitive stimuli and low inhibition, an HL agent need not travel far in order to find sufficiently appetitive stimuli, and since mating takes three time steps (plus two time steps for boi refraction), it could be that the model inadvertently creates pockets of infection-free agents, defined by their proximity to a HL agent. If the model mapped agents' paths over the landscape, it could assess whether or not high excitation agents travel less than other agents. If the model assessed total frequency of mating in addition to Total Partners, it would also be possible to assess whether or not distribution of frequency of mating matches Total Partners. If an agent can

have high frequency without high partners, that would help support the idea of protective pockets.

Goils had a higher ratio of partners to lifespan timesteps than bois. This could be puzzling, since the initial assumption is that bois and goils would necessarily have exactly the same number of partners, on average, in an exclusively heterosexual system. How could it be that the goils had a higher ratio? As it turns out, this funding is a product of the mathematics within the model – since goils were, on average, infected earlier, they tended to have shorter lifespans. Total Partners was calculated by dividing an agent's number of unique partners by their lifespan. With the smaller divisor, the goils' ratio of partners to lifespan was larger. This mathematical difference also contributed to the necessity of comparing Total Partners of bois and goils separately. Interestingly, empirical data in humans reflects this phenomenon; adolescent girls who are sexually active with older male partners are more at risk for STIs and unintended pregnancy (Darroch, et al, 1999).

Sexual Motivation as a Frequency Dependent Trait

A primary goal of this model was to assess the plausibility of the idea that sexual motivation functions as a frequency dependent trait. The results are ambiguous and further evidence is required in order to establish the possibility one way or the other. While risk varied across landscapes in the model – i.e., the disease diffused differently within different sexual landscapes – that variance was constrained by absolute values of SIS and SES. Other evolutionary explanations for variability within a trait include the possibility that the trait was not under selection pressure or that the variability is the normal variability around an optimal state of an adaptation.

The SMPs which were influenced least by landscape were HL, LH, and MM. HL is the high risk profile identified by empirical evidence, and LH is the profile which leads to proneness to sexual dysfunction. MM is the profile most represented in the real population, and would represent the "optimal" profile if the variability in sexual motivation represented normal variability around an optimal state. These profiles, then, have the most stable risk, least influenced by landscape. MM had the middle average rank overall while HL had the highest risk overall and LH by far the lowest overall rank. If MM, as the most common SMP in the human population, is "optimal," the model indicates that "optimal" is not a function of minimum risk.

In order to explore this question further, the model must incorporate benefits, in addition to costs, to sexual behavior; specifically, sex must be reproductive. In terms of natural selection, the evolutionary payoff of reproductive success may mirror the risk, with LH having the smallest reproductive payoff, HL the highest, and MM the middle. Thus the costs and benefits of each SMP would be balanced. If all SMPs can be successful by balancing health cost with reproductive benefit, that could account for variability within the population. The balanced costs and benefits might mean that even the extreme SMPs will not be extinguished in the population.

Implications for Disease Diffusion

Individual Susceptibility. Certain individual agents in the model were more at risk than others. In general, the higher an agent's SES and the lower its SIS, the more likely it was to be infected sooner. In this way, gender influenced risk insofar as the goils had lower SES and higher SIS on average compared to the bois. Risk was also influenced by gender in terms of "biological" susceptibility. Goils had twice the risk of infection when

mating with an infected agent, compared to bois, and consequently were infected sooner and died younger than the bois.

Group Susceptibility. At the same time, it appears that the sociosexual structure of an individual's group also influences risk. Landscapes with a greater proportion of high risk agents exhibited an earlier Age at Infection, on average, and greater Total Partners. The possibility also exists that being in the immediate vicinity of a high SES, low SIS agent opposite sex conspecific could potentially reduce an individual agents' risk, by protecting the agent in a pocket of activity in its local environment, which remains infection free. Thus characteristics of the sociosexual landscape could both prevent and promote risk.

Changing behavior. It appears that SIS and SES contribute significantly to risk behavior, and therefore they might be valuable targets for health interventions. Yet major questions remain about the vulnerability of SIS and SES to environmental forces and intentional intervention. SIS and SES interact with the environment to motivate behavior, which means they are sensitive to environmental forces, and mood, stress, and environmental threats can all influence their functioning. From this, it may be assumed that SIS and SES are responsive to changes both internal and external to the individual. At the same time, SIS and SES are proposed to be more or less stable over an individual's lifetime (Bancroft and Janssen, 2000). To what extent can educational or cognitive behavioral interventions facilitate intentional control over these two mechanisms? What environmental factors maximize the benefits of each, while minimizing the risks? The model does not directly test any intervention strategies; instead it investigates the influence of environmental changes (i.e., sexual landscape) on the risk associated with

SMP. Adding interventions to the model can help to examine this key question of the changeability of SIS and SES.

Benefits and Drawbacks of ABM for Modeling Human Sexual Systems

The model was a drastic simplification of real human systems and the results reflect that. The primary consequences of the simplicity of the model is the lack of generalizability of the results. While the results generated some interesting and unanticipated parallels to the empirical data, it would not be appropriate to assert that the model therefore reflects reality. However, insofar as it reproduced some of the important (in terms of sexual risk) patterns found in real systems, the model is a valuable contribution to research in sexual risk behavior.

Though highly simplified, the model allowed for the manipulation of populations over long periods of time, a process which would have been both time and money intensive to do with humans. By increasing the verisimilitude of the model, these drawbacks of oversimplification can possibly be overcome, while still keeping the model sufficiently manageable to allow for policy and theory testing.

Health behavior theory and interventions increasingly emphasize the importance of accounting for multi-level interactions between an individual, his or her social circle, and the social ecological environment in which health choices are made. ABM is valuable because of its specific ability to model these interactions. It offers a controlled, though simplified, approach to understanding the ways that environment shapes behavior, and that individual behaviors give rise to large scale, system-level patterns.

Conclusions

The following conclusions can be drawn from study results and the previous discussion of the results:

1. In the model, the disease diffused differently through different sexual landscapes, and consequently the risk associated with any given sexual motivation profile varied according to the representation of that profile in the population. This was not a function of absolute percentage of the population with a given SMP, but was instead more closely associated with proportion of the population, i.e., percent within a particular landscape. Change of risk across landscapes was constrained within the influences of SIS, SES, and gender.
2. SES was a better predictor than SIS of Total Partners, while SIS was a better predictor than SES of Age at Infection, which parallels human data.
3. In the model, there was a normal distribution of Total Partners. The addition of a landscape "tracer," intended to function as a preferential attachment mechanism, did not change the shape of the distribution, but did lead to greater correlation between an agent's SES and its partner's SIS or SES.
4. HL, LH, and MM were the SMPs least influenced by landscape. This indicates that they are the most stable SMPs.
5. The NORMAL landscape generated the strongest relationship of all the landscapes between SMP and Age at Infection, the most direct measure of risk, thus supporting the validity of the model.

Implementations

ABM is a tool which has particular benefits and particular drawbacks, as illustrated by the present project. Several avenues of research related to sexual risk taking

and disease diffusion can make use of this tool, including the study of epidemics, the development and implementation of health interventions, and understanding and management individual risk behavior.

ABM, Epidemics, and Public Health Interventions

ABM is a practical tool for understanding the movement of diseases through social networks and the impact of interventions on that movement. It makes a valuable addition to epidemiological modeling, since network models, while valuable, rely on static, typically homogeneous nodes. ABM allows the pattern of disease diffusion to emerge from the behaviors of autonomous, heterogeneous agents, which is a much more realistic description of human systems.

Because one can manipulate individual-level parameters and observe system-level consequences, and vice versa, it is also an excellent method for assessing hypotheses and policies related to disease diffusion. ABM is used to test the consequences of land use policy, traffic laws, and imperialism (Macy and Willer, 2002), and, likewise, may be applied to health policies, such as condom and contraception availability, sexuality education, and medication and vaccine availability. The dissemination of an HIV vaccine, for example, can be anticipated using ABM, modeling different scenarios for global dissemination for the most efficient and effective strategy. These models can take into account infrastructure issues like national and local policies, roads, and population density, as well as social issues like stigma, discrimination, and distrust of vaccines, among others anticipated by Newman and colleagues (2004).

The practical application of the results of agent based models of human sexual health behavior will rely on an accurate understanding of how the results are relevant to

the target population and the implementing organization. The results of this study in particular are based on a model so removed from real systems that to draw dramatic conclusions in terms of practice would be specious. In general, the adaptation of model results to public health interventions must be conservative and generated in combination with empirical data relevant to the target population. Some of the results from the model reflect empirical data and further substantiate the potential for effective interventions.

A practical aspect of agent based models is the theoretical couch of complexity theory applied to human social systems. "Evolutionary engineering" is an approach to system change which intervenes on the system in which the target system runs (Bar-Yam and Kuras, 2003). What characteristics of human sociosexual systems, then, will adapt to generate the desired change, in response to what environmental changes? Answering this question will generate a better understanding of how diseases diffuse through human sociosexual systems, and thus indicate which elements of the system are most vulnerable to intervention.

A key limitation, and the most powerful in terms of the utility of an evolutionary engineering approach, is our fundamental lack of knowledge about the dynamics of social processes (Puddifoot, 1998). Also, if SIS and SES are traits and therefore stable over time, then not everyone will be responsive to changes in the environment. Understanding under what circumstances SIS or SES can be influenced by environmental changes will be crucial to applying complexity and the dual control model to public health practice.

ABM and Individual HRSB

Modeling interaction. The present model was based on the hypothesis that an individual's risk was influenced by the sociosexual landscape. ABM lends itself to such a

question because it allows for the manipulation of both individual-level characteristics (sexual motivation) and system-level characteristics (sexual landscape), offering a controlled way to assess the mutual influences of the two levels of analysis. If "risk" is defined as the probability that a particular behavior will result in negative health consequences, then determining how the structure of the environment influences that probability is valuable. Even with greater understanding of how individual differences in personality or temperament influence risk, only by assessing the *interaction* of individual with environment will researchers generate an accurate model of sexual risk behavior.

Youth populations. ABM has the advantage of not requiring human subjects, which eliminates the necessity of following human subjects protocols. Consequently, ABM can be used to model the behavior of subjects whose behavior is difficult or impossible to study directly. In particular, since SIS and SES are taken to be traits which are more or less stable across the lifespan, it could be valuable to study the expression of these traits in early adolescence. While directly studying children's sexual behavior poses a variety of challenges, modeling that behavior based on epidemiological data and survey data can provide a way to understand the dynamics of adolescents' sexual behavior without skewing the sample toward only those children whose parents consented to a study.

Generating questions. Further, ABM is a useful tool for thinking about individual differences in sexual motivation, since building the model forces precise thinking about the assumptions underlying a construct. In the present model, for example, the oscillation of female sexual motivation was incorporated into the framework of the Dual Control Model, leading to the hypothesis that it is SIS and not SES which oscillates. While for the

sake of the model this hypothesis was assumed to be true, the question of SIS or SES (or both) oscillating remains and can be addressed empirically. Another example from this model is the increase in SES with sexual abstinence. While it is broadly accepted that sexual motivation is an incentive system which becomes more sensitive with abstinence, the cap to that sensitivity is not clear. In the model, a more or less arbitrary cap was assigned in order to prevent highly unrealistic behavior, because no empirical data exists. However, the question about humans remains: what mechanism, if any, leads to the maximization of sexual excitation? The process of developing the model can accentuate areas of scarce or no empirical data and thus generate questions for future investigation.

Reciprocal testing. A further use for agent based modeling in the study of individual risk behavior is the reciprocal hypothesis testing between empirical data and model data. The emergence of patterns in model data that reflect empirical data helps to affirm the validity of the model. The emergence of model data that has not been tested empirically can inspire empirical research which can further validate the model, as well as advance the theoretical model of sexual risk behavior.

Recommendations

Recommendations for future research fall into three categories: (1) elaborations and improvements on agent based models of human sexual behavior; (2) investigation into the evolution of human sexuality; and (3) further empirical and clinical work on sexual risk behavior. Suggestions for modeling focus on increasing the verisimilitude of the model, as well as making it more immediately applicable to health intervention needs. In recommending next steps for examining the evolution of human sexual motivation, the emphasis is on understanding women, as theoretically and biologically more complex

than men in several ways, and focusing on cost-benefit tradeoffs. Future research on high risk sexual behavior is suggested in order to examine more explicitly the interaction between individual- and system-level factors with a complexity-based approach, particularly with regard to emotion and personality research.

Agent Based Models

Sexual Reproduction and Evolution. This model of sexual motivation can be developed into a model of the evolution of sexual motivation, including death by infection and sexual reproduction. By incorporating sexual reproduction via genetic algorithm, researchers can observe what SMPs are selected for across generations of agents. This is the next step in developing tests of the idea that sexual motivation is a frequency dependent trait, since a model which incorporates sexual reproduction allows the co-evolution of boi and goil agents in the context of both sexually transmitted infection (cost) and sexual reproduction (benefit).

Social verisimilitude. Incorporating social norms, courtship and relationship duration, recovery from infection, and the other known influences on sexual risk listed in Chapter 1 will increase the verisimilitude of the model, though at the cost of simplicity.

Mate value. An important aspect of social verisimilitude in sexual reproduction models is the display and assessment of mate value among conspecifics. Several models of assortative mating exist and were discussed in Chapter 2. Other important improvements to the functioning of mate value include imperfections in agents' mate value detection mechanism, so that they do not have perfect information about others' and their own mate values, and incorporating a reputation function, where agents learn about the MV of potential partners through others. This would likely contribute to

assortative mating in the model, distributing partners and other resources more to agents of high mate value.

Competing motivations. In real human systems, sex is one of several competing motivations. Humans must also meet other basic needs like hunger and thirst, along with other social motivations such as intragroup cooperation and competition, intergroup dynamics, parenting, and exploration. In particular, exploring the influence of an attachment mechanism on the social structure will help address the quandary of the different distributions of number partners and frequency of intercourse. Models of human society will necessarily require multiple competing motivations, allowing the decision process of an agent to be shaped by multiple states of deprivation and satiation.

Learning. As a sort of intragenerational genetic algorithm, learning is a crucial element of an ABM of human motivation (Savage, 2000). There is evidence, for example, that the conditionability of women's sexual response varies across the menstrual cycle (Graham, Janssen, and Sanders, 2000; Slob, Bax, Hop, Rowland, and van der Werff, 1996), and men's judgments of attractiveness can be changed with exposure to different faces (Jones, 1996). It is unclear how SIS or SES promotes or inhibits learning, so the incorporation of a learning mechanism can help generate as well as test new hypotheses.

SIS-I. SIS in the model functioned as a sort of hybrid of SIS-I and SIS-II, the two separate inhibition mechanisms outlined by the dual control model. The two are responsive to different kinds of stimuli – SIS-I is characterized as responding to threats of performance failure, while SIS-II is characterized as responding to threats of performance consequences. But, for example, it is not clear which mechanism causes refraction, or if it

is both. Thus two different functions of adding a second SIS mechanism to the model present themselves. First, by making SIS more realistic – a change which makes the most sense when the model includes elements of the environment for both SIS systems to respond to – the model increases its verisimilitude and may generate results more similar to the empirical data. Second, by modeling SIS-I in the model, it contributes to research examining the function of SIS-I in sexual motivation.

Protective behaviors. Perhaps most important in terms of making the model practical to interventionists is the incorporation of risk reduction behaviors, such as condom use or contraceptive use and vaccination behaviors. Such a function would be relatively straightforward, since there is reliable data about vaccine acceptance and the factors which influences condom and contraceptive use, error, and failure. In addition, it would be possible to model the interaction between individuals and landscapes which had risk reducing devices available in the environment.

Risk inducing behaviors. Equally importantly, the model can incorporate behaviors which increase individual and group risk, such as drug and alcohol use, poverty and the use of sex for economic gain, and living in a culture which subjugates women, lacks access to healthcare and other resources, or enforces health-damaging social norms.

Environmental factors. On another level, the incorporation of environmental factors which shape individuals' risk behavior can also add not just to the verisimilitude but also the practicality of a model of sexual behavior. The present model incorporates one environmental factor, in the form of the tracer, which functions as a fairly abstract representation of place reputation. As outlined in Chapter 1, several environmental and social factors are known to influence sexual behavior in humans and other animals.

Substantiating models of environmental forces on sexual behavior will generate better theoretical grounds for developing evolutionary engineering health interventions.

More sophisticated risk measures. Perhaps most important to increasing the predictive reliability of the model is the incorporation of more sophisticated measures of risk. Variables like Percent Infected were influenced by the birth-death cycle, obscuring their results and causing their interpretation to be qualified. Future developments of the model can generate more sophisticated data by more completely anticipating possible results. For example, it is unclear how Percent Infected relates to average Total Partners. If the model mapped agents' paths over the landscape, it would be possible to assess whether or not the high excitation of agents results in pockets of agents protected from infection, due to a lower number of unique partners, as described above.

Evolution of Human Sexuality

Women's sexual motivation. In recommending next steps in examining the evolution of human sexual motivation, the emphasis is on understanding women, as theoretically and biologically more complex than men in several ways. In addition to the possibility of oscillation of motivation across the menstrual cycle, a key issue in women's sexual motivation is what social, parenting, or resource benefit there is to sexual behavior for women. It is likely the case that men too gain non-reproductive benefits from sexual behavior, but since they are virtually always fertile, for men all penile vaginal intercourse is potentially reproductive. For women, who are fertile only about one day in every month, intercourse is only potentially reproductive a small proportion of the time. How does this difference between women and men influence the differences between women's and men's sexual motivation?

Also, women's greater biological susceptibility to infection from sexual contact poses a risk above and beyond unwanted pregnancy. In the model, goils were twice as susceptible as the bois. Partial correlation controlling for total number of partners showed that only HL and LH had significant differences in Age at Infection between bois and goils. Thus low SES and high SIS at the goils' levels had a stronger protective effect for goils than bois, while the reverse put them at greater risk than bois. Possibly due to the goils' greater susceptibility, the strength of their "brakes" relative to their "gas" is all the more important for reducing risk. Understanding the balance of these higher biological risks with lower frequency of fertility and possibly more complex social benefits of sex will be an important part of understanding sexual risk behavior in women.

Costs and benefits of sex. Another crucial target of study is the balance of cost and benefit associated with sexual behavior. In present day human society and in the evolutionary history of human sexuality, the costs and benefits of sex may be many and varied. For example, while reproductive success demands the production of offspring, the production of too many offspring might also have been counterproductive in the environment of evolutionary adaptiveness, if parents lacked the resources to raise those offspring to reproductive age. While the social benefits of sex seen in primate communities help to stabilize social networks, it might also serve to attach an adult to an inadequate mate. While some health risks are relatively benign, such as most strains of HPV in the modern world, others are powerfully virulent, such as HIV in the modern world and bacterial infections in early Europe.

Model results suggest that human sexual motivation is not, on average, the least risky sexual motivation. Instead, most humans have the SMP which, in the model,

exhibits an intermediate level of risk. Future research can examine the possibility that the reproductive benefit of sex balances out the health cost. As described above, defining "cost" and "benefit" is a massively complex undertaking, suited to modeling due to this methodology's flexibility and control. Studying the balance of sexual cost and benefit might help explain the variability observed in human sexual motivation: if extreme SMPs' reproductive costs and benefits counterbalance each other, they may continue into the next generation and thus never be extinguished from the population.

High Risk Sexual Behavior

Interaction between individual and system. Above all, future research on high risk sexual behavior is suggested to examine more explicitly the interaction between individual- and system-level factors with a complexity-based approach. System level characteristics of disease diffusion, such as the amalgamation of risk around particular subpopulations, emerge from individual behaviors. At the same time, it appears that risk for some individuals will be influenced by the representation of SMPs in the population. Understanding the nature of this influence will help generate a more complete theoretical account of individual sexual risk taking and population level disease diffusion.

Part of understanding the influence of sexual landscape on individual risk will include understanding the nature of sexual incentive. SES responds to incentive, but it is not clear what constitutes an incentive. For example, women respond genitally to a wider range of stimuli than men, including images of monkeys engaged in sexual behavior (Chivers and Bailey, in press). Since sex for humans is not solely reproductive, with social and psychological benefits, it is likely that "incentive" is far broader than merely a potential partner's mate value and includes social incentive cues and psychological

incentive cues. Also, the role of learning will emerge here too as an important issue. Understanding what constitutes an incentive could generate greater understanding of human sexual responsiveness and can help inform health interventions.

SES. In tandem with understanding incentive is understanding SES. Since SES is responsive to incentives to the environment and increases with deprivation, several issues are important in understanding how SES can be managed. It is not clear what aspect of sexual behavior leads to a reduction in SES, whether it is orgasm, ejaculation (in men), or a psychological association of some sexual behavior with a decrease in anxiety, as in some compulsive behavior. The ability to measure changes in SES and then assessing what environmental or biological factors cause it to increase or decrease will help to inform interventions. It might be the case that ejaculation in men adequately reduces SES to prevent them from making riskier sexual choices, in which case interventions which educate men to masturbate to manage high sensitivity to sexual incentive stimuli might prove effective. If instead it is particular behaviors which reduce SES, then interventions which use behavioral or cognitive behavioral interventions to train individuals to associate lower risk behaviors with the decrease in anxiety would be more effective. In either case, understanding the functioning of SES is important to generating interventions optimized to the nature of SES.

The meaning of "altering" SES is unclear. If SES is a trait, what does it mean that it has changed? It may be that a SES score on a questionnaire is indicative of a range of possible sensitivities to appetitive stimuli. It may be that SES itself can be changed through intervention, or might be that SES can only be managed. Many questions exist about SES, including the extent to which SES is situational, its susceptibility to mood,

stress, or environmental factors, and its responsiveness to interventions which give individuals cognitive skills to manage sexual decisions rationally. Modeling sexual motivation interventions after interventions designed for other trait-state personality dimensions may help both with theoretical clarification of the model, as well as with clinical and public health interventions.

Conclusion

The research focused on building and analyzing an agent-based model of disease diffusion in order to explore the hypothesis that the relative risk associated with an individual's "sexual motivation profile" (SMP) is influenced by the distribution of strategies represented in the population. Analysis emphasized the influence of individual-level motivations and behaviors on disease diffusion in the system. Results of the model indicate that in the model the relative risk of a SMP does vary depending on the relative representation within a population, and also that risk is influenced distinctly by agents' sexual excitation (SES) and sexual inhibition (SIS) systems.

Both of the primary hypotheses were partially supported. In the model, the risk associated with any given sexual motivation profile varied according to the representation of that profile in the population. This was not a function of absolute percentage of the population with a given SMP, but was instead more closely associated with proportion of the population, i.e., percent within a particular landscape. However, rank change of an SMP was constrained by the absolute risk of SIS and SES separately. SES was a better predictor than SIS of total number of partners, while SIS was a better predictor than SES of Age at Infection. The model generated a nonlinear distribution of Total Partners that was not scale-free. The NORMAL landscape generated the strongest relationship

between SMP and Age at Infection, adding support to the idea that the model represented risk as it is enacted in real human sociosexual systems.

Theoretical, empirical, and practical implications of this model include offering opportunities for testing theories and policies and testing and generating hypotheses. Future developments of the model may include genetic algorithms to allow for the evolution of sexual motivation; risk reducing behaviors such as condom use; and more social and mate value verisimilitude. ABM has a variety of benefits and drawbacks as a tool for studying sexual risk behavior, but it has the potential to serve as a valuable tool for assessing policies, generating hypotheses, and testing theories. Future work should focus on increasing the verisimilitude of agents and their environments, in order to make models more practical for designing and testing intervention and policy strategies.

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Table 1 HIV compared to Hypothetical Disease

	HIV	HD
Incubation period	10 years	Roughly 1 year
Gender difference	Double infection risk for females	Double
Transmissible	Almost immediately after infection	Immediately
Biological vulnerability	Pre-existing infection	None.
Detectable Markers	None until progression to AIDS	None.
Probability of infection per heterosexual contact	0.0003 to 0.0014	.3 (bois) and .6 (goils)
Modes of transmission	Sexual contact, vaginal birth, breast feeding, needle sharing and other blood-borne modes	Sexual

Table 2 SIS and SES Means and SD

	SES mean	SES SD	SIS mean	SIS SD
Men	56.74	7.69	51.25	8.25
women	27.62	4.43	31.68	4.73

Table 3 SIS and SES Scores by Gender

	H SES	H SIS	M SES	M SIS	L SES	L SIS
Boi	6.4	3.3	5.6	2.8	4.8	2.4
Goil	6.0	3.6	5.1	3.1	4.2	2.6

Table 4 Percent of Agents per SMP by Landscape

SES	SIS	RISKY	DYSF	LIN-RISK	LIN-DYS	NORMAL
H	L	35.4	4.0	1.1	21.1	2.5
H	M	17.7	4.4	3.6	18.6	3.5
H	H	11.8	5.0	6.1	16.1	10.0
M	L	8.8	5.9	8.6	13.6	20.0
M	M	7.0	7.0	11.2	11.2	28.0
M	H	5.9	8.8	13.6	8.6	20.0
L	L	5.0	11.8	16.1	6.1	10.0
L	M	4.4	17.7	18.6	3.6	3.5
L	H	4.0	35.4	21.1	1.1	2.5

Table 5 Tracer Excitation Ratio

	50^{th}	90^{th}	95^{th}
BOIS	1.4	1.8	2.0
GOILS	3.0	4.0	4.5

TABLE 6 Pair Comparisons: Age at Infection

	LH		LM		LL		MH		MM		ML		HH		HM		HL	
	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G
HL	*HL*	**LH**	HL	*HL*	HL	*HL*	*HL*	**MH**	HL	*HL*	HL	*HL*	HL	***HH***	HL	***HM***	HL	*HL*
HM	*HM*	HM	*HM*	HM	***LL***	HM	*HM*	HM	*HM*	HM	**ML**	***ML***	*HM*	HM	*HM*	HM		
HH	*HH*	HH	*HH*	HH	***LL***	HH	*HH*	HH	***MM***	HH	**ML**	***ML***	*HH*	HH				
ML	ML	*ML*	ML	*ML*	ML	*ML*	ML	*ML*	ML	*ML*	ML	*ML*						
MM	*MM*	**LH**	*MM*	MM	***LL***	MM	*MM*	**MH**	*MM*	MM								
MH	*MH*	MH	***LM***	MH	***LL***	MH	*MH*	MH										
LL	*LL*	**LH**	*LL*	**LM**	*LL*	LL												
LM	*LM*	**LH**	*LM*	LM														
LH	*LH*	LH																

Boldface indicates column higher risk, plainface the row higher risk. Italics indicates higher risk gender. Underline indicates no clear gender difference.

TABLE 7 Pair Comparisons: Total Partners

	LH		LM		LL		MH		MM		ML		HH		HM		HL	
	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G
HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	*HL*	HL	*HL*
HM	HM	HM	HM	HM	HM	*HM*	HM	HM	HM	*HM*	HM	*HM*	HM	*HM*	HM	*HM*		
HH	HH	HH	*HH*	HH	**LL**	*HH*	*HH*	HH	**MM**	*HH*	*HH*	HH	*HH*	HH				
ML	**LH**	ML	ML	ML	ML	*ML*	ML	ML	**MM**	*ML*	ML	*ML*						
MM	MM	MM	MM	*MM*	MM	*MM*	MM	*MM*	MM	*MM*								
MH	MH	MH	MH	***LM***	**LL**	**LL**	MH	*HM*										
LL	LL	LL	**LM**	LL	LL	*LL*												
LM	LM	LM	LM	*LM*														
LH	LH	*LH*																

Boldface indicates column higher risk, plainface the row higher risk. Italics indicates higher risk gender. Underline indicates no clear gender difference.

TABLE 8 Pair Comparisons: Percent Infected

	LH		LM		LL		MH		MM		ML		HH		HM		HL	
	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G	B	G
HL	*HL*	HL	*HL*	HL	HL	HL	*HL*	HL	*HL*	HL	*HL*	**ML**	*HL*	HL	*HL*	HL	HL	*HL*
HM	HM	HM	*HM*	HM	*HM*	HM	*HM*	HM	***MM***	**MM**	***ML***	**ML**	*HM*	HM	*HM*	HM		
HH	*HH*	HH	*HH*	**LM**	***LL***	**LL**	*HH*	**MH**	***MM***	**MM**	***ML***	**ML**	*HH*	HH				
ML	*ML*	ML	*ML*	**LM**	***LL***	**LL**	*ML*	ML	*ML*	ML	*ML*	ML						
MM	*MM*	MM	*MM*	**LM**	***LL***	MM	*MM*	MM	*MM*	MM								
MH	*MH*	MH	***LM***	MH	***LL***	**LL**	*MH*	HM										
LL	*LL*	LL	***LM***	LL	*LL*	LL												
LM	***LH***	LM	*LM*	LM														
LH	*LH*	LH																

Boldface indicates column higher risk, plainface the row higher risk. Italics indicates higher risk gender. Underline indicates no clear gender difference.

Table 9 Risk Ratios for SMPs for each Risk Ranking

	ADB	ADG	PDB	PDG	PIDB	PIDG	GDB	GDG	AN	PN	PIN	GN
HH	0.5	0.625	0.25	0.75	0.375	0.125	0.375	0.5	0.5625	0.5	0.25	0.4375
HM	0.625	0.75	0.875	0.875	0.625	0.625	0.7083	0.75	0.6875	0.875	0.625	0.7292
HL	1	0.5	1	1	1	0.875	1	0.7917	0.75	1	0.9375	0.8958
MH	0.125	0.75	0.25	0.125	0.125	0.375	0.1667	0.4167	0.4375	0.1875	0.25	0.2917
MM	0.5	0.25	0.625	0.625	0.5	0.5	0.5423	0.4583	0.375	0.625	0.5	0.5
ML	0.875	0.875	0.375	0.625	0.625	0.5	0.625	0.6667	0.875	0.5	0.5625	0.6458
LH	0	0.5	0.125	0	0.125	0	0.0833	0.1667	0.25	0.0625	0.0625	0.125
LM	0.125	0.125	0.125	0.125	0.25	0.625	0.1667	0.2917	0.125	0.125	0.4375	0.2292
LL	0.75	0	0.625	0.125	0.625	0.625	0.6667	0.25	0.375	0.375	0.625	0.4583

Table 10 Rank for SMPs for each Risk Ranking

	ADB	ADG	PDB	PDG	PIDB	PIDG	GDB	GDG	AN	PN	PIN	GN
HH	6	4	6	3	6	6	6	4	4	4	7	6
HM	4	2	2	2	2	2	2	2	3	2	2	2
HL	1	5	1	1	1	1	1	1	2	1	1	1
MH	8	3	7	6	8	7	7	6	5	7	8	7
MM	5	7	3	4	5	5	5	5	6	3	5	4
ML	2	1	5	5	3	6	4	3	1	5	4	3
LH	9	6	9	9	9	9	9	9	8	9	9	9
LM	7	8	8	7	7	3	8	7	9	8	6	8
LL	3	9	4	8	4	4	3	8	7	6	3	5

Table 11 RISK RANK CODES

ADB	Age, Dimorphic - Bois	ADG	Age, Dimorphic - Goils	AN	Age, Neuter
PDB	Partners, Dimorphic - Bois	PDG	Partners, Dimorphic - GoilsPercent	PN	Partners, Neuter Percent Infected,
PIDB	Percent Infected, Dimorphic - Bois	PIDG	Infected, Dimorphic – Goils	PIN	Neuter
GDB	General, Dimorphic – Bois	GDG	General, Dimorphic - Goils	GN	General, Neuter

TABLE 12 Percent SMP by Risk Rank – Age at Infection: Bois

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
ML	17.7	4.4	18.6	3.6	3.5
LL	11.8	5.0	16.1	6.1	10.0
HM	8.8	5.9	13.6	8.6	20.0
HH	7.0	7.0	11.2	11.2	28.0
MM	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 13 Percent SMP by Risk Rank – Age at Infection: Goils

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
ML	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
MH	11.8	5.0	16.1	6.1	10.0
HH	8.8	5.9	13.6	8.6	20.0
HL	7.0	7.0	11.2	11.2	28.0
LH	5.9	8.8	8.6	13.6	20.0
MM	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LL	4.0	35.4	1.1	21.1	2.5

TABLE 14 Percent SMP by Risk Rank – Age at Infection: Neuter

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
ML	35.4	4.0	21.1	1.1	2.5
HL	17.7	4.4	18.6	3.6	3.5
HM	11.8	5.0	16.1	6.1	10.0
HH	8.8	5.9	13.6	8.6	20.0
MH	7.0	7.0	11.2	11.2	28.0
MM	5.9	8.8	8.6	13.6	20.0
LL	5.0	11.8	6.1	16.1	10.0
LH	4.4	17.7	3.6	18.6	3.5
LM	4.0	35.4	1.1	21.1	2.5

TABLE 15 Percent SMP by Risk Rank – Total Partners: Bois

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
MM	11.8	5.0	16.1	6.1	10.0
LL	8.8	5.9	13.6	8.6	20.0
ML	7.0	7.0	11.2	11.2	28.0
MH	5.9	8.8	8.6	13.6	20.0
HH	5.0	11.8	6.1	16.1	10.0
LH	4.4	17.7	3.6	18.6	3.5
LM	4.0	35.4	1.1	21.1	2.5

TABLE 16 Percent SMP by Risk Rank – Total Partners: Goils

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
HH	11.8	5.0	16.1	6.1	10.0
MM	8.8	5.9	13.6	8.6	20.0
ML	7.0	7.0	11.2	11.2	28.0
LL	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 17 Percent SMP by Risk Rank – Total Partners: Neuter

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
MM	11.8	5.0	16.1	6.1	10.0
ML	8.8	5.9	13.6	8.6	20.0
HH	7.0	7.0	11.2	11.2	28.0
LL	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 18 Percent SMP by Risk Rank – Percent Infected: Bois

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
ML	11.8	5.0	16.1	6.1	10.0
LL	8.8	5.9	13.6	8.6	20.0
MM	7.0	7.0	11.2	11.2	28.0
HH	5.9	8.8	8.6	13.6	20.0
LM	5.0	11.8	6.1	16.1	10.0
MH	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 19 Percent SMP by Risk Rank – Percent Infected: Goils

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
LL	11.8	5.0	16.1	6.1	10.0
LM	8.8	5.9	13.6	8.6	20.0
ML	7.0	7.0	11.2	11.2	28.0
MM	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
HH	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 20 Percent SMP by Risk Rank – Percent Infected: Neuter

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
MM	11.8	5.0	16.1	6.1	10.0
ML	8.8	5.9	13.6	8.6	20.0
HH	7.0	7.0	11.2	11.2	28.0
LL	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 21 Percent SMP by Risk Rank – General: Bois

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
LL	11.8	5.0	16.1	6.1	10.0
ML	8.8	5.9	13.6	8.6	20.0
MM	7.0	7.0	11.2	11.2	28.0
HH	5.9	8.8	8.6	13.6	20.0
LM	5.0	11.8	6.1	16.1	10.0
MH	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 22 Percent SMP by Risk Rank – General: Goils

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
ML	11.8	5.0	16.1	6.1	10.0
HH	8.8	5.9	13.6	8.6	20.0
MM	7.0	7.0	11.2	11.2	28.0
MH	5.9	8.8	8.6	13.6	20.0
LM	5.0	11.8	6.1	16.1	10.0
LL	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

TABLE 23 Percent SMP by Risk Rank – General: Neuter

SMP	RISKY	DYSF	LINRISK	LINDYS	NORMAL
HL	35.4	4.0	21.1	1.1	2.5
HM	17.7	4.4	18.6	3.6	3.5
ML	11.8	5.0	16.1	6.1	10.0
MM	8.8	5.9	13.6	8.6	20.0
LL	7.0	7.0	11.2	11.2	28.0
HH	5.9	8.8	8.6	13.6	20.0
MH	5.0	11.8	6.1	16.1	10.0
LM	4.4	17.7	3.6	18.6	3.5
LH	4.0	35.4	1.1	21.1	2.5

Table 24 Mean Risk Ranks and Outcome Ranks for all Risk Measures

GENDER	SMP	Mean Age Rank	Age Outcome Rank	Mean Partner Rank	Partner Outcome Rank	Mean Percent Rank	Mean Overall Rank	Overall Outcome Rank	Neuter Overall Outcome Rank
BOIS	LL	4.33	5	5.66	6	4	5.33	5	4
	LM	7.66	8	1.5	9	3.167	7.66	9	7
	LH	9	9	4.33	7	4.33	6.66	7	8
	ML	3.5	3	3.16	8	6.83	6.66	8	8
	MM	3.5	4	7.16	5	2.16	5.66	6	6
	MH	7.33	7	7.33	4	7.83	4.33	4	5.5
	HL	2.66	2	9	1	8.66	1.33	1	1.5
	HM	2.16	1	7.83	2	3	3.33	2	3
	HH	4.83	6	7.66	3	6.33	4	3	2
GOILS	LL	5.83	6	9	1	5.33	3.66	3	-
	LM	7	8	5	3	5.5	4.66	5	-
	LH	8.33	9	1.66	8	4.33	8	9	-
	ML	4	3	1.16	9	2.83	7	8	-
	MM	4	4	2.66	7	5.16	5.33	6	-
	MH	5.83	7	2.83	6	5	6.33	7	-
	HL	3.33	2	3.66	5	5.66	3	2	-
	HM	2	1	4.66	4	3.83	4.33	4	-
	HH	4.66	5	5.66	2	6	2.66	1	-

Table 25 Overall Correlation of Age at Infection and Total Partners, Correlation Age at Infection and SMP

	BASE	RISKY	DYSF	LINRISK	LINDYS	NORM
Correlation between Age at Infection and Total Partners	-.797**	-.608**	-.726**	-.780**	-.619**	-.556**
Correlation between Age at Infection and SMP	-.089	-.115	-.118	-.233*	-.044	-.279*

Pearson's r, * $p \leq .005$, ** $p < .001$

Table 26 Correlation among Risk Measures (Significant Correlations Only)

GENDER	SMP	N	Correlation between Total Partners and Percent Infected
BOIS	LL	6	-0.879*
	LM	6	-0.851*
	LH	6	-0.872*
	ML	6	-0.879*
	MM	6	-0.884*
	MH	6	-0.839*
	HL	6	-839*
	HM	6	-0.906**
	HH	6	-0.876*
GOILS	LL	6	-0.913**
	MM	6	-0.948**
	MH	6	-0.867*

Pearson's r, *$p < .05$, ** $p < .005$

Table 27 Correlations: SMP and Age at Infection and Total Partners and Age at Infection, by Risk Ranking within Landscape

Landscape	Risk Rank	$\bar{x}$	SD	R^2 SMP and Age at Infection	Pearson's r, Total Partners and Age at Infection
RISKY	AD	99.78	15.33	.02	-.557*
	AN	93.74	13.73	.017	-.566*
	PD	84.87	13.28	.017	-.329
	PN	105.44	17.04	.014	-.638**
	PID	95.46	15.47	.006	-.531*
	PIN	96.28	14.54	.028	-.551*
	GD	116.02	16.62	.010	-.564*
	GN	107.30	17.24	.039	-.730**
DYSF	AD	119.85	5.47	.124**	-.771**
	AN	105.21	6.89	.200**	-.936**
	PD	84.87	13.28	.017	-.329**
	PN	117.82	6.92	.269**	-.808**
	PID	166.48	8.42	.454**	-.789**
	PIN	158.56	8.19	.154**	-.689**
	GD	133.59	8.14	.120**	-.796**
	GN	137.77	6.43	.321**	-.748**
LINRISK	AD	120.93	119.17	.119**	-.797**
	AN	112.69	17.86	.090**	-.795**
	PD	107.75	15.62	.067**	-.781**
	PN	102.10	14.24	.024	-.589
	PID	111.18	18.19	.059	-.710**
	PIN	98.59	15.08	.025	-.745**
	GD	117.09	19.29	.145**	-.893**
	GN	113.67	16.11	.006	-.781**
LINDYS	AD	136.76	13.23	.027	-.414*
	AN	137.33	40.16	.015	-.619**
	PD	143.16	13.97	.257**	-.881**
	PN	132.58	15.69	.009	-.519*
	PID	160.07	19.97	.102**	-.537*
	PIN	179.05	19.28	.000	-.157
	GD	129.55	14.47	.175**	-.562**
	GN	140.22	38.37	.026	-.541*
NORM	AD	138.65	5.91	.126**	-.699**
	AN	122.91	8.15	.190**	-.779**
	PD	127.96	6.20	.092**	-.840**
	PN	122.45	6.72	.200**	-.828**
	PID	130.78	8.33	.177**	-.522*
	PIN	125.32	6.07	.118**	-.701**
	GD	140.03	8.39	.079**	-.761**
	GN	135.37	5.33	.157**	-.683**

* $p \leq .05$, ** $p < .005$,

Table 28 Age at Infection, Total Partners, and Percent Infected by Landscape within Gender

		Age at Infection	Age Rank	Total Partners	Partner Rank	Percent Infected	Percent Rank
BOIS	RISKY	114.39	1	.0217	4	96.34	4
	DYSF	132.36	3	.0219	5	94.47	1
	LINRISK	123.94	2	.0188	1	97.44	5
	LINDYS	137.32	5	.0205	3	94.97	2
	NORM	133.48	4	.0202	2	96.09	3
GOILS	RISKY	85.59	1	.0244	2	87.86	4
	DYSF	123.21	3	.0254	1	85.48	2
	LINRISK	95.69	2	.0215	5	88.66	5
	LINDYS	151.47	5	.0242	3	85.14	1
	NORM	128.67	4	.0240	4	87.01	3

Table 29 Bois' Rank Change: Age at Infection

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	10.4	3.84	.296*
	HL	30	9.47	3.88	.001
2	LL	15	11.13	3.34	.414**
	ML	10	9.33	4.32	.096
	HM	10	4.0	3.88	.220**
3	ML	10	12.6	2.61	.444**
	HM	20	9.3	4.13	.189**
4	LM	10	14.3	3.8	.081
	ML	10	9.6	3.37	.082
	HM	10	7.8	4.08	.433**
5	LM	10	14.4	4.06	.204
	ML	10	8.9	3.93	.150
	HH	10	10.7	4.16	.063
6	LM	10	14.9	3.54	.143
	MH	25	14.04	3.7	.224**
7	LL	10	10.8	3.82	.195**
	HH	25	11.48	4.75	.159**
8	LM	10	14.6	3.75	.129
	MH	15	14.33	3.13	.219**
9	LH	30	16.36	2.97	.121**
	MM	10	11.2	3.99	.218*

* $p \leq .05$, ** $p \leq .005$

Table 30 Goils' Rank Change: Age at Infection

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	7.9	2.42	.077
	HL	30	4.07	5.34	.048
2	LL	10	7.5	4.69	.059
	ML	15	5.67	4.48	.038
	HL	10	6.2	6.27	.312*
3	ML	15	6.07	4.35	.013
	HM	25	4.92	4.39	.036
4	LL	10	7.3	3.53	.578**
	LM	10	6.4	3.85	.259*
	HM	10	6.2	4.87	.006
5	LL	10	10.8	4.57	.013
	LM	10	8.5	4.14	.219*
	HH	10	5.1	3.28	.062
6	LM	10	9.6	4.3	.159
	MH	25	8.96	4.66	.399**
7	LL	10	9.7	5.03	.032
	HH	25	6.88	3.62	.006
8	LM	10	9.5	4.75	.478**
	LH	15	11.4	4.81	.383**
	MH	10	8.1	3.6	.586**
9	LH	25	10.88	5.23	.035
	MM	10	5.8	4.26	.069

* $p \leq .05$, ** $p \leq .005$

Table 31 Bois' Rank Change: Total Partners

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	10.2	2.53	.056
	HL	30	6.9	2.45	.080*
2	LL	15	11.67	1.35	.033
	ML	10	9.9	2.92	.188
	HM	10	8.3	1.77	.018**
3	ML	10	10.5	2.01	.197*
	HM	20	8.95	1.96	.005*
4	LM	10	15.7	1.43	.000
	ML	10	8.9	1.37	.236**
	HM	10	8.3	2.31	.234*
5	LM	10	15.6	1.43	.018
	ML	10	8.9	1.37	.036
	HH	10	11.3	4.24	.069
6	LM	10	16.2	0.92	.164
	MH	25	14.36	3.12	.008
7	LL	10	10.2	2.62	.009
	HH	25	12.96	1.67	.158**
8	LM	10	13.2	5.35	.023
	MH	15	15.53	0.99	.015
9	LH	30	16.7	3.39	.058
	MM	10	10.5	1.51	.198*

* $p \leq .05$, ** $p \leq .005$

Table 32 Goils' Rank Change: Total Partners

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	7.4	3.86	.095
	HL	30	3.03	5.12	.098*
2	LL	10	6.3	.82	.033
	ML	15	3.93	3.9	.083
	HL	10	1.6	.55	.389**
3	ML	15	4.6	5.08	.053
	HM	25	2.12	.83	.043
4	LL	10	6.5	2.17	.231*
	LM	10	10.6	3.1	.188
	HM	10	5.2	6.29	.068
5	LL	10	7.3	3.59	.007
	LM	10	12	3.5	.007
	HH	10	6.1	4.25	.123
6	LM	10	9.8	2.1	.062
	MH	25	11.28	2.72	.000
7	LL	10	8.3	4.00	.012
	HH	25	4.4	1.35	.011
8	LM	10	11.8	3.74	.004
	LH	15	16	1.69	.188*
	MH	10	10.9	2.38	.166
9	LH	25	15.36	2.93	.148*
	MM	10	5.3	1.83	.060

* $p \leq .05$, ** $p \leq .005$

Table 33 Bois' Rank Change: Percent Infected

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	5.4	2.8	.026
	HL	30	3.27	2.61	.122**
2	LL	15	4.27	1.79	.074
	ML	10	3.6	2.22	.190
	HM	10	2.6	2.55	.026
3	ML	10	3.6	2.07	.188
	HM	20	3.35	2.08	.001
4	LM	10	7.2	1.99	.014
	ML	10	3.4	1.84	.034*
	HM	10	4	1.89	.189
5	LM	10	6.7	2.21	.059
	ML	10	4.1	2.02	.060
	HH	10	4.3	1.89	.056
6	LM	10	7.2	1.48	.064
	MH	25	6	2.16	.003
7	LL	10	4.3	2.11	.001
	HH	25	4.28	1.99	.000
8	LM	10	7.4	2.01	.002
	MH	15	6.33	2.44	.065
9	LH	30	8.26	1.96	.060
	MM	10	4	2.11	.000

* $p \leq .05$, ** $p \leq .005$

Table 34 Goils' Rank Change: Percent Infected

Assigned Rank	SMP	N	$\bar{x}$	SD	R^2 Outcome Rank * Landscape
1	MM	10	15.00	1.83	.007
	HL	30	13.7	3.73	.026
2	LL	10	15.2	2.20	.056
	ML	15	14.87	2.61	.068
	HL	10	13	4.88	.004
3	ML	15	13.53	2.42	.002
	HM	25	14	2.86	.140**
4	LL	10	15.9	1.97	.322**
	LM	10	14.4	3.1	.230*
	HM	10	15.1	2.92	.032
5	LL	10	15.5	2.12	.000
	LM	10	13	1.87	.026
	HH	10	11.6	2.72	.037
6	LM	10	13.8	2.15	.481**
	MH	25	13.4	2.55	.001
7	LL	10	15	1.7	.123
	HH	25	12.56	2.33	.148*
8	LM	10	15.2	2.39	.035
	LH	15	13.87	2.27	.007
	MH	10	14.8	1.81	.002
9	LH	25	13.08	4.22	.019
	MM	10	13.9	2.23	.055

* $p \leq .05$, ** $p \leq .005$

Table 35 SIS and SES Minimum, Maximum, Mean, and Standard Deviation, by Gender

		Min	Max	$\bar{x}$	SD
Bois	Excitation	3.26	6.4	4.74	0.702
	Inhibition	1.63	3.3	2.4	.38
Goils	Excitation	0.0	6.0	4.33	.77
	Inhibition	0.0	2.49	1.6	.35

Table 36 Bois SES R^2 Rank by Risk Measures

Assigned Rank	SES	R^2 RANK x LANDSCAPE Age	R^2 RANK x LANDSCAPE Partners	R^2 RANK x LANDSCAPE Percent Infected
1	M	.296**	.056	.026
	H	.001	.080*	.122*
2	L	.414**	.033	.074*
	M	.096*	.188**	.190**
	H	.220**	.018	.026
3	M	.444**	.197**	.188**
	H	.189**	.005	.001
4	L	.081*	.000	.014
	M	.082*	.236**	.034
	H	.433**	.234**	.189**
5	L	.204**	.018	.059
	M	.150**	.036	.060*
	H	.063*	.069*	.056
6	L	.143*	.164**	.064*
	M	.224**	.008	.003
7	L	.195**	.009	.001
	H	.159**	.158**	.000
8	L	.129*	.023	.002
	M	.219**	.015	.065*
9	L	.121*	.058	.060*
	M	.218**	.198**	.000

* $p \leq .05$, ** $p \leq .01$

Table 37 Bois SIS R^2 Rank by Risk Measures

Assigned Rank	SIS	R^2 RANK x LANDSCAPE Age	R^2 RANK x LANDSCAPE Partners	R^2 RANK x LANDSCAPE Percent Infected
1	L	.001	.080*	.122*
	M	.296**	.056	.026
2	L	.143*	.073*	.007
	M	.531**	.400**	.003
3	L	.104*	.007	.030
	M	.389**	.111*	.005
4	L	.062*	.008	.259**
	M	.139*	.001	.004
5	L	.226**	.015	.089*
	M	.130*	.008	.034
	H	.063*	.069*	.056
6	M	.171**	.017	.002
	H	.226**	.025	.011
7	L	.549**	.052	.005
	H	.101*	.059	.000
8	M	.135*	.030	.001
	H	.126*	.007	.149*
9	M	.218**	.198**	.000
	H	.121*	.058	.060*

* $p \leq .05$, ** $p \leq .01$

Table 38 Goils SES R^2 Rank by Risk Measures

Assigned Rank	SES	R^2 RANK x LANDSCAPE Age	R^2 RANK x LANDSCAPE	R^2 RANK x LANDSCAPE Partners
1	M	.077*	.095*	.007
	H	.048	.098*	.026
2	L	.059	.033	.056
	M	.038	.083*	.068*
	H	.188*	.103*	.014
3	M	.013	.053	.002
	H	.036	.043	.140*
4	L	.357*	.120*	.258**
	M	.022	.024	.032
	H	.006	.068*	.032
5	L	.074*	.005	.008
	M	.000	.058	.009
	H	.062*	.123*	.037
6	L	.159**	.062*	.481**
	M	.399**	.000	.001
7	L	.032	.012	.123*
	H	.006	.011	.148*
8	L	.403**	.011	.001
	M	.609**	.057	.053
9	L	.035	.148*	.019
	M	.202**	.011	.008

* $p \leq .05$, ** $p \leq .01$

Table 39 Goils SIS R^2 Rank by Risk Measures

Assigned Rank	SIS	R^2 RANK x LANDSCAPE Age	R^2 RANK x LANDSCAPE Partners	R^2 RANK x LANDSCAPE Percent Infected
1	L	.048	.098*	.026
	M	.077*	.095*	.007
2	L	.109*	.038	.000
3	L	.013	.053	.002
	M	.036	.043	.140*
4	L	.077*	.096*	.038
	M	.150**	.000	.076*
5	L	.003	.000	.007
	M	.065*	.000	.016
	H	.062*	.123*	.037
6	M	.159**	.062*	.481**
	H	.431**	.000	.003
7	L	.032	.012	.123*
	H	.006	.011	.148
8	M	.479**	.000	.002
	H	.380**	.063*	.005
9	M	.069*	.060*	.055
	H	.084*	.072*	.011

* $p \leq .05$, ** $p \leq .01$

Table 40 Correlation between SIS and SES with Partner SIS and SES

Tracer	Gender		SES	SIS
-	Goils	Partner SES	.206*	
		Partner SIS	.265*	.256*
5	Goils	Partner SES	.366***	.384***
		Partner SIS	.258*	.346***
	Bois	Partner SES	.-.314**	-.287*
10	Bois	Parter SES	-.252*	
50	Bois	Partner SIS	-.247*	

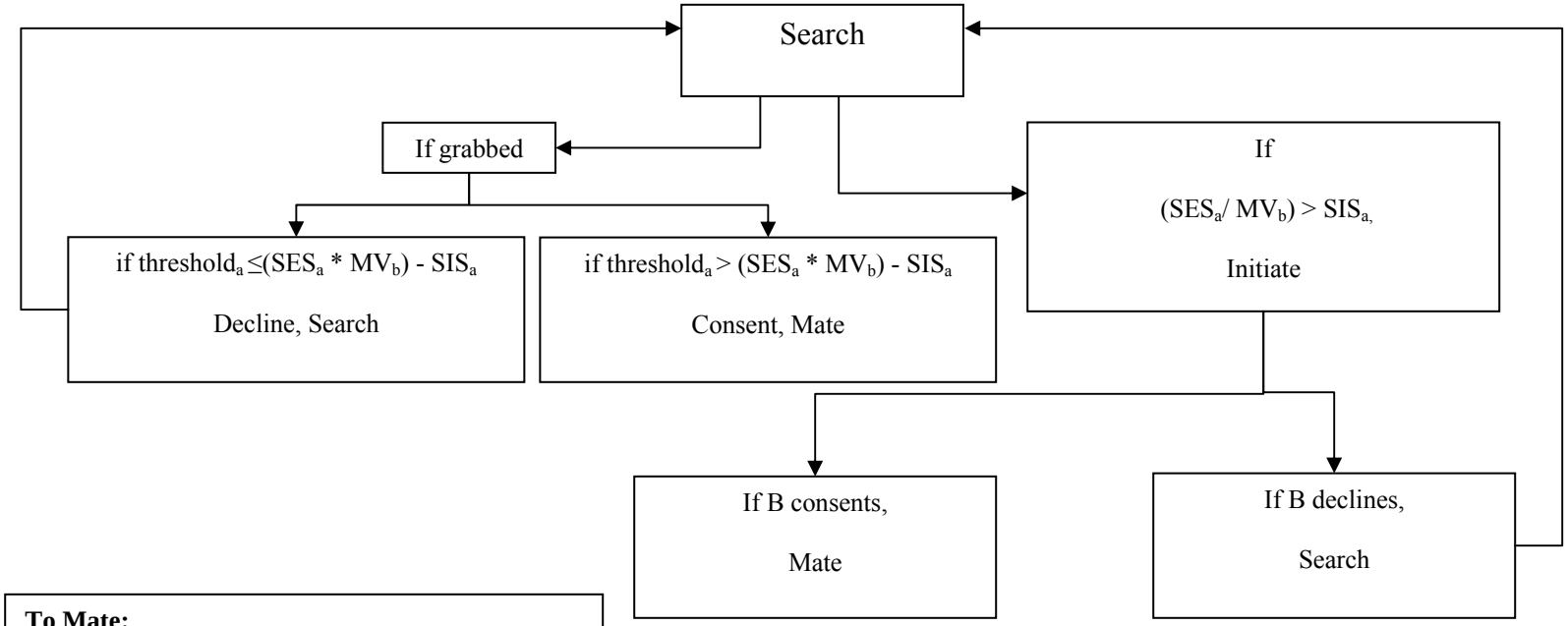
Pearson's r, * $p \leq .05$, ** $p \leq .01$ *** $p \leq .005$

Table 41 Measure of Association: Age at Infection to SMP in NORMAL Landscape

	AD	AN	PD	PN	PID	PIN	GD	GN
R^2	.162*	.190**	.092	.200**	.177*	.118*	.079	.157*

* $p \leq .05$, ** $p \leq .005$

Figure 1 Agent Mating Decision Flow Chart



To Mate:

Unavailable 3 timesteps

+1 partner to tally

Set SES to baseline

SIS spike 2 timesteps, move two timesteps (bois)

search

Flow chart of agent decision process. An agent searches until it is grabbed or its motivation crosses threshold. If it is grabbed, it consents or declines based on its own motivation level, and if it initiates, it receives a consent or decline from the potential partner. Mating requires five total timesteps, three of unavailability to other agents, and two for the bois' refraction.

Figure 2 Max Time: Age at Infection by SMP and Gender

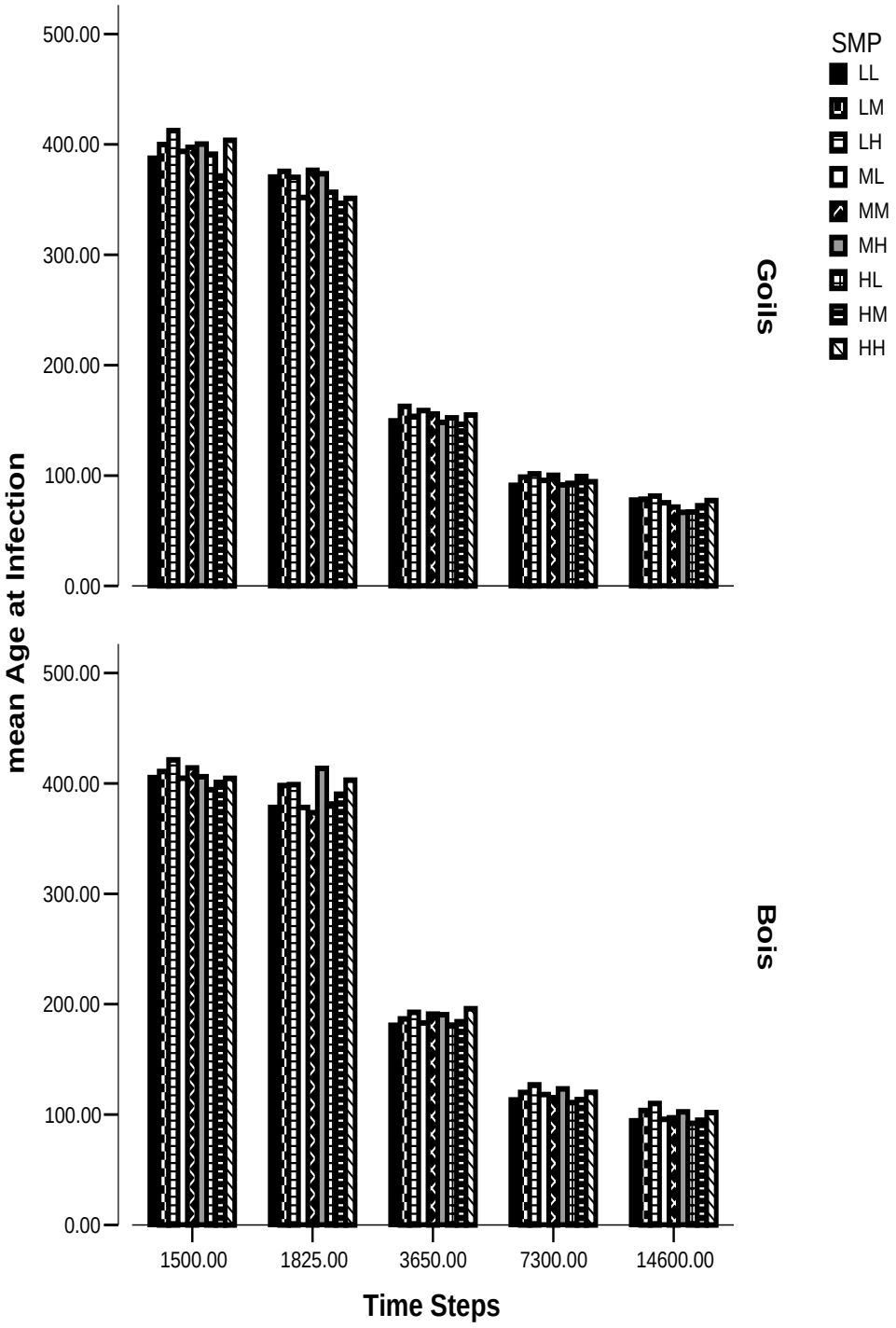


Figure 3: Max Time: Age at Infection over Time Steps

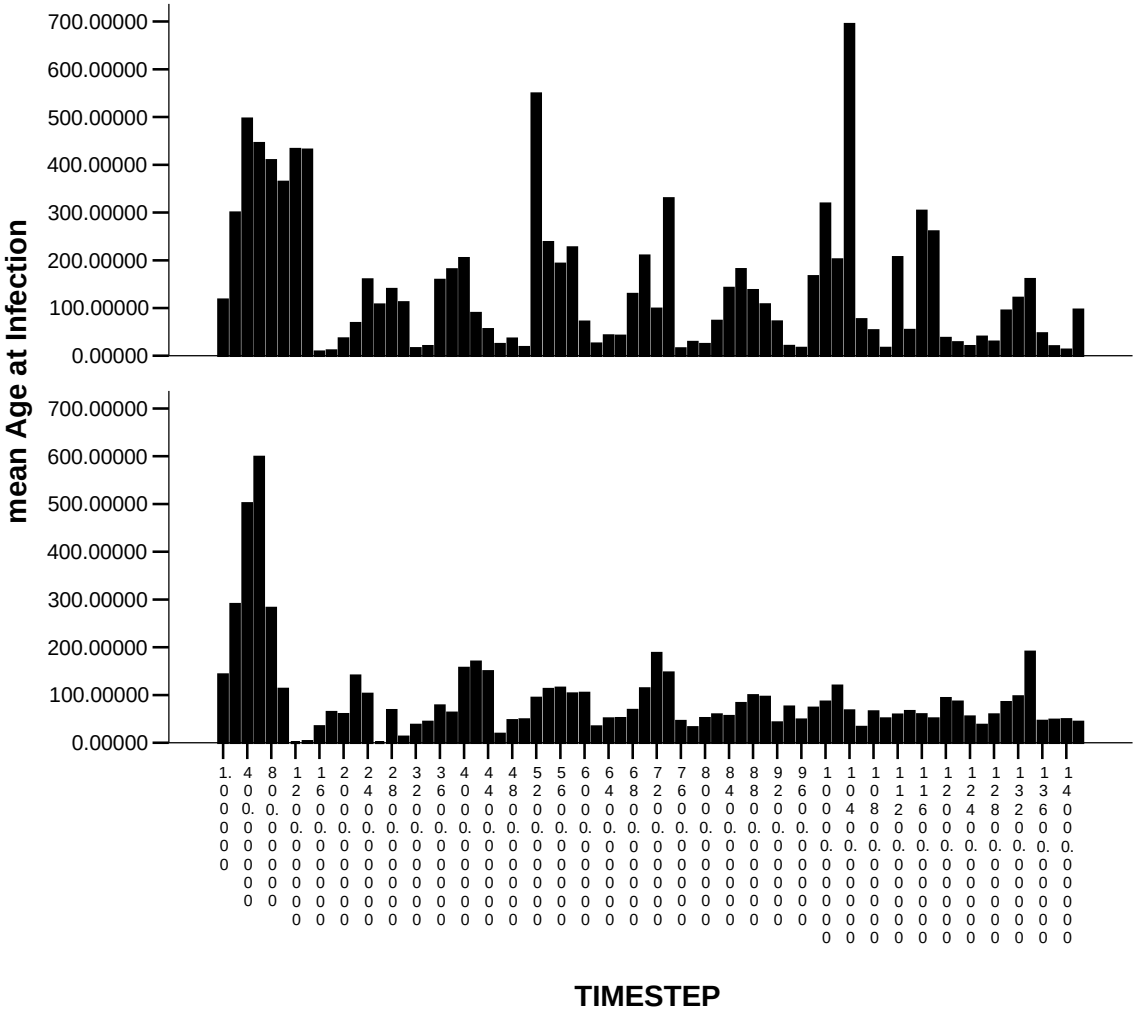


Figure 4 Max Time: Number of Partners by SMP and Gender

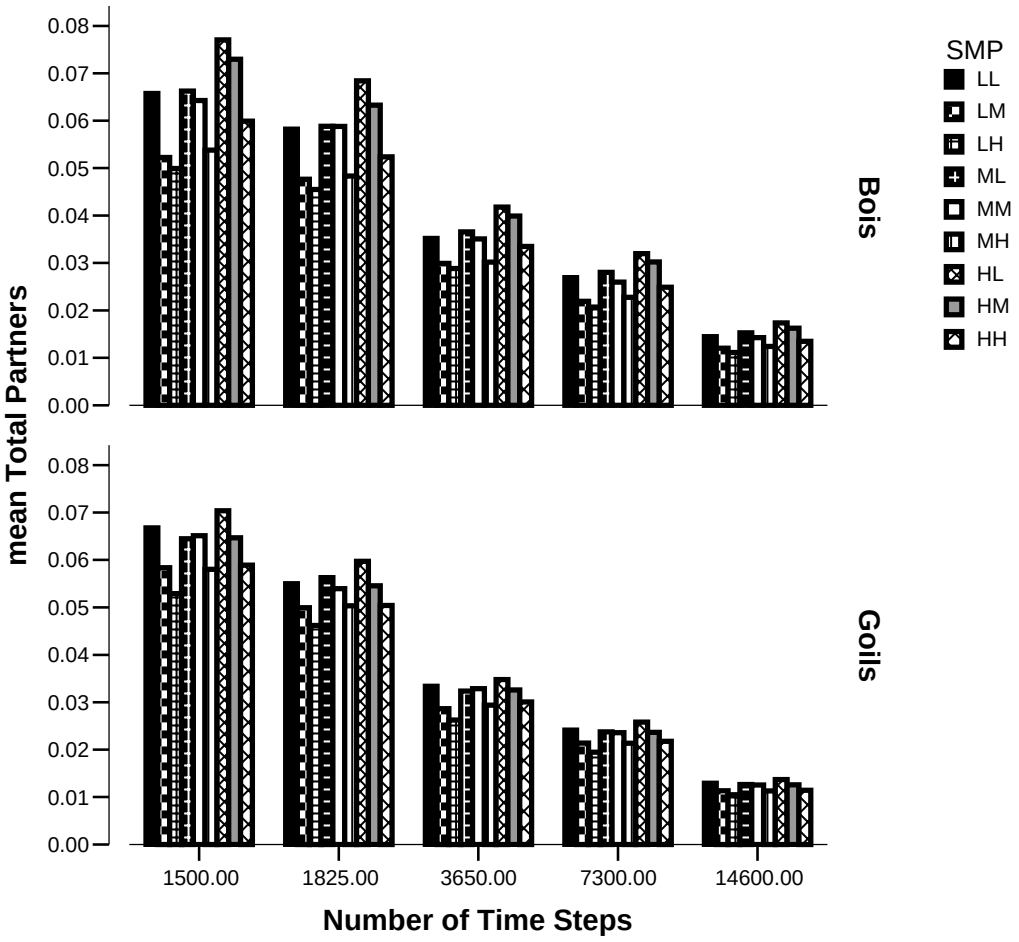


Figure 5 Number of Simulations: Age at Infection by SMP

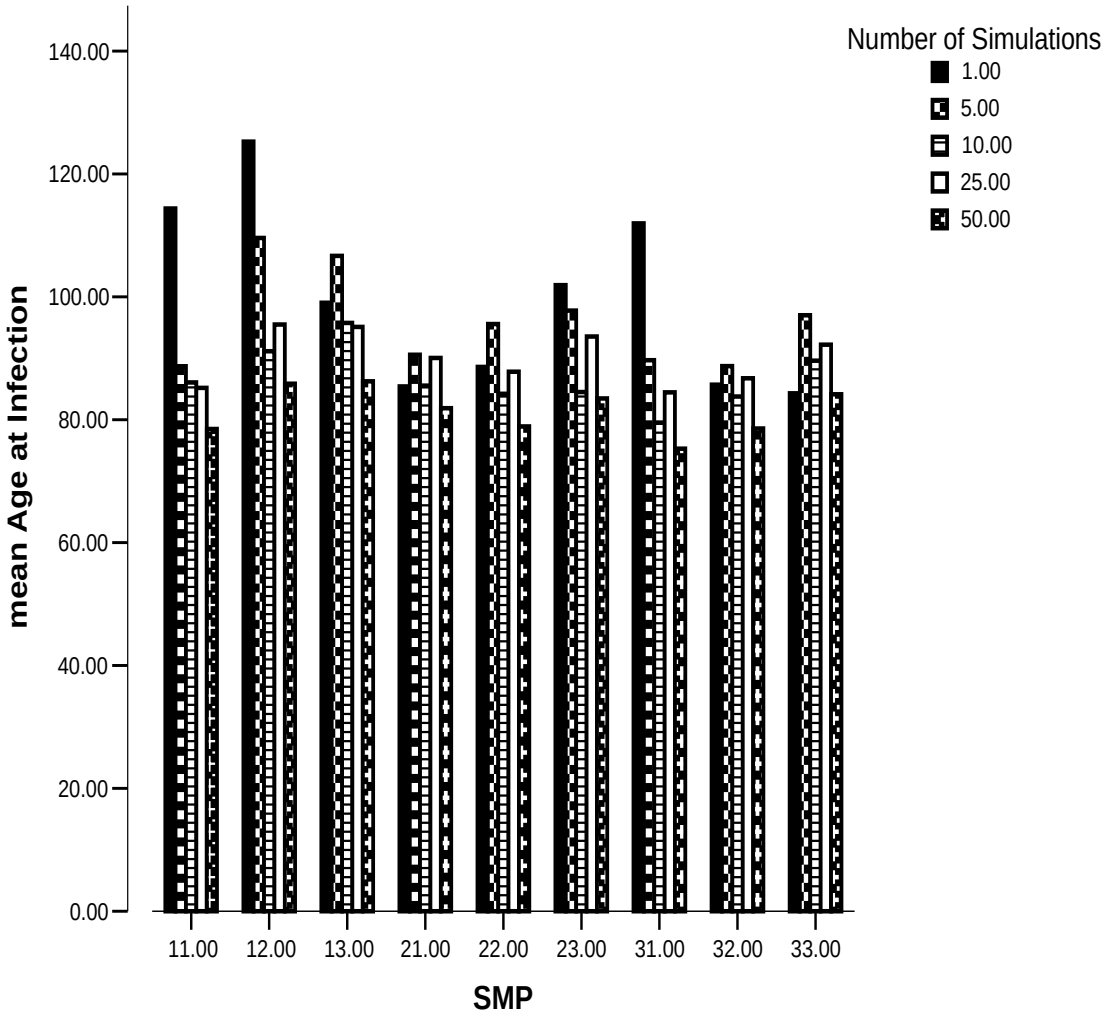


Figure 6 Number of Simulations: Total Partners by SMP

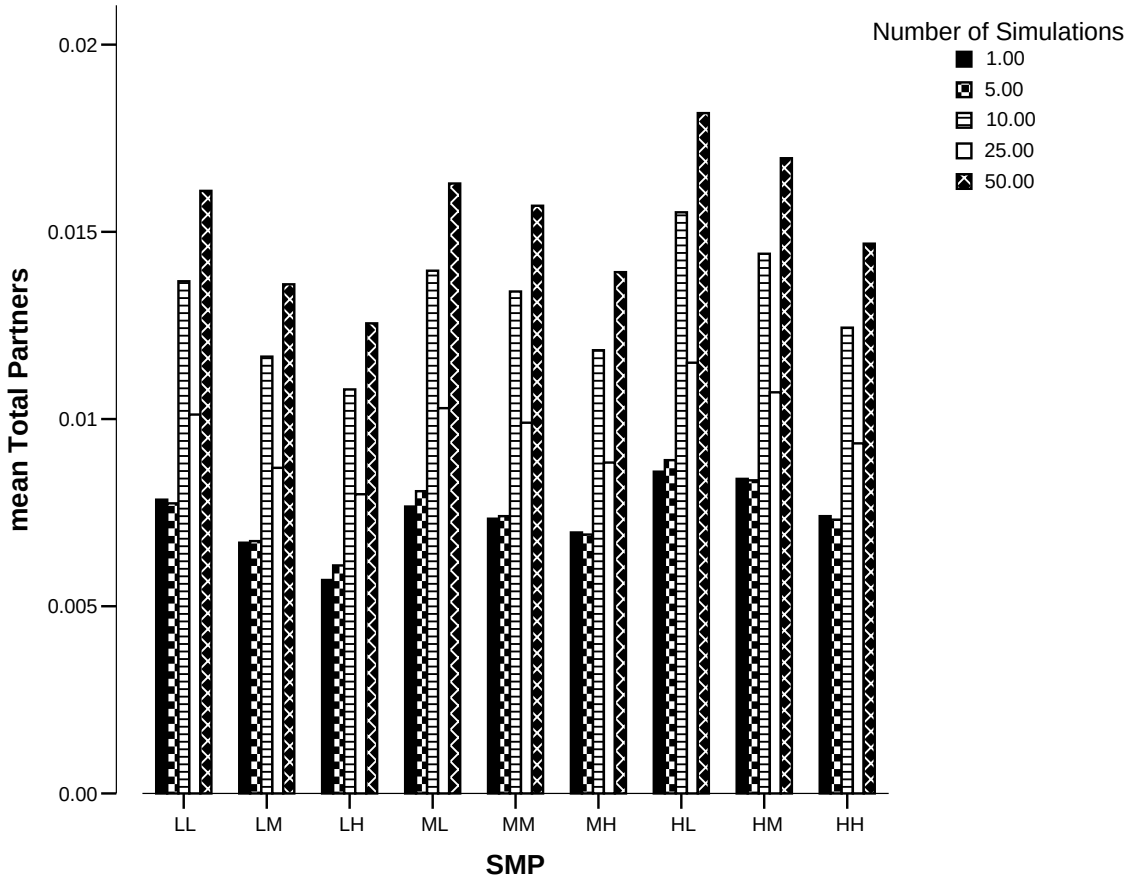


Figure 7 Number of Simulations: Age at Infection by Gender

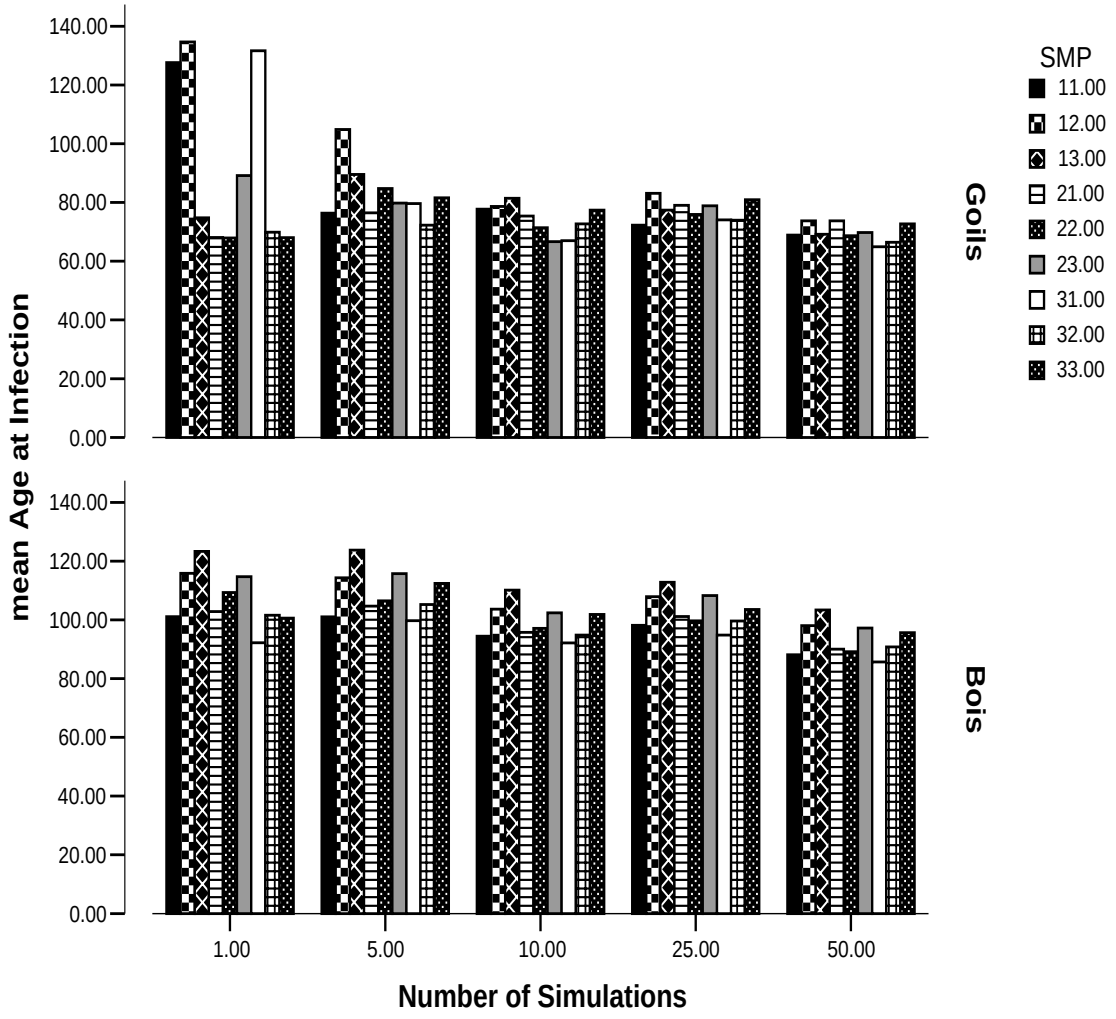


Figure 8 Bois Mean Outcome Rank for Age at Infection, NORM Landscape

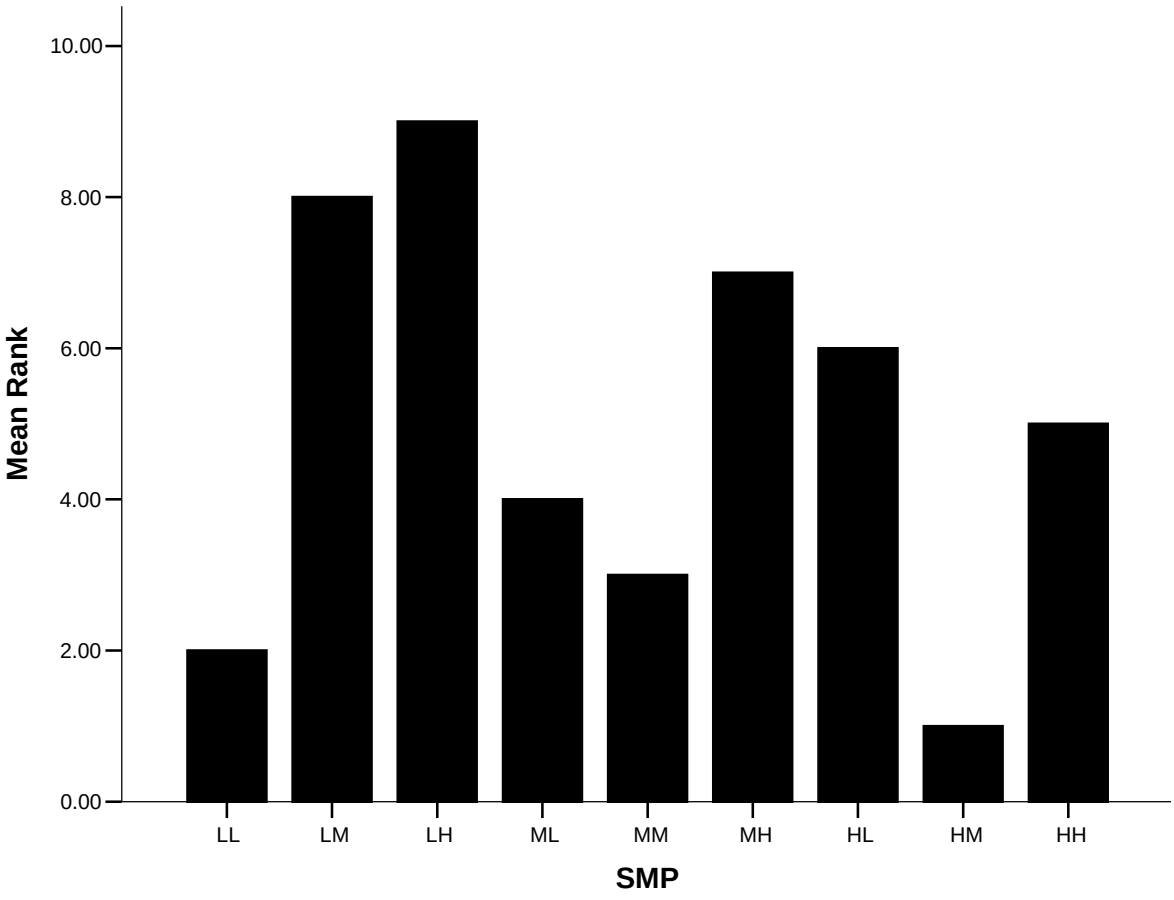


Figure 9 Goils Mean Outcome Rank for Age at Infection, NORM Landscape

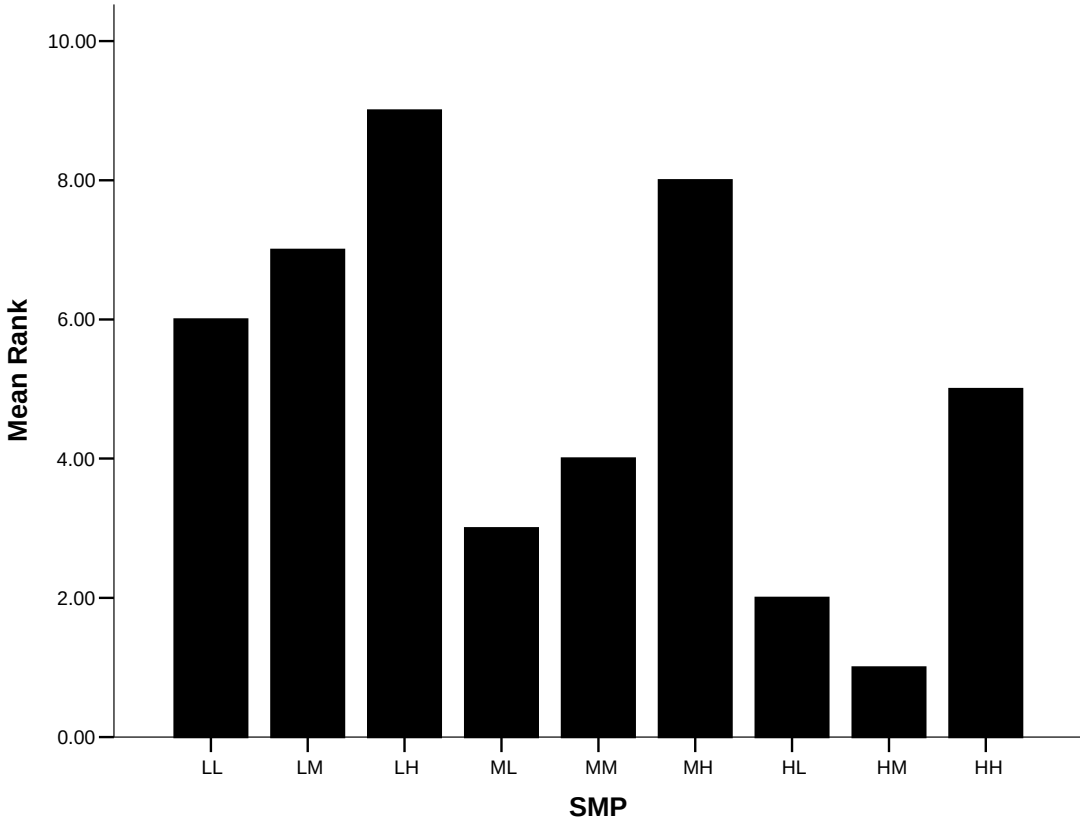


Figure 10 Bois Mean Outcome Rank for Total Partners, NORM Landscape

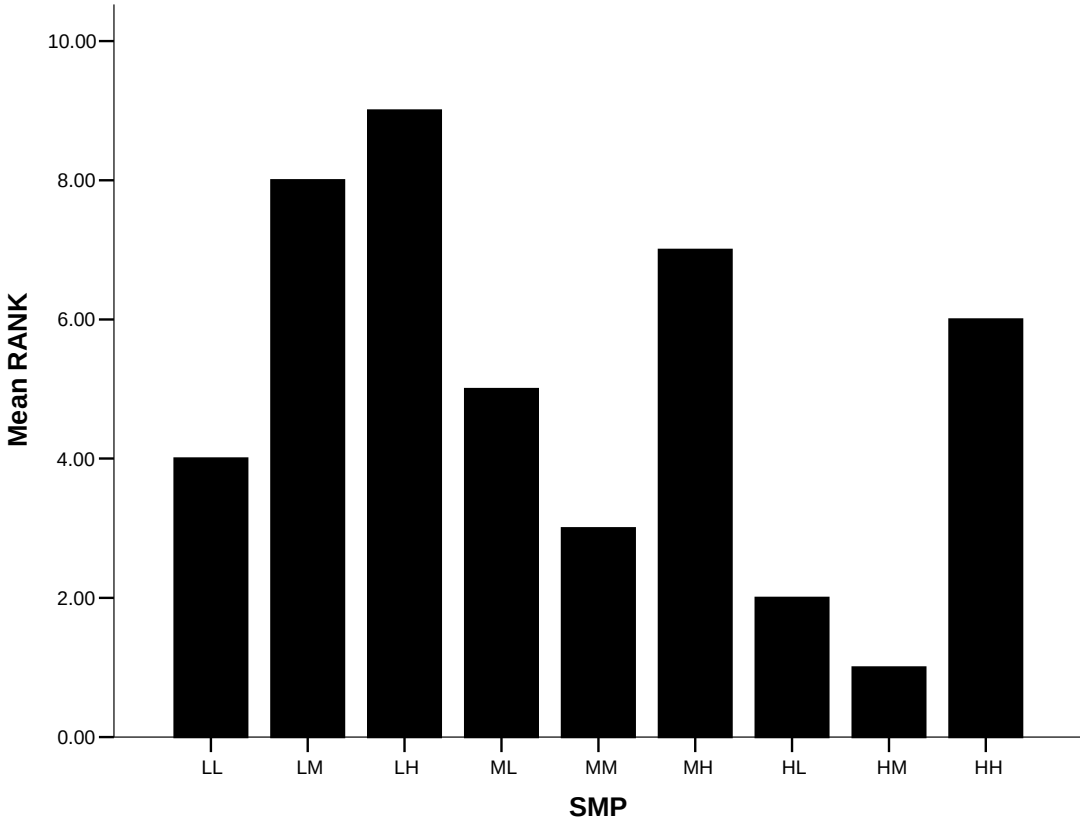


Figure 11 Goils Mean Outcome Rank for Total Partners, NORM Landscape

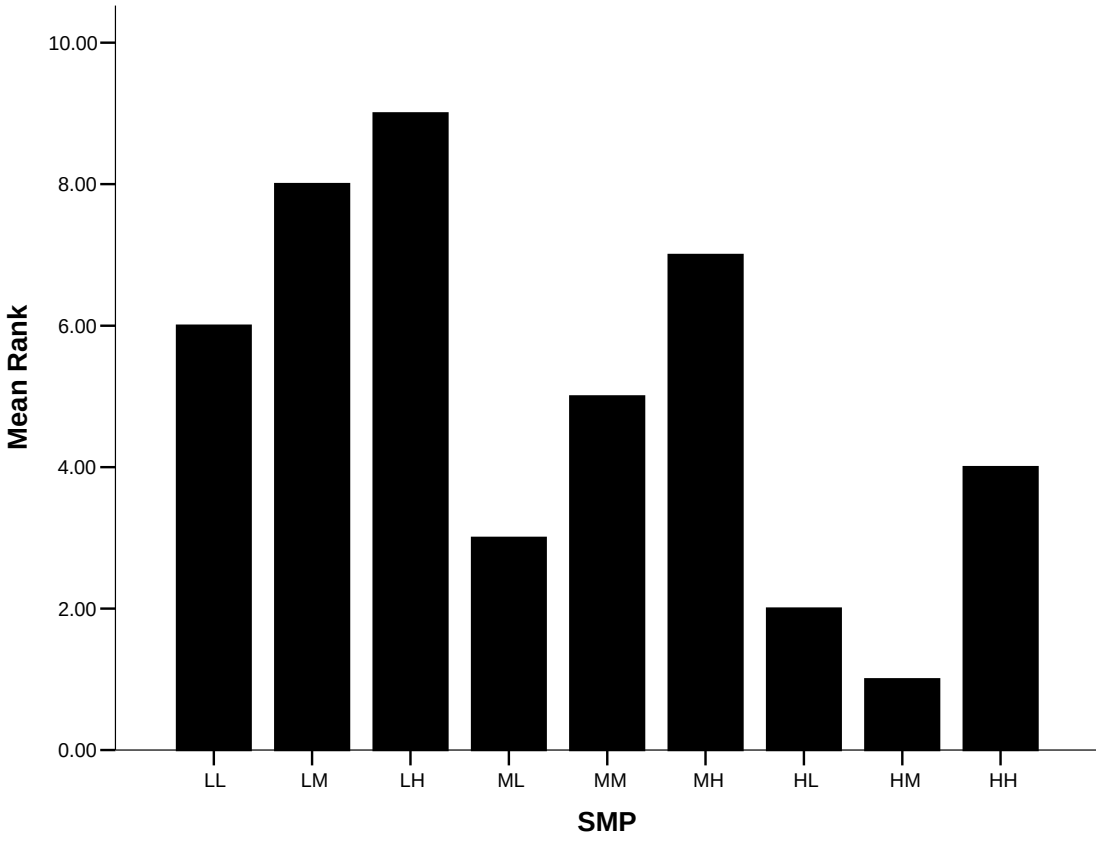


Figure 12 Bois Mean Outcome Rank for Percent Infected, NORM Landscape

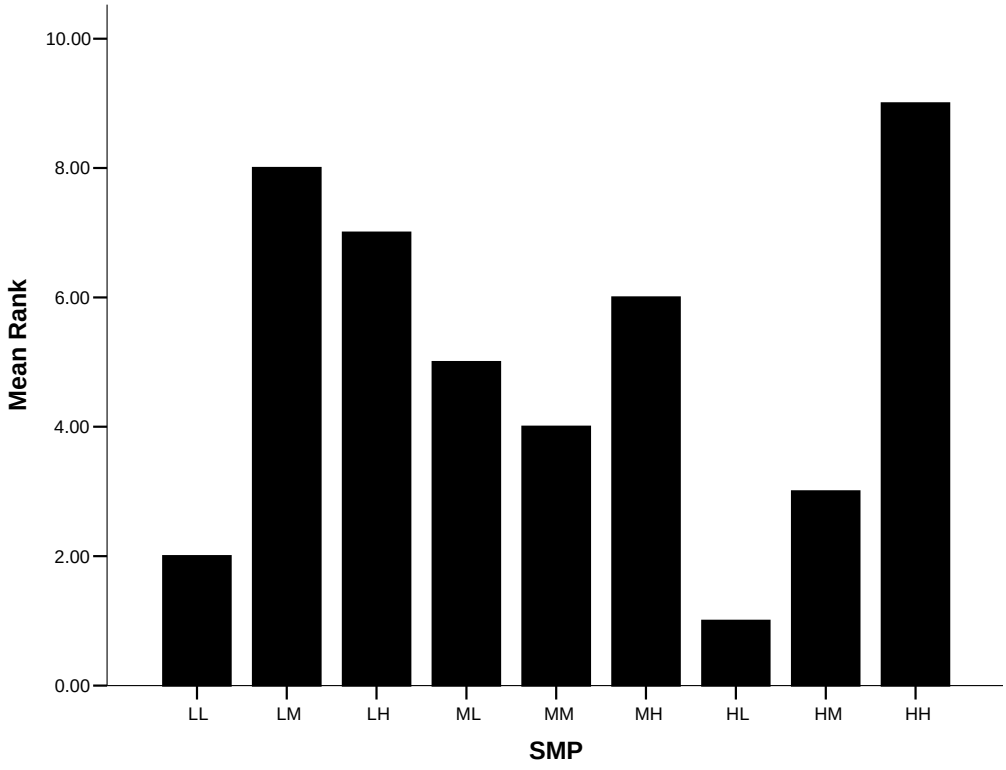


Figure 13 Goils Mean Outcome Rank for Percent Infected, NORM Landscape

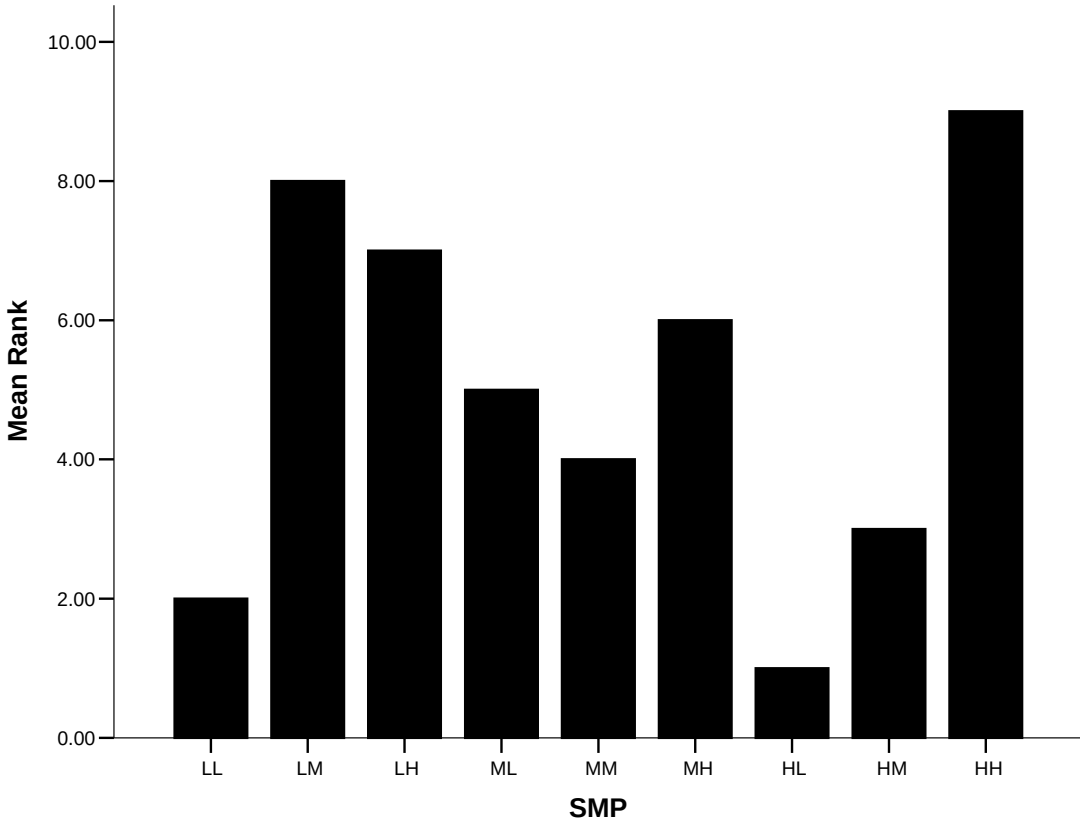


Figure 14 Distribution of Total Partners

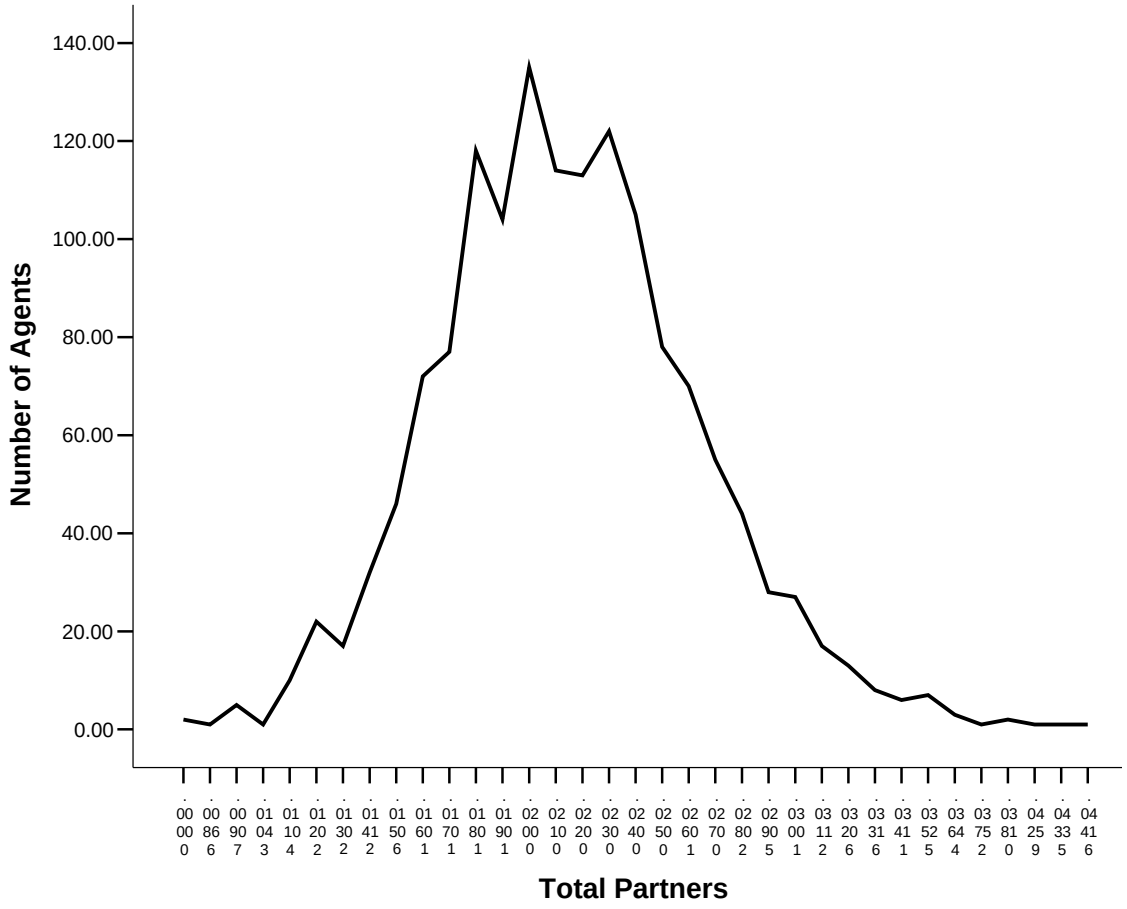


Figure 15 Distribution of Total Partners – Tracer On

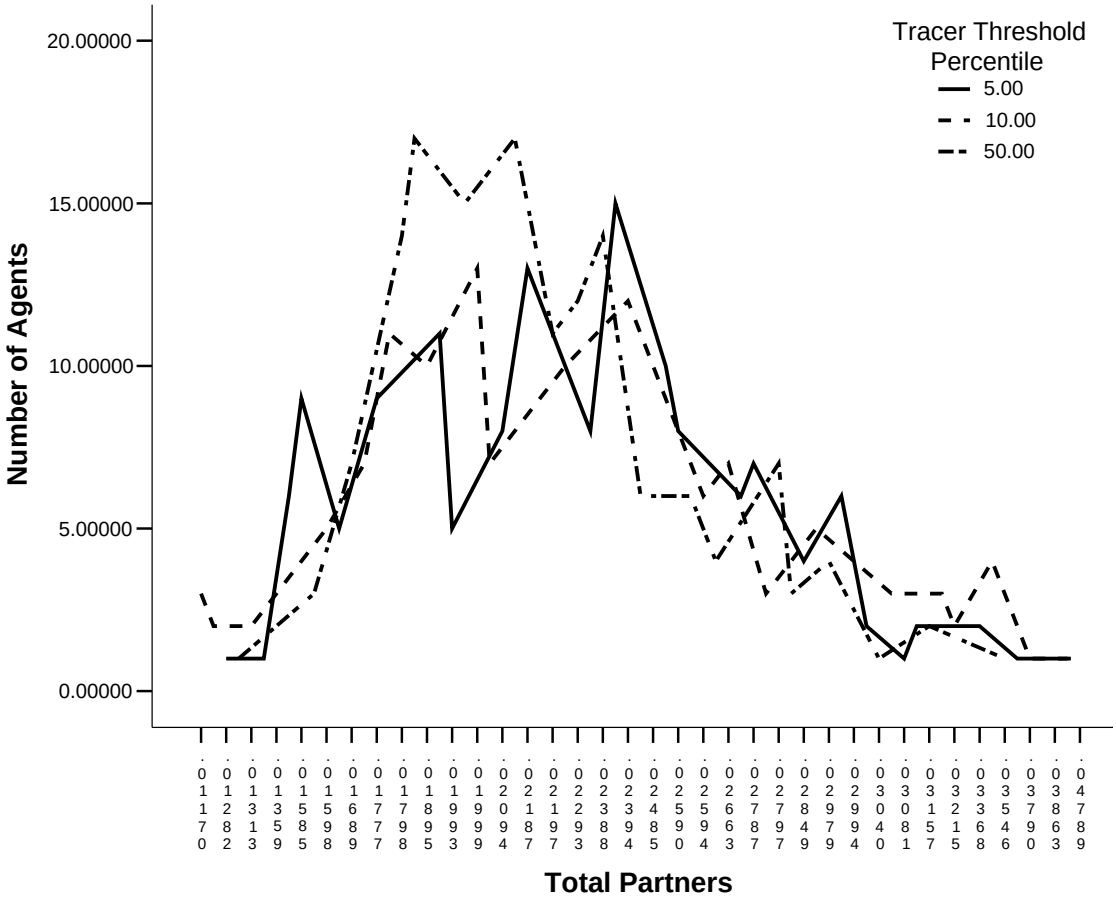


Figure 16 Association between Rank Change and Landscape by SMP and Gender: Age at Infection

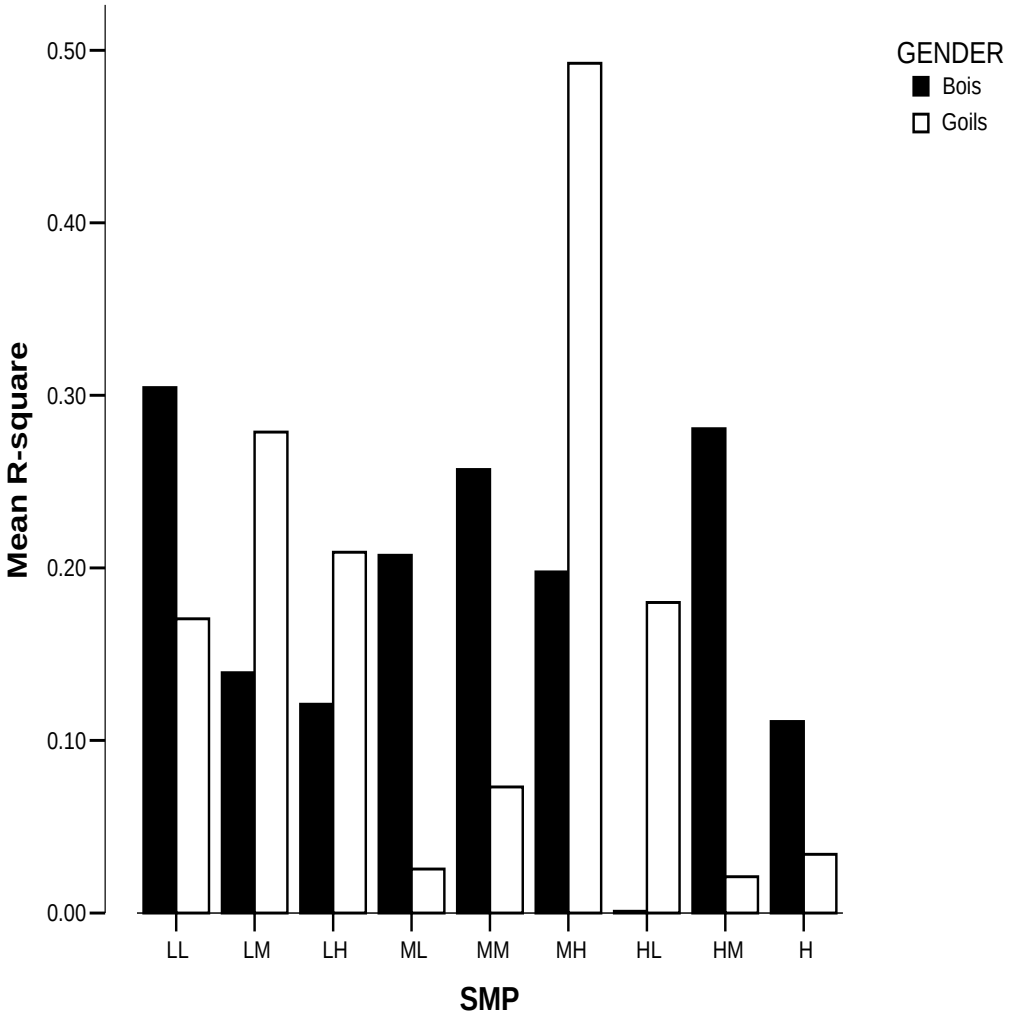


Figure 17 Association between Rank Change and Landscape by SMP and Gender: Total Partners

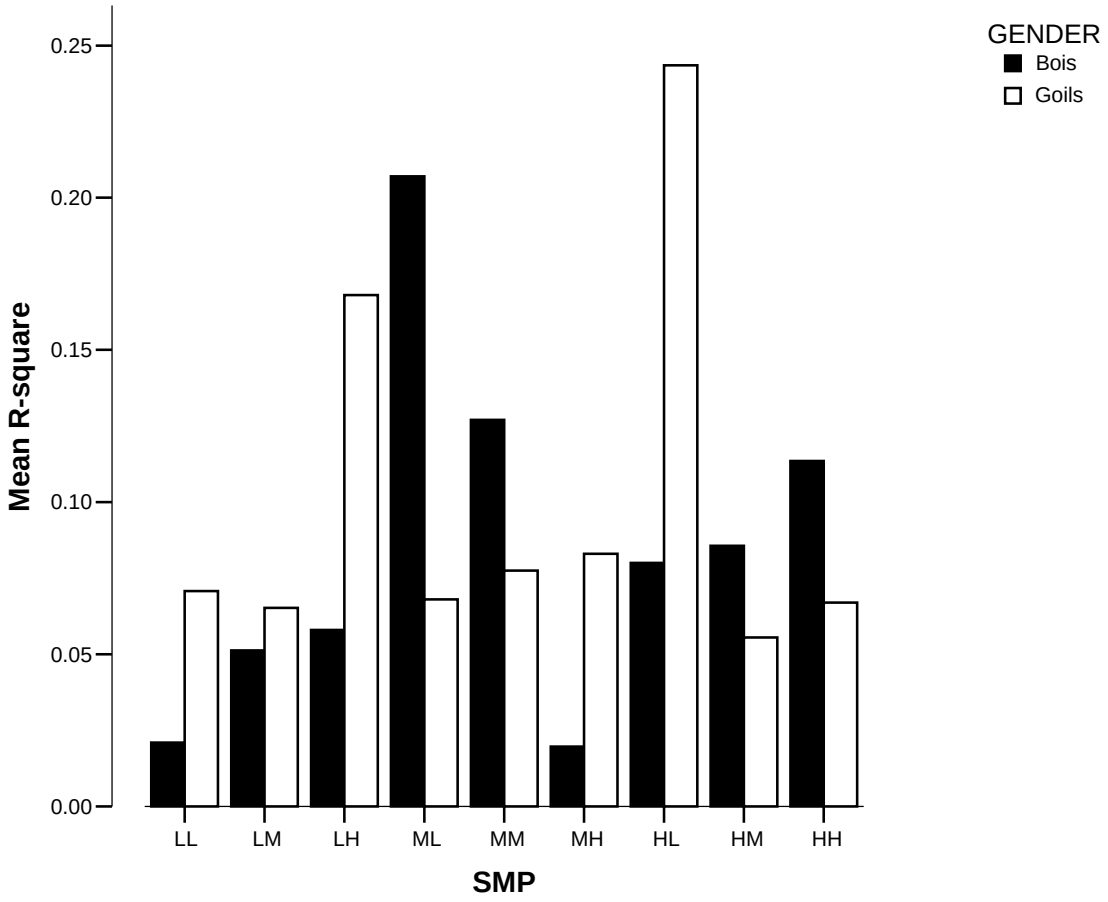


Figure 18 Association between Rank Change and Landscape by SMP and Gender: Percent Infected

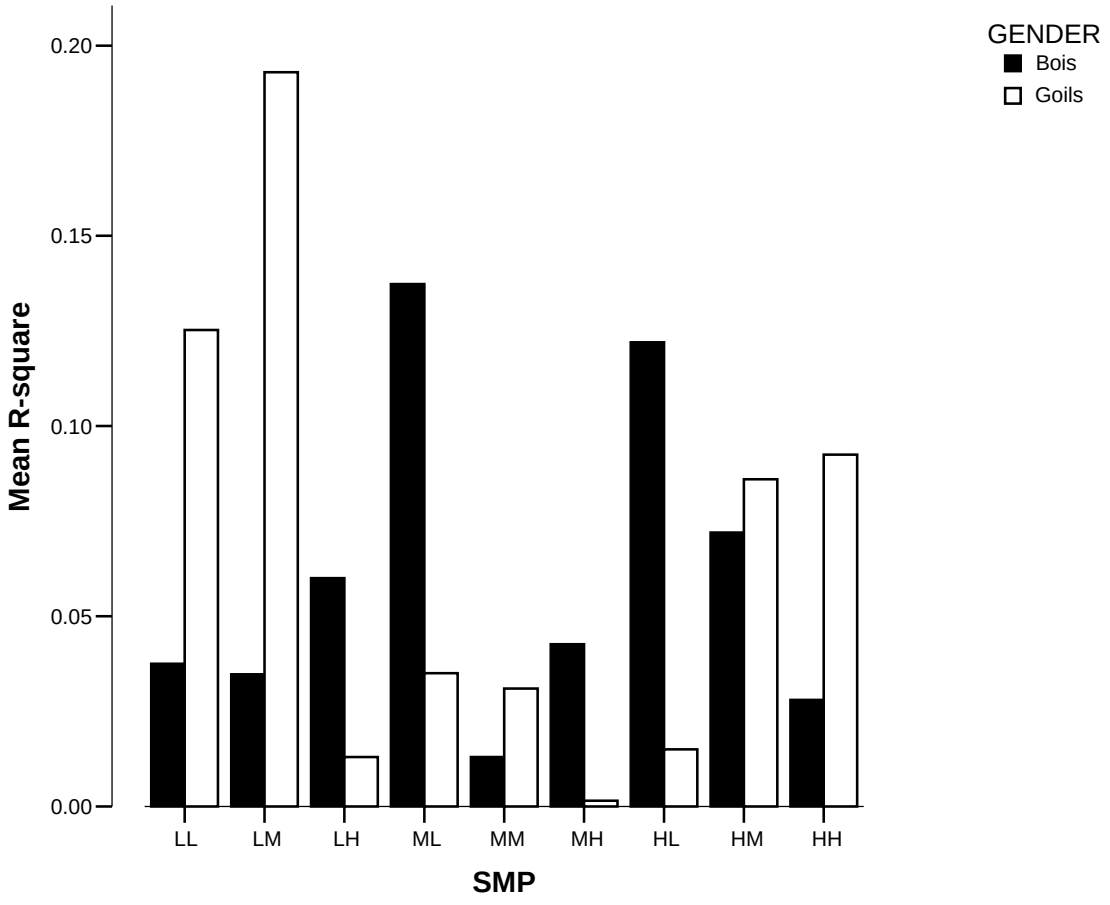


Figure 19 Distribution of Number of Partners – NORM Landscape Only

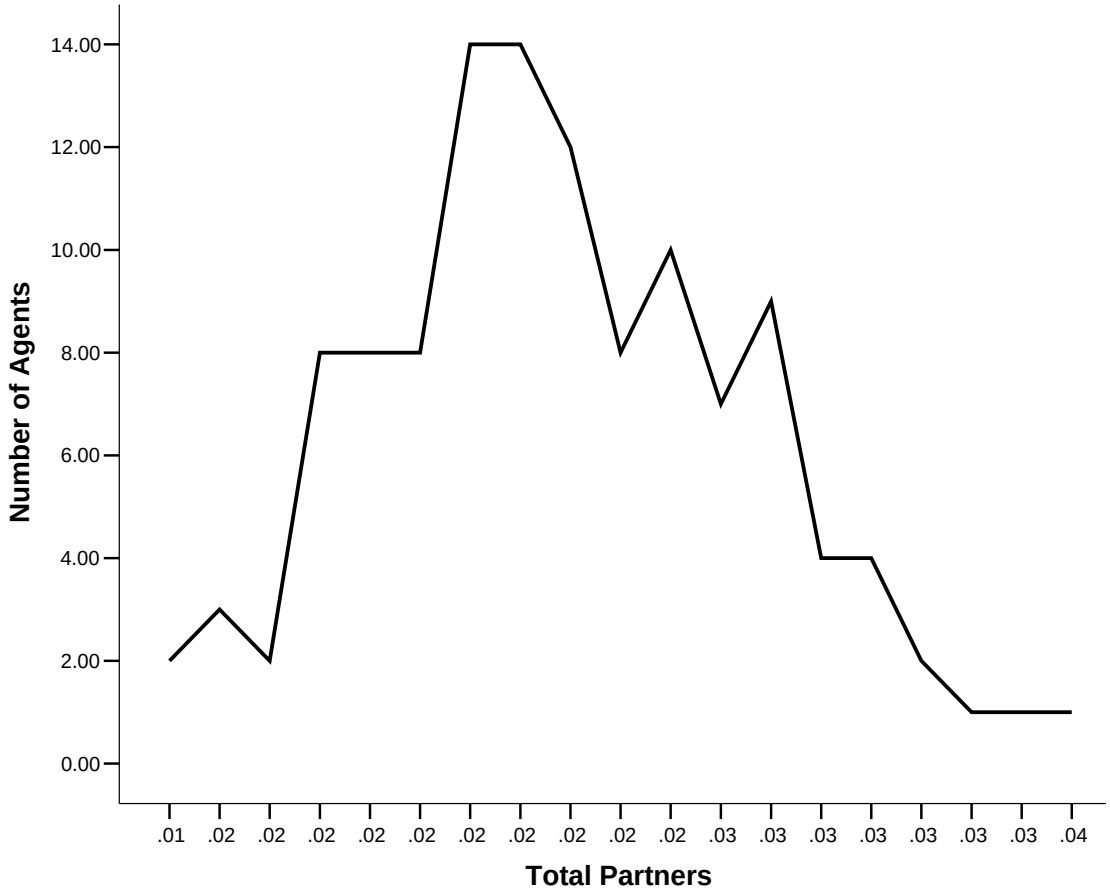
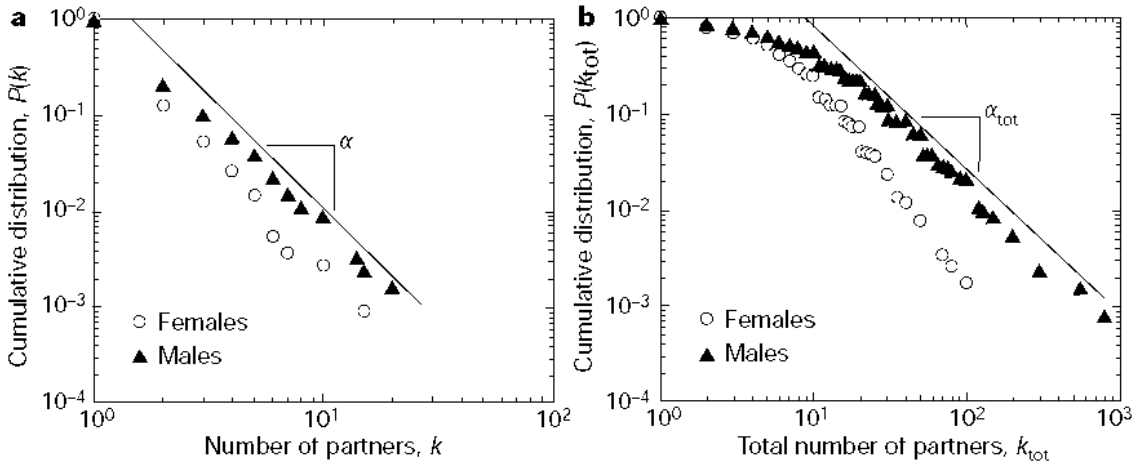
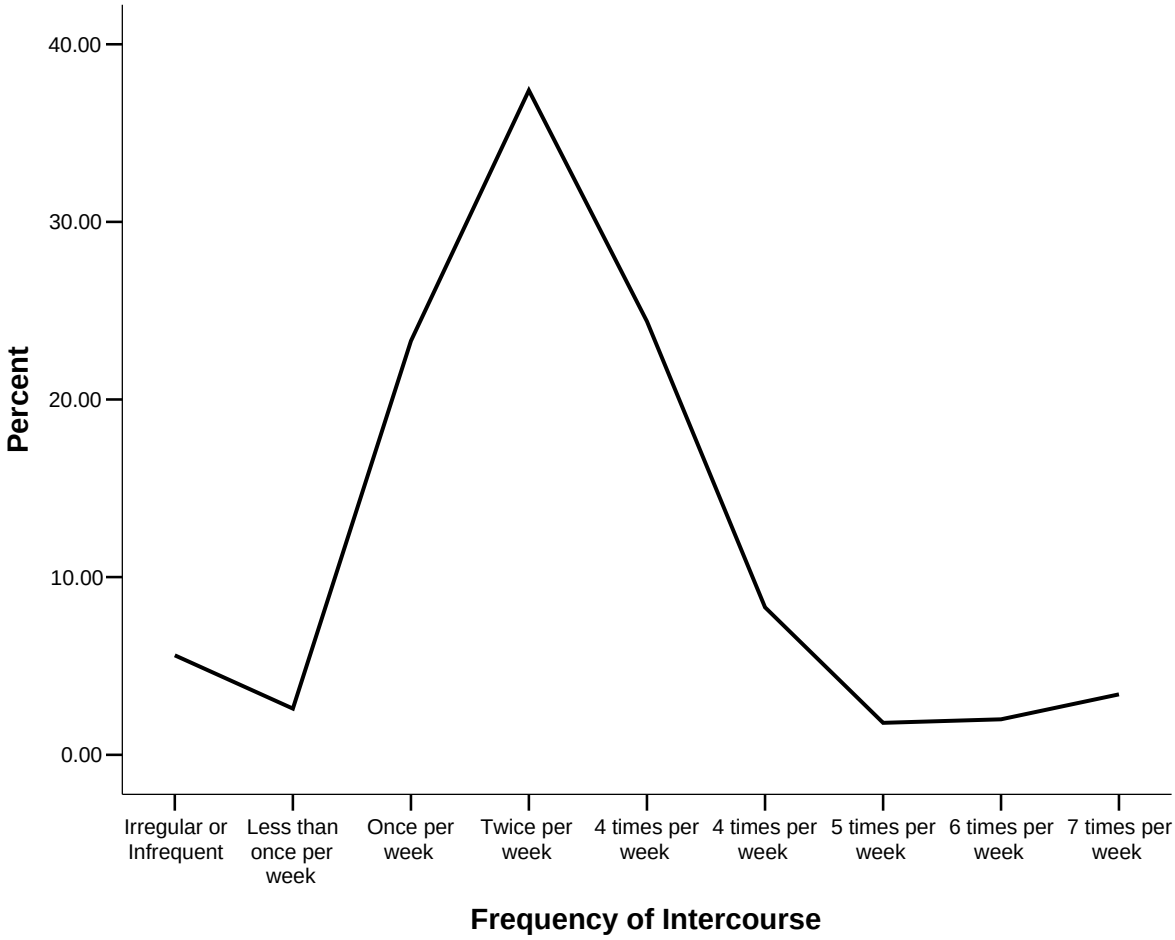


Figure 20 Scale Free Distribution of Number of Sex Partners



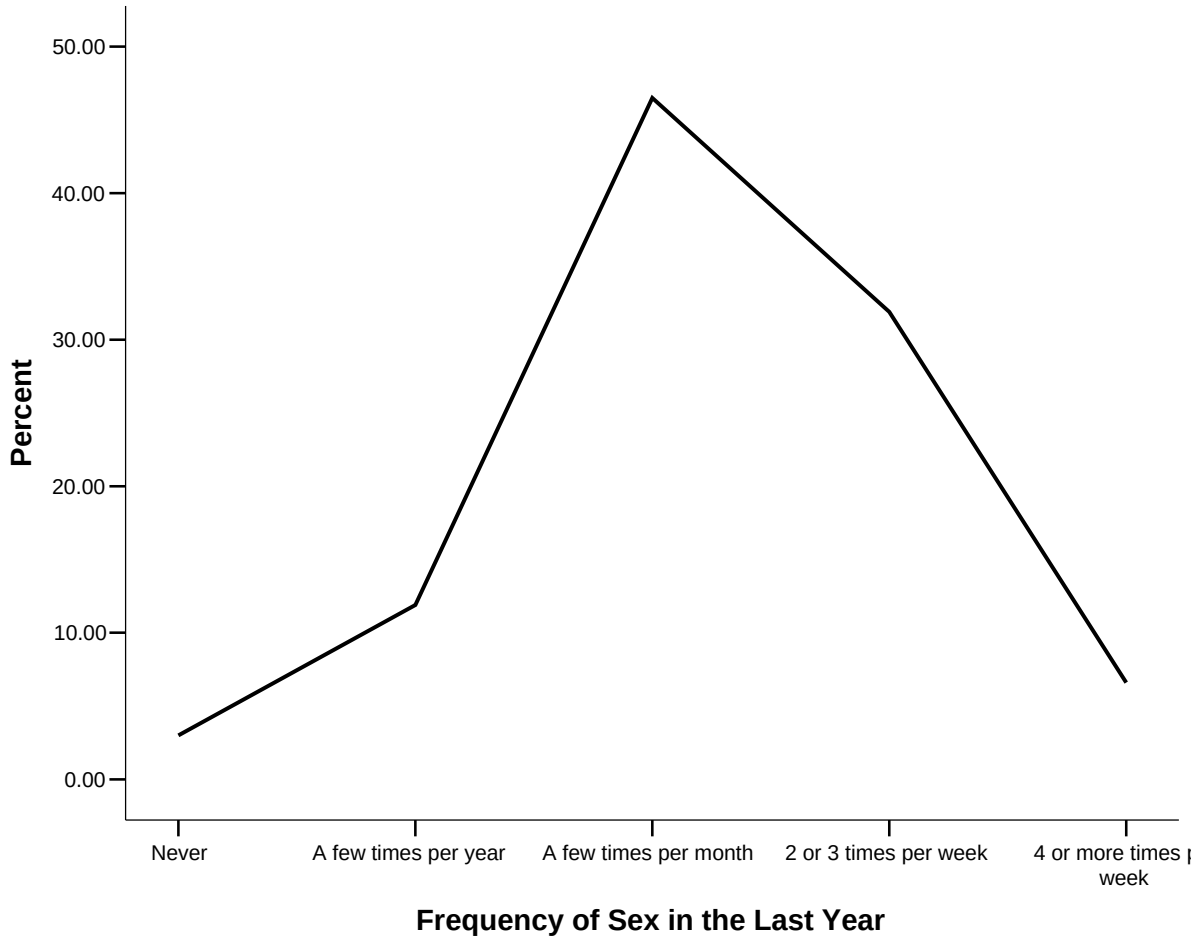
From Liljeros, et al, 2001

Figure 21 Frequency of Intercourse



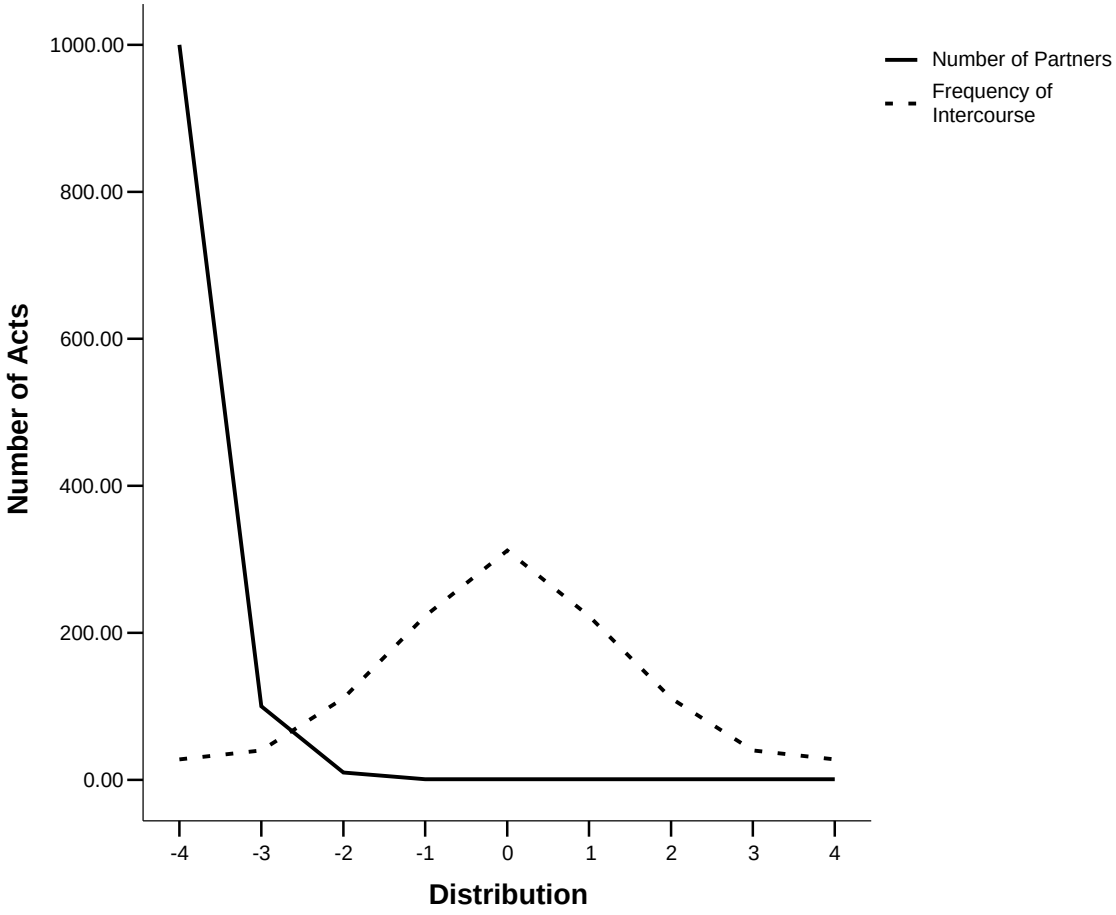
Adapted from Kopp, 1934

Figure 22 Frequency of Sex in the Past Year



Adapted from Laumann et al, 1994

Figure 23 Comparison of Theoretical Distributions of Number of Partners to Frequency of Intercourse over One Year



APPENDIX 1 Agent.cs

```
using System;
using System.Collections.Generic;
using System.Text;

namespace InfectionModel
{
    abstract public class Agent
    {
            protected readonly float SES_CELIBACY_INCREMENT = 0.01F;

            /// <summary>
            /// mate value on a 1-10 scale, 10=high
            /// </summary>
            protected int mate_value;

            public int MateValue {
                  get { return mate_value; }
                  set { mate_value = value; }
            }
        /// <summary>
        /// status of infection
        /// </summary>
            private bool infected;

            public bool Infected {
                  get { return infected; }
                  set { infected = value; }
            }
        /// <summary>
        /// x location on grid
        /// </summary>
            protected int x_location;

            public int X_location {
                  get { return x_location; }
                  set { x_location = value; }
            }
        /// <summary>
        /// y location on grid
        /// </summary>
            protected int y_location;

            public int Y_location {
                  get { return y_location; }
                  set { y_location = value; }
            }

        /// <summary>
        /// integer ID for this agent
        /// </summary>
            private int id;

            public int Id {
                  get { return id; }
                  set { id = value; }
```

```
    }
/// <summary>
/// list of PartnerData describing partners at time of coupling
/// </summary>
    protected List<PartnerData> partners;

    public List<PartnerData> Partners {
          get { return partners; }
          set { partners = value; }
    }
/// <summary>
/// timestep of infection
/// </summary>
    private long time_of_infection;

    public long TimeOfInfection {
          get { return time_of_infection; }
          set { time_of_infection = value; }
    }


    // Stats.

    /// <summary>
/// total # unique partners at infection time
/// </summary>
    private int num_partners_at_infection;
    public int NumPartnersAtInfection {
          get { return num_partners_at_infection; }
          set { num_partners_at_infection = value; }
    }

    private float excitationAtInfection;

    public float ExcitationAtInfection {
          get { return excitationAtInfection; }
          set { excitationAtInfection = value; }
    }
    private float inhibitionAtInfection;

    public float InhibitionAtInfection {
          get { return inhibitionAtInfection; }
          set { inhibitionAtInfection = value; }
    }
    private float infectingPartnerExcitation;

    public float InfectingPartnerExcitation {
          get { return infectingPartnerExcitation; }
          set { infectingPartnerExcitation = value; }
    }
    private float infectingPartnerInhibition;

    public float InfectingPartnerInhibition {
          get { return infectingPartnerInhibition; }
          set { infectingPartnerInhibition = value; }
    }
```

```
            ////////////////////////////////


            /// <summary>
            /// probability of infection from interaction with infected
agent
            /// </summary>
            protected float infection_probability;

            public float Infection_probability {
                  get { return infection_probability; }
                  set { infection_probability = value; }
            }

            /// <summary>
            /// % of time saying yes to proposition
            /// </summary>
            protected float acceptance_percent;
            /// <summary>
            /// raw count of # "yes" acceptances in respose to
propositions
            /// </summary>
            private long acceptance_count;

            public long AcceptanceCount {
                  get { return acceptance_count; }
                  set { acceptance_count = value; }
            }
            /// <summary>
            /// raw count of # incoming propositions
            /// </summary>
            private long incoming_proposition_count;

            public long IncomingPropositionCount {
                  get { return incoming_proposition_count; }
                  set { incoming_proposition_count = value; }
            }
            /// <summary>
            /// raw count of propositions to other agents
            /// </summary>
            private long outgoing_proposition_count;

            public long OutgoingPropositionCount {
                  get { return outgoing_proposition_count; }
                  set { outgoing_proposition_count = value; }
            }
            /// <summary>
            /// base SMS profile
            /// </summary>
            protected SMSProfile smsProfile;

            public SMSProfile SmsProfile {
                  get { return smsProfile; }
                  set { smsProfile = value; }
```

```
            }

            abstract public float Sis(long timestep);

            public float Ses(float timestep) {
                        // SES increases incrementally across timesteps
of celibacy.
                        return GetNumericSES(SmsProfile.Excitation) +
(timestep-last_mate_time) * SES_CELIBACY_INCREMENT;
            }

            public bool ExcitedByTracer(long timestep, float
tracerExcitationRatio) {
                  return (Ses(timestep) / Sis(timestep)) >
tracerExcitationRatio;
            }


            abstract public float GetNumericSIS(SMSLevel level);
            abstract public float GetNumericSES(SMSLevel level);

            /// <summary>
            /// time steps since mating
            /// </summary>
            private long last_mate_time;

            public long LastMateTime {
                  get { return last_mate_time; }
                  set { last_mate_time = value; }
            }

            /// <summary>
            /// alive or dead?
            /// </summary>
            protected bool alive = true;

            public bool Alive {
                  get { return alive; }
                  set { alive = value; }
            }



            protected readonly long timeOfBirth = 0;

            public long TimeOfBirth {
                  get { return timeOfBirth; }
            }


            protected long timeOfDeath = 0;

            public long TimeOfDeath {
                  get { return timeOfDeath; }
                  set { timeOfDeath = value; }
            }
```

```
		public long calculateLifespan(long maxTime) {
			if (!alive)
				return timeOfDeath - timeOfBirth;
			else
				return maxTime - timeOfBirth;
		}

		public long calculateLifespanAfterInfection(long maxTime) {
			if (!alive)
				return timeOfDeath - time_of_infection;
			else
				return maxTime - time_of_infection;
		}

		/// <summary>
		/// mating/searching state
		/// </summary>
		protected AgentActionState actionState;

		public AgentActionState ActionState {
			get { return actionState; }
			set { actionState = value; }
		}

		public Agent() {
			partners = new List<PartnerData>();
		}

		public Agent(long timeOfBirth) {
			partners = new List<PartnerData>();
			this.timeOfBirth = timeOfBirth;
		}

		protected void DiseaseFreeCopy(Agent dest, int newId) {
			dest.id = newId;
			dest.infection_probability = infection_probability;
			dest.mate_value = mate_value;
			dest.smsProfile = smsProfile;
		}

		abstract public Agent Clone(int newID, long timeOfBirth);

		public List<PartnerData> UniquePartners {
			get {
				List<PartnerData> unique = new
List<PartnerData>();
				bool found;
				foreach (PartnerData pAll in partners) {
					found = false;
					foreach (PartnerData pUnique in unique) {
						if (pUnique.id == pAll.id)
							found = true;
						break;
					}
					if (!found)
						unique.Add(pAll);
				}
```

```
                        return unique;
                    }
                }
            }
        }
```

```
using System;
using System.Collections.Generic;
using System.Text;
using System.ComponentModel;
using System.Windows.Forms;
using System.Threading;

namespace InfectionModel {
      public class Simulation {

            /// <summary>
            /// Enable the use of tracer grid in making agent
decisions.
            /// </summary>
            private bool enableTracer = false;

            public bool EnableTracer {
                  get { return enableTracer; }
                  set { enableTracer = value; }
            }

            /// <summary>
            /// Records SMP stats for min,max,average SES/SIS at each
timestep for each agent.
            /// </summary>
            private bool collectSMPStats = false;

            public bool CollectSMPStats {
                  get { return collectSMPStats; }
                  set { collectSMPStats = value; }
            }

            /// <summary>
            /// Are we in the process of running lots of simulations
sequentially, or just 1?
            /// </summary>
            private bool runningMany = false;
            private int simulationNum;

            public bool RunningMany {
                  get { return runningMany; }
                  set { runningMany = value; }
            }

            public int totalNumberAgents {
                  get { return agents.Count + deadAgents.Count; }
            }

            private int nextId;

            private bool stop;
            public bool Stop {
                  get { return stop; }
                  set { stop = value; }
            }
```

```
private bool paused;
public bool Paused {
      get { return paused; }
      set { paused = value; }
}



private struct DirectionVector {
      public int x;
      public int y;

      public DirectionVector(int xInit, int yInit) {
            x = xInit;
            y = yInit;
      }
}

private ShowProgressDelegate ShowProgress;
private ShowProgressManyDelegate ShowProgressMany;

/// <summary>
/// a list of all the living agents in the simulation
/// </summary>
private List<Agent> agents;

internal List<Agent> Agents {
      get { return agents; }
      set { agents = value; }
}

/// <summary>
/// a list of all the dead agents in the simulation
/// </summary>
private List<Agent> deadAgents;

internal List<Agent> DeadAgents {
      get { return deadAgents; }
      set { deadAgents = value; }
}

/// <summary>
/// grid contains pointers to agents in each square
/// </summary>
private Agent[,] grid;

/// <summary>
/// grid contains amounts of tracer in each
/// </summary>
private float[,] tracerGrid;

public float[,] TracerGrid {
      get { return tracerGrid; }
}

/// <summary>
```

```
            /// Max amount of tracer possible in a grid square.
            /// </summary>
            private const float TRACER_MAX_AMOUNT = 5.0f;

            /// <summary>
            /// random # generator
            /// </summary>
            private Random rand;
            /// <summary>
            /// current time step
            /// </summary>
            private long timestep;
            /// <summary>
            /// maximum time step
            /// </summary>
            /// <remarks></remarks>

            private SimulationStats stats;

            public SimulationStats Stats {
                  get { return stats; }
                  set { stats = value; }
            }

            /// <summary>
            /// ref to persisted simulation parameters
            /// </summary>
            private SimulationParameters parameters;

            public SimulationParameters Parameters {
                  get { return parameters; }
                  set { parameters = value; }
            }

            private List<SMSProfile> smsProfileList;

            public List<SMSProfile> SmsProfileList {
                  get { return smsProfileList; }
                  set { smsProfileList = value; }
            }

            private List<DirectionVector> directionVectors;

            public Simulation() {
                  // Load simulation details.
                  parameters =
Properties.Settings.Default.SimulationParams;
                  if(parameters == null) {
                        parameters = new SimulationParameters();
                        Properties.Settings.Default.SimulationParams =
parameters;
                  }

                  // Load all known SMS types.
                  smsProfileList =
Properties.Settings.Default.smsProfileList;
                  if (smsProfileList == null) {
```

```
                        smsProfileList = new List<SMSProfile>();
                        Properties.Settings.Default.smsProfileList =
smsProfileList;
                  }

#if DEBUG
                  rand = new Random(0);  // Use a fixed seed for
debugging.
#else
                  rand = new Random();
#endif

                  // Generate direction vectors.
                  directionVectors = new List<DirectionVector>(8);
                  for (int x = -1; x <= 1; x++)
                        for (int y = -1; y <= 1; y++)
                              if (!(x == 0 && y == 0))
                                    directionVectors.Add(new
DirectionVector(x, y));

            }

            /// <summary>
            /// runs the simulation the given # of times. returns stats
array as results.
            /// </summary>
            public SimulationStats[] RunManySimulations(int
numSimulations) {
                  SimulationStats[] stats = new
SimulationStats[numSimulations];

                  // Run the specified # of simulations, unless someone
presses the stop button.
                  for (simulationNum = 0; simulationNum <
numSimulations && !stop; simulationNum++) {
                        // Init simulation.
                        Initialize(ShowProgress);

                        // Run the simulation.
                        stats[simulationNum] = RunSimulation();

                        // Clean extra stuff from stats to save memory.
                        stats[simulationNum].RemoveExtraStats();

                        // Update the display.
                        ShowProgressMany(timestep, simulationNum);
                  }

                  return stats;
            }


            /// <summary>
            /// runs the simulation. returns stats as results.
            /// </summary>
            //public SimulationStats RunSimulation(GridControl gc,
Label lblStatus) {
```

```
            public SimulationStats RunSimulation() {
                  Agent[] agentsListFixed;

                  // Run the simulation until saturation or MAX_TIME.
                  for (timestep = 0; stats.time_to_saturation == 0 &&
                                                timestep <
parameters.MaxTime - 1 &&
                                                !stop; timestep++) {

                        if (enableTracer) {
                              // Allow tracer to evaporate.
                              for (int x = 0; x <=
tracerGrid.GetUpperBound(0); x++) {
                                    for (int y = 0; y <=
tracerGrid.GetUpperBound(1); y++) {
                                          float tracer = tracerGrid[x,
y];
                                          // TODO: pick evaportation
formula.
                                          tracerGrid[x, y] = tracer -
parameters.TracerEvaporationRate; // evaporate!
                                          // Make sure tracer doesn't
go negative.
                                          if (tracerGrid[x, y] < 0)
                                                tracerGrid[x, y] = 0;
                                    }
                              }
                        }

                        // Iterate over agents; choose action for each.

                        // Copy agent list so it doesn't get modified
by death.
                        agentsListFixed = new Agent[agents.Count];
                        agents.CopyTo(agentsListFixed);

                        // Alternate agent decision order at each
timestep.
                        Monitor.Enter(this);
                        try {
                              if (timestep % 2 == 0) {
                                    for (int i = 0; i <
agentsListFixed.Length; i++) {

      SimulateAgentDecision(agentsListFixed[i]);
                                    }
                              } else {
                                    for (int i = agentsListFixed.Length
- 1; i >= 0; i--) {

      SimulateAgentDecision(agentsListFixed[i]);
                                    }
                              }

                              // Only update time-based stat recording
if running 1 simulation only.
                              if (!runningMany) {
```

```
                                    // Update stats.

      stats.total_infected_at_time[timestep + 1] =
stats.total_infected_at_time[timestep];

      stats.total_infected_at_time_float[timestep + 1] =
stats.total_infected_at_time[timestep];

      stats.percent_infected_at_time_float[timestep + 1] =
stats.total_infected_at_time_float[timestep + 1] /

                                                totalNumberAgents;
                                    foreach (SMSStats smsStats in
stats.by_sms) {

      smsStats.total_number_bois_at_time[timestep + 1] =
smsStats.total_number_bois_at_time[timestep];

      smsStats.total_number_goils_at_time[timestep + 1] =
smsStats.total_number_goils_at_time[timestep];

      smsStats.total_infected_bois_at_time[timestep + 1] =
smsStats.total_infected_bois_at_time[timestep];

      smsStats.total_infected_goils_at_time[timestep + 1] =
smsStats.total_infected_goils_at_time[timestep];

      smsStats.total_infected_bois_at_time_float[timestep + 1] =
smsStats.total_infected_bois_at_time[timestep];

      smsStats.total_infected_goils_at_time_float[timestep + 1] =
smsStats.total_infected_goils_at_time[timestep];

      smsStats.percent_infected_bois_at_time_float[timestep + 1] =
smsStats.total_infected_bois_at_time_float[timestep + 1] /


      smsStats.total_number_bois_at_time[timestep + 1];

      smsStats.percent_infected_goils_at_time_float[timestep + 1] =
smsStats.total_infected_goils_at_time_float[timestep + 1] /


      smsStats.total_number_goils_at_time[timestep + 1];
                                          //if
(smsStats.total_number_agents_at_time[timestep + 1] == 0)
                                          //          throw new
Exception("0 agents in SMSStats.");

                                          // TODO.
                                    }
                              }

                              if (runningMany) {
                                    if (timestep % 100 == 0) {
                                          // Update the display.
```

```
                                        ShowProgressMany(timestep,
simulationNum);
                                  }
                            } else {
                                  // Update the display.
                                  ShowProgress(timestep);
                            }


                      }
                      finally {
                            Monitor.Exit(this);
                      }

                      // Test for pause button click.
                      while (paused) {
                            Thread.Sleep(200);      // Sleep until
pause button is pressed again.
                      }
                }

                // Copy agent lists into final stats list.
                stats.agents = new Agent[agents.Count +
deadAgents.Count];
                agents.CopyTo(stats.agents);
                deadAgents.CopyTo(stats.agents, agents.Count);

                // Fill in stats details.
                stats.RecordAverages();
                return stats;
          }

          private void SimulateAgentDecision(Agent agent) {

                // If desired, update stats based on each agent's
properties at each time step.
                if (collectSMPStats) {
                      RecordSMPStats(agent);
                }

                // test for death.
                if (!agent.Alive) return;

                // If agent is infected, check for death due to
disease.
                if (agent.Infected && timestep -
agent.TimeOfInfection >= parameters.TimeToDeath) {
                      KillAgent(agent);
                      return;
                }

                // Make a decision based on current state and SMS.
                switch (agent.ActionState) {
                      case AgentActionState.Searching:
                            ConsiderMates(agent);
```

```
                              // Make sure the agent didn't start
mating; if not, move away.
                              if (agent.ActionState ==
AgentActionState.Searching)
                                    MoveAgent(agent);

                              break;
                        case AgentActionState.MatingTimeStep1:
                              agent.ActionState =
AgentActionState.MatingTimeStep2;
                              break;
                        case AgentActionState.MatingTimeStep2:
                              agent.ActionState =
AgentActionState.MatingTimeStep3;
                              break;
                        case AgentActionState.MatingTimeStep3:
                              if (agent is Boi) {
                                    agent.ActionState =
AgentActionState.Refactory;

      ((Boi)agent).Refactory_time_remaining =
parameters.RefactoryTimeSteps;
                              }
                              else
                                    agent.ActionState =
AgentActionState.Searching;
                              break;
                        case AgentActionState.Refactory:
                              Boi b = (Boi)agent;

                              // MOVE AWAY!
                              MoveAgentRel(b, b.RefactoryXDirection,
b.RefactoryYDirection);

                              // Either go back to searching in next
time step or mainting state
                              // but decrease refraction time.
                              if (b.Refactory_time_remaining <= 0)
                                    b.ActionState =
AgentActionState.Searching;
                              else
                                    b.Refactory_time_remaining--;

                              break;
                        default:
                              throw new
ArgumentOutOfRangeException("ActionState", "Invalid AgentActionState");
                  }
            }

            /// <summary>
            /// Record stats for SMP groups based on individual agent
properties at each timestep of simulation.
            /// </summary>
            /// <param name="agent"></param>
            private void RecordSMPStats(Agent agent) {
                  // Update SMP stats.
```

```
                  AverageGroupStats ags =
stats.averages.getAgentGroup(agent);
                  ags.agentTimesteps++;
                  float ses, sis;
                  ses = agent.Ses(timestep);
                  sis = agent.Sis(timestep);
                  if (ses < ags.minSes) ags.minSes = ses;
                  if (sis < ags.minSis) ags.minSis = sis;
                  if (ses > ags.maxSes) ags.maxSes = ses;
                  if (sis > ags.maxSis) ags.maxSis = sis;
                  ags.totalSis += sis;
                  ags.totalSes += ses;
            }

            // Dead -- kill it.
            private void KillAgent(Agent agent) {
                  agent.Alive = false;
                  deadAgents.Add(agent);
                  agents.Remove(agent);
                  grid[agent.X_location, agent.Y_location] = null;
                  // Make a new one and add to grid.
                  Agent a = agent.Clone(nextId++, timestep);
                  agents.Add(a);
                  AssignFreeGridLocation(a);

                  // Update stats: add one to total agent count of this
type due to new birth.
                  SMSStats stat = FindSmsStat(a);
                  if (a is Boi)
                        stat.total_number_bois_at_time[timestep]++;
                  else
                        stat.total_number_goils_at_time[timestep]++;

                  // Record time of death.
                  a.TimeOfDeath = timestep;
            }

            // Check out possible mates.
            private void ConsiderMates(Agent agent) {
                  List<Agent> potentialMates = new List<Agent>(8);
                  Agent otherAgent;
                  Boi b;
                  Goil g;
                  int x, y;

                  // Loop through all directions and make a list of
agents that are potential mates.
                  foreach (DirectionVector dv in directionVectors) {
                        // Map the direction vector to a particular
square.
                        FindMoveDest(agent, dv.x, dv.y, out x, out y);

                        // Find agent in destination square.
                        otherAgent = grid[x, y];
                        if (otherAgent == null) continue;

                        // Make sure agent is of opposite sex.
```

```
                        if (agent.GetType() == otherAgent.GetType())
continue;

                        // Make sure other agent is not busy mating.
                        if (otherAgent.ActionState ==
AgentActionState.MatingTimeStep1 ||
                              otherAgent.ActionState ==
AgentActionState.MatingTimeStep2 ||
                              otherAgent.ActionState ==
AgentActionState.MatingTimeStep3)
                              continue;

                        // Make sure the other agent generates large
enough excitation to initiate a mate proposition.
                        if (WouldMate(agent, otherAgent)) {
                              // Add it!
                              potentialMates.Add(otherAgent);
                        }
                  }

                  // If no mates found, nothing to do. Return.
                  if (potentialMates.Count == 0)
                        return;

                  // Randomly pick a potential mate and make a
proposition.
                  otherAgent =
potentialMates[rand.Next(potentialMates.Count)];
                  // Update stats for proposition counts.
                  agent.OutgoingPropositionCount++;
                  otherAgent.IncomingPropositionCount++;

                  // Test outcome of proposition.
                  if (WouldMate(otherAgent, agent)) {
                        // Accepted. Update simulation.
                        agent.ActionState =
AgentActionState.MatingTimeStep1;
                        otherAgent.ActionState =
AgentActionState.MatingTimeStep1; //TODO: Note subtelty: if A is before
B in

                                    //processing in list, B will
advance an extra stage in a moment....
                        // Dump some tracer at both agents' locations.
                        if (enableTracer) {
                              AddTracer(agent.X_location,
agent.Y_location);
                              AddTracer(otherAgent.X_location,
otherAgent.Y_location);
                        }

                        // Update stats.
                        agent.LastMateTime = timestep;
                        otherAgent.LastMateTime = timestep;
                        otherAgent.AcceptanceCount++;

                        // Record partner data.
```

```
                        agent.Partners.Add(new PartnerData(otherAgent,
timestep, otherAgent.Ses(timestep), otherAgent.Sis(timestep)));
                        otherAgent.Partners.Add(new PartnerData(agent,
timestep, agent.Ses(timestep), agent.Sis(timestep)));

                        // For bois, set refactory direction.
                        if (agent is Boi) {
                              b = (Boi)agent;
                              g = (Goil)otherAgent;
                        } else {
                              g = (Goil)agent;
                              b = (Boi)otherAgent;
                        }

                        GetNegDirectionBetween(b, g, out x, out y);
                        b.RefactoryXDirection = x;
                        b.RefactoryYDirection = y;

                        // Test each direction for infection.
                        if (agent.Infected && !otherAgent.Infected) {
                              if (rand.NextDouble() <
otherAgent.Infection_probability) {
                                    // Oops.. Just infected the other
agent. Update.
                                    InfectAgent(otherAgent, agent);
                              }
                        } else if (otherAgent.Infected &&
!agent.Infected) {
                              if (rand.NextDouble() <
agent.Infection_probability) {
                                    // Oops.. Just got infected.
Update.
                                    InfectAgent(agent, otherAgent);
                              }
                        }
                  } else {
                        // Declined. Update stats.

                        // TODO.

                  }
            }

            /// <summary>
            /// Increment the amount of tracer at the specified x,y
location.
            /// Tracer caps out at 5. Currently this just increments
tracer by 1.0
            /// with a max of 5.0.
            /// </summary>
            /// <param name="x"></param>
            /// <param name="y"></param>
            private void AddTracer(int x, int y) {
                  tracerGrid[x,y] += 1.0f;
                  if(tracerGrid[x,y] > TRACER_MAX_AMOUNT)
                        tracerGrid[x,y] = TRACER_MAX_AMOUNT;
            }
```

```
            /// <summary>
            /// Update stats for becoming infected.
            /// </summary>
            /// <param name="agent"></param>
            /// <param name="infectingAgent"></param>
            private void InfectAgent(Agent agent, Agent infectingAgent)
{
                  agent.Infected = true;
                  agent.TimeOfInfection = timestep;
                  agent.NumPartnersAtInfection =
agent.UniquePartners.Count;
                  stats.total_infected_at_time[timestep]++;
                  stats.total_infected_at_time_float[timestep] =
stats.total_infected_at_time[timestep];

                  SMSStats smsStat = FindSmsStat(agent);

                  if (agent is Boi) {

      smsStat.total_infected_bois_at_time[timestep]++;

      smsStat.total_infected_bois_at_time_float[timestep] =
smsStat.total_infected_bois_at_time[timestep];
                  } else {

      smsStat.total_infected_goils_at_time[timestep]++;

      smsStat.total_infected_goils_at_time_float[timestep] =
smsStat.total_infected_goils_at_time[timestep];
                  }

                  agent.ExcitationAtInfection = agent.Ses(timestep);
                  agent.InhibitionAtInfection = agent.Sis(timestep);
                  agent.InfectingPartnerExcitation =
infectingAgent.Ses(timestep);
                  agent.InfectingPartnerInhibition =
infectingAgent.Sis(timestep);
            }

            private SMSStats FindSmsStat(Agent agent) {
                  foreach (SMSStats stat in stats.by_sms)
                        if (agent.SmsProfile == stat.smsProfile)
                              return stat;

                  throw new Exception("Agent's sms profile not found in
stats.by_sms");
            }


            // Answers the question "Would this initiator mate with
receiver?"
            private bool WouldMate(Agent initiator, Agent receiver) {
                  return (initiator.Ses(timestep) * receiver.MateValue
/ parameters.ExcitationDivisor > initiator.Sis(timestep));
            }
```

```
            // Move towards potential mates.
            private void MoveAgent(Agent agent) {
                  int x, y, xOffset, yOffset;
                  double xCentroid = 0, yCentroid = 0,
curMateValueMass;
                  float tracer;
                  double distance, angle;
                  Agent dest;

                  // Calculate the centroid of MateValue in the agent's
L1 neighborhood,
                  // where MateValues are scaled down proportional to
the squared distance.

                  for (xOffset = -parameters.VisionRadius; xOffset <=
parameters.VisionRadius; xOffset++) {
                        for (yOffset = -parameters.VisionRadius;
yOffset <= parameters.VisionRadius; yOffset++) {
                              // Skip the agent itself.
                              if (xOffset == 0 && yOffset == 0)
continue;

                              // Make sure there is an agent in
candidate square.
                              FindMoveDest(agent, xOffset, yOffset, out
x, out y);
                              dest = grid[x,y];
                              if (dest == null) continue;

                              // Make sure agent is of opposite sex.
                              if (agent.GetType() == dest.GetType())
continue;

                              // There is an agent. Calculate squared
distance.
                              distance = Math.Abs(xOffset) *
Math.Abs(xOffset) + Math.Abs(yOffset) * Math.Abs(yOffset);

                              // Now, calculate tracer amount in the
square, if enabled.
                              if (enableTracer) {
                                    float tracerExcitationRatio =
(agent is Boi) ?

      parameters.TracerExcitationRatioBois :
parameters.TracerExcitationRatioGoils;

                                    if (agent.ExcitedByTracer(timestep,
tracerExcitationRatio))
                                          tracer = tracerGrid[x, y];
                                    else
                                          tracer = -tracerGrid[x, y];
                              } else {
                                    tracer = 0;
                              }
```

```
                              // Add to centroid. Not actual centroid,
but vector is correct direction. Would need
                              // to sum mateValueMass and divide by it
latest to get actual centroid. But
                              // we'll just take vector arctangent
anyway so it doesn't matter.
                              curMateValueMass = ((dest.MateValue +
tracer) / distance); // Effective mate value due to distance.
                              xCentroid += xOffset * curMateValueMass;
                              yCentroid += yOffset * curMateValueMass;
                        }
                  }

                  // Map the centroid vector onto a neighbor-square
vector constrained to -1,0,1 in either direction.
                  // 8 possibilities (moves to adjacent grid points)
based on angle.

                  // If vector is (0,0), pick a random direction.
                  if (Math.Abs(yCentroid) < 0.01 && Math.Abs(xCentroid)
< 0.01) {
                        DirectionVector dir =
directionVectors[rand.Next(8)];
                        xOffset = dir.x;
                        yOffset = dir.y;
                  } else {
                        // Calculate angle from 0 to 2Pi of vector.
                        angle = Math.Atan2(yCentroid, xCentroid); //
Branch cut thing at Pi/-PI on -x axis.

                        if (angle < -0.875 * Math.PI || angle >= 0.875
* Math.PI) { // Between -7Pi/8 and -Pi or 7Pi/8 to Pi radians.
                              xOffset = -1;
                              yOffset = 0;
                        } else if (angle < -0.625 * Math.PI) { //
Between -5Pi/8 and -7Pi/8.
                              xOffset = -1;
                              yOffset = -1;
                        } else if (angle < -0.375 * Math.PI) { //
Between -5Pi/8 and -3Pi/8.
                              xOffset = 0;
                              yOffset = -1;
                        } else if (angle < -0.125 * Math.PI) { //
Between -3Pi/8 and -1Pi/8.
                              xOffset = 1;
                              yOffset = -1;
                        } else if (angle < 0.125 * Math.PI) { //
Between -Pi/8 and Pi/8.
                              xOffset = 1;
                              yOffset = 0;
                        } else if (angle < 0.375 * Math.PI) { //
Between Pi/8 and 3Pi/8.
                              xOffset = 1;
                              yOffset = 1;
                        } else if (angle < 0.625 * Math.PI) { //
Between 3Pi/8 and 5Pi/8.
                              xOffset = 0;
```

```
                        yOffset = 1;
                    } else {//if (angle < 0.876 * Math.PI) { //
Between 5Pi/8 and 7Pi/8.
                        xOffset = -1;
                        yOffset = 1;
                    } //else
                        //throw new ArithmeticException("angle
not defined: angle = " + angle);
                }

                // Move towards the centroid!
                MoveAgentRel(agent, xOffset, yOffset);
            }

            // Calculate the distance vector between two neighboring
agents, for use in refactory direction calculation.
            // Returns negative of result, to simplify things.
            void GetNegDirectionBetween(Agent a, Agent b, out int x,
out int y) {
                x = a.X_location - b.X_location;
                y = a.Y_location - b.Y_location;
                if (x < -1)
                    x = 1;
                else if (x > 1)
                    x = -1;

                if (y < -1)
                    y = 1;
                else if (y > 1)
                    y = -1;
            }



            private void MoveAgentRel(Agent agent, int xOffset, int
yOffset) {
                int newX, newY;

                // Verify no agents in destination square...
otherwise pick new random direction if possible.
                // Chooses a good square to move the agent to.
                PickFreeDestination(agent, xOffset, yOffset, out
newX, out newY);

                // Clear grid to prepare move.
                grid[agent.X_location, agent.Y_location] = null;

                // Move the agent.
                agent.X_location = newX;
                agent.Y_location = newY;

                // Move on the grid.
                grid[agent.X_location, agent.Y_location] = null;
                grid[newX, newY] = agent;
            }
```

```
            // Finds an empty destination square, preferably the one
specified. But picks another randomly
            // if the destination is full.
            private void PickFreeDestination(Agent agent, int xOffset,
int yOffset, out int x, out int y) {
                  DirectionVector dir;
                  Agent dest;
                  int count=0;

                  FindMoveDest(agent, xOffset, yOffset, out x, out y);
                  dest = grid[x, y];

                  while (dest != null && count++ < 3) {
                        dir = directionVectors[rand.Next(8)];     //
Pick a random direction.
                        FindMoveDest(agent, dir.x, dir.y, out x, out
y);
                        dest = grid[x, y];
                  }

                  // If no free square found, just leave the agent in
its current square.
                  if (dest != null) {
                        x = agent.X_location;
                        y = agent.Y_location;
                  }
            }

            // Calculates a destination square after move with torus
wraparound.
            private void FindMoveDest(Agent agent, int xOffset, int
yOffset, out int x, out int y) {
                  /// Find the X coord.
                  x = agent.X_location + xOffset;
                  if (x >= this.parameters.GridSize)
                        x = 0;
                  else if (x < 0)
                        x = this.parameters.GridSize - 1;

                  // Find the Y coord.
                  y = agent.Y_location + yOffset;
                  if (y >= this.parameters.GridSize)
                        y = 0;
                  else if (y < 0)
                        y = this.parameters.GridSize - 1;
            }


            /// <summary>
            /// resets the simulation, creates agents, etc
            /// </summary>
            public void Initialize(ShowProgressDelegate
showProgressDelegate) {
                  ShowProgress = showProgressDelegate;
                  agents = new List<Agent>(parameters.NumAgents);
                  deadAgents = new List<Agent>(parameters.NumAgents*8);
```

```
                  grid = new Agent[parameters.GridSize,
parameters.GridSize];
                  tracerGrid = new float[parameters.GridSize,
parameters.GridSize];
                  stats = new SimulationStats(parameters.MaxTime,
smsProfileList);
                  stop = false;
                  paused = false;

                  timestep = 0;

                  // Create the agents in the grid based on settings
for
                  // SMS distribution and bois/goils ratio. We first
add bois, then goils.
                  for(int i = 0; i < parameters.NumAgents; i++) {

                        Agent agent;

                        // Choose a gender.
                        if(i < (parameters.NumBois)) {
                              // It's a boi!
                              agent = new Boi();
                        } else {
                              // It's a goil!
                              agent = new Goil(rand.NextDouble());
                        }

                        // Set ID.
                        agent.Id = nextId++;

                        // Set mate value.
                        agent.MateValue = (i % 10) + 1;  // Mate value
varies from 1-10 with an equal # of each.

                        // Add to the list.
                        agents.Add(agent);

                        // Add to the grid: select a free x,y spot.
                        AssignFreeGridLocation(agent);

                        // Set the agent SMS and other params.
                        // SMS depends on the landscape and the current
agent # we're initializing.
                        if (agent is Boi) {
                              decimal percent=0; // This is the current
percent of the way through the profiles.

                              foreach (SMSPercentage smsPercent in
parameters.SmsPercentages) {
                                    // Tally the current position in
the smsProfile list.
                                    percent +=
smsPercent.PercentageBois;
                                    if (percent > 100)
```

```
                                          throw new
ArgumentOutOfRangeException("SMS percent bois > 100%! can't generate
agents.");

                                    if (i < percent *
parameters.NumBois / 100) {
                                          // We found the right
profile. Set agent profile to this one.
                                          agent.SmsProfile =
smsPercent.SmsProfile;
                                          // We're done.
                                          break;
                                    }

                              }
                        } else {
                              // It's a goil.
                              decimal percent=0; // This is the current
percent of the way through the profiles.

                              foreach (SMSPercentage smsPercent in
parameters.SmsPercentages) {
                                    // Tally the current position in
the smsProfile list.
                                    percent +=
smsPercent.PercentageGoils;
                                    if (percent > 100)
                                          throw new
ArgumentOutOfRangeException("SMS percent goils > 100%! can't generate
agents.");

                                    if (i - parameters.NumBois <
percent * parameters.NumGoils / 100) { // adjust for leading bois in
list.
                                          // We found the right
profile. Set agent profile to this one.
                                          agent.SmsProfile =
smsPercent.SmsProfile;
                                          // We're done.
                                          break;
                                    }
                              }
                        }

                        // Update SMS stats.
                        SMSStats stat = FindSmsStat(agent);
                        if (agent is Boi)
                              stat.total_number_bois_at_time[0]++;
                        else
                              stat.total_number_goils_at_time[0]++;

                  } // for

                  // Done making agents!

                  // Infect one at random.
                  int n = rand.Next(parameters.NumAgents);
```

```
                  agents[n].Infected = true;

                  // Update stats for initial infection.
                  agents[n].TimeOfInfection = 0;
                  stats.total_infected_at_time[0] = 1;
                  SMSStats statInitial = FindSmsStat(agents[n]);

                  if (agents[n] is Boi) {
                        statInitial.total_infected_bois_at_time[0] = 1;

      statInitial.total_infected_bois_at_time_float[0] = 1;

      statInitial.percent_infected_bois_at_time_float[0] = 1.0F /
statInitial.total_number_bois_at_time[0];
                  } else {
                        statInitial.total_infected_goils_at_time[0] =
1;

      statInitial.total_infected_goils_at_time_float[0] = 1;

      statInitial.percent_infected_goils_at_time_float[0] = 1.0F /
statInitial.total_number_goils_at_time[0];
                  }
            }

            // Add agent to the grid: select a free x,y spot.
            private void AssignFreeGridLocation(Agent agent)
            {
                  int x, y;

                  do
                  {
                        x = rand.Next(parameters.GridSize);
                        y = rand.Next(parameters.GridSize);
                  } while (grid[x, y] != null);

                  agent.X_location = x;
                  agent.Y_location = y;
                  grid[x, y] = agent;
            }


            public void InitializeMany(ShowProgressManyDelegate
showProgressManyDelegate) {
                  ShowProgressMany = showProgressManyDelegate;
                  runningMany = true;
            }
      }


      public delegate SimulationStats RunSimulationDelegate();
      public delegate SimulationStats[] RunManySimulationsDelegate(int
numSimulations);

}
```

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EDUCATION

2006 Ph.D.	Health Behavior, minor Human Sexuality (Anticipated) Department of Applied Health Science, School of Health, Physical Education, and Recreation Indiana University, Bloomington, Indiana
2004	One-Week Intensive Seminar, Complex Physical, Biological, and Social Systems New England Complex Systems Institute, Boston, Massachusetts
2002 M.S.	Counseling and Counselor Education Department of Counseling and Educational Psychology School of Education Indiana University, Bloomington, Indiana
1998 B.A.	Psychology, minors Cognitive Science and Philosophy Department of Psychology College of Arts and Sciences University of Delaware, Newark, Delaware

TEACHING EXPERIENCE

2003-present — *Associate Instructor*, Indiana University, Bloomington, Indiana

- Human Sexuality (HPER-F 255)
  Fall 2003 (98 students), Fall 2004 (71 students), Spring 2005 (148 students), Summer 2005 (12 students), Fall 2005 (150 students); Spring 2006 (90 students); Summer 2006 (12 students)
- Practicum in College Sex Education (HPER-H 350/540)
  Fall 2003 (8 students), Fall 2005 (3 students); Summer 2006 (4 students)
- Marriage and Family Interaction (HPER-F 258)
  Spring 2004 (75 students), Spring 2005 (71 students)
- Leading Family Process Discussion Groups (HPER-H 355)
  Spring 2004 (5 students)
- Stress Management (HPER-H 180)
  Summer 2004 (30 students)

2001 — *Discussion Leader*, HPER-H 540 Practicum in College Sex Education

TEACHING COMPETENCY

Human Sexuality, Marriage and Family Interaction, College Health Education Methods, Health Counseling, Counseling Theories, Theories of Health Behavior, Evolutionary Psychology, Health Psychology, Personality Psychology, Abnormal Psychology, Qualitative and Quantitative Social Science Research Methods

EMILY NAGOSKI

RESEARCH INTERESTS

Sexual motivation; high risk sexual behavior and sexual compulsivity; evolutionary perspectives on human sexuality; gender differences in sexuality; primate sexuality; computational modeling of complexity in social systems; attachment and human relationships; undergraduate sexual and reproductive health education.

PUBLICATIONS IN PREPARATION

Lohrmann, D., Angermeier, L., and Nagoski, E. (in preparation). Infrastructure status assessment for a coordinated school health program: a case study.

Nagoski, E. (in preparation). Mothering and Mating: parental investment, the dual control model, and women's sexual motivation.

Nagoski, E. and Janssen, E. (in preparation). A Map of Sexual Motivation.

POSTERS AND PRESENTATIONS

Butler, S., Nagoski, E, and Herbenick, D. (2005, October). *Peers in the Classroom: Using Peer Facilitators to Enhance Human Sexuality Discussion Sessions.* Paper presented at the Mid America College Health Association Conference, Lexington, KY.

Nagoski, E. (2005, July). *Different for Goils: an agent based model of parental investment and sexual motivation.* Paper presented at the World Congress on Sexology, Ontario, Canada.

Nagoski, E. (2005, June). *Different for Goils: an agent based model of parental investment and sexual motivation.* Poster session presented at the national conference of the Human Behavior and Evolution Society Conference, Austin, TX.

Butler, S., Herbenick, D., and Nagoski, E. (2005, May). *Contemporary Pedagogic Strategies to Undergraduate Human Sexuality Courses: Approaches at Indiana University and Other Institutions*. Poster session presented at the national conference of the American Association of Sex Educators, Counselors, and Therapists Conference, Portland, OR.

Nagoski, E. (2004, November). *Community-Based HIV Prevention Maximized for Complex Systems.* Poster session presented at the national conference of the American Public Health Association Conference, Washington, D.C.

Nagoski, E. (2004, May*). Sexual Bodies, Sexual Mind, Sexual World: Accounting for Interaction with a Complex Systems Approach to Sex Research.* Paper presented at the Society for the Scientific Study of Sexuality Midwest and Eastern Regional Conference, Madison, WI.

Nagoski, E and Brown, K. (2004, May). *The Role of a Speakers Bureau in Facilitating a Supportive Campus Climate.* Paper presented at the American Association of Sex Educators, Counselors, and Therapists Conference, Chicago, IL.

Nagoski, E. (2002, August). *Training GLBT Allies: an Experiential Approach*. Poster session presented at the national conference of the American Psychological Association Conference, Chicago, IL.

Nagoski, E. (2001, February). *College Campuses, the Internet, and Sexuality*. Paper presented at the Midwestern Bisexual, Lesbian, Gay, Transgender, and Allies College Conference, Urbana-Champaign, IL.

Nagoski, E. (2000, August). *Counselor Responses in an Online Assessment: Toward a Research Agenda*. Poster session presented at the national conference of the American Psychological Association Conference, Washington, D.C.

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ADVISING AND CAMPUS LIFE WORK EXPERIENCE

2006	*Health Educator* – Health and Wellness Education, Indiana University Health Center, Bloomington, Indiana Present sexual health education programming to small groups of undergraduates in residence halls. Programs emphasize reproductive choices and STI prevention.
2002-2003	*Academic Advisor* – University Division, Indiana University, Bloomington, Indiana Advised students in the selection of courses and academic programs, university policies and procedures, and development of academic, career, and personal goals. Supported students on academic probation and presented educational programs about admission requirements.
2001-2002	*Counseling Intern* – Gay, Lesbian, Bisexual, and Transgender Student Support Services Office, Indiana University, Bloomington, Indiana Coordinated student groups, conducted group, couples, and individual counseling, and provided training for peer supporter undergraduates, with gay, lesbian, bisexual, and transgender undergraduates and their allies.
2001-2002	*Director of Public Relations* – Graduate and Professional Student Organization, Indiana University, Bloomington, Indiana Designed and maintained web page, designed fliers and other advertising media, coordinated subcommittees, and organized university-wide social functions for graduate students.
2000-2002	*Project Coordinator* – Kinsey Institute Sexuality Information Service for Students, Bloomington, Indiana Designed, implemented, and evaluated interactive online sexuality information service for undergraduates. Wrote content, designed web page, and monitored traffic to site. Provided sexual health programs for undergraduate residence halls and classrooms.
2000, Spring	*Graduate Assistant* – Residential Programs and Services, Indiana University, Bloomington, Indiana Supervised 8 undergraduate Resident Assistants and 400 undergraduates in residence halls.
1999, Fall	*Resident Assistant* – Residential Programs and Services, Indiana University, Bloomington, Indiana Supervised 50 graduate and undergraduate residents in residence hall for students over 21.

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FELLOWSHIPS, GRANTS, AND AWARDS

2006	Foundation for the Scientific Study of Sexuality Student Research Grant
2005	Indiana University School of Health, Physical Education, and Recreation Grant-in-Aid of Research
2005	Indiana University School of Health, Physical Education, and Recreation Travel Grant
2004	Indiana University School of Health, Physical Education, and Recreation Doctoral Fellowship
2004	Indiana University School of Health, Physical Education, and Recreation Travel Grant
2004	Indiana University Student Alumni Association Student Choice Award for Teaching Nominee
1998	University of Delaware Senior Psychology Research Award
1998	Dean's List, University of Delaware
1997	Dean's List, University of Delaware

PROFESSIONAL AFFILIATION

2005-present	Society for the Scientific Study of Sexuality (SSSS)
2004-present	American Public Health Association (APHA)
2004-present	American Association of Sex Educators, Counselors, and Therapists (AASECT)

VOLUNTEER WORK

2005-present	*Host*, "It's Only Sex" segment on "bloomingtOUT" radio show, WHFB, Bloomington, Indiana; http://podcastalley.com/podcast_details.php?pod_id=5510
2003-present	*Volunteer*, Gay, Lesbian, Bisexual, and Allies Speakers Bureau, Bloomington, Indiana
2002-2004	*Crisis Line Volunteer*, Middleway House Domestic Violence Shelter, Bloomington, Indiana
2000-2002	*Volunteer Organizer*, IU Allies, Indiana University, Bloomington, Indiana
1999-2001	*Peer Educator*, Raising Awareness of Interactions in Sexual Encounters (RAISE), Indiana University, Bloomington, Indiana
1996-1998	*Sexual Health Peer Educator*, Wellspring, University of Delaware, Newark, Delaware

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