

SimARC: An Ontology-driven Behavioural Model of Alcohol Abuse

Francois Lamy
CRICS
Charles Sturt University
Bathurst, Australia
flamy1978@gmail.com

Pascal Perez
SMART Infrastructure
UOW
Wollongong, Australia
pascal.perez@uow.edu.au

Alison Ritter
NDARC
UNSW
Sydney, Australia
alison.ritter@unsw.edu.au

Michael Livingston
TPADC
Uni. of Melbourne
Melbourne, Australia
michaell@turningpoint.org.au

Abstract—Alcohol-related problems (assaults, accidents and/or crimes) and alcohol abuse are recurrent societal problems leading to high social costs. Finding adapted policies to tackle this issue isn't a trivial task due to the highly complex nature of alcohol consumption as many interrelated risk factors interact in a hardly predictable way. This paper describes an agent-based simulation model, called SimARC (Simulation of Alcohol-Related Consequences), aiming at exploring the complex interplay of these factors following a generative process whereby theory and model co-evolve within iterative loops. To explore the complexity of alcohol use and abuse, we need not only to include the aforementioned risk factors but also their evolution and highly dynamical interactions across scales. Therefore, our agent-based model aims to encapsulate several levels of reality. Considering an ontology as catalog of elements and relation amongst those elements, our ontology-driven behavioral model includes: neuro-biological responses to alcohol use (individual level), peer influence channeled through various social networks (meso-level) and societal responses to alcohol-related problems (meta-level). This ontological framework aims to establish a robust test-bed to analyze – in silico – the plausible consequences of various public policies related to alcohol abuse in public venues. After a brief review of the literature, we present SimARC's core structure and preliminary results.

Keywords—agent-based model; ontology; alcohol; social simulation; public health.

I. INTRODUCTION

In its «Global Status Report on Alcohol and Health 2011», World Health Organization (WHO) points that alcohol «is a causal factor in more than 60 major types of diseases and injuries and results in approximately 2.5 million deaths each year [...] Thus, 4% of all deaths worldwide are attributable to alcohol» [1]. Furthermore, a recent report from the Independent Scientific Committee on Drugs (ISCD) indicates via that alcohol, in term of social cost, is more dangerous than heroin and crack [2]. In the same vein, Collins and Lapsley have estimated at 15.3 billions AU\$ (11.6 billions €) the social cost of alcohol [3].

Moreover, both in Europe and Australia, «binge drinking» (heavy drinking session leading to intoxication) is on the rise inducing greater chances of individual harms (i.e., falls, pedestrian/car accidents) as well as greater risks of violence (i.e., brawl, degradation, violent assaults) [4][5].

Due to its legal status and large availability alcohol has become a major health problem for governments [6][7] who generally attempt to solve this problem by different combinations of public policies, such as alcohol taxation, prevention campaigns or reduction in availability [8][9]. Beside, net revenues associated with alcohol consumption largely make up for subsequent expenditures in the Australian federal budget [3].

Hence, alcohol-related social harm remains a difficult research topic [10] as consumption patterns adapt quickly to new policies. For example, individual change their drinking habits (i.e., "preloading" episode, shifting from one type of alcohol to another one) or license premises adapting their marketing to remain competitive. As well as all the others drug uses, alcohol consumption and its aftermaths are complex social phenomena: they result from the interaction of many risk and protective factors that dynamically evolve through time [11].

These factors belong to distinct levels of analysis: genetic predispositions; neurophysiology and neuro-pharmacology of alcohol; individual psychology; social and environmental conditions; current laws; economical constrains or cultural norms [12]. We consider here three levels of analysis: a micro-level (the individual, his neurologic, physiologic and social characteristics), a meso-level (groups, peer influence and significant others) and a macro-level (public policies, urban geography and societal responses).

Our work aims to create a social simulation, which integrates three levels of analysis in order to get a better understanding of alcohol use and misuse. Once calibrated and validated, this type of simulation model could be used to inform policy-making debate on alcohol [13]. To describe this simulation, we will review the different components of the model, then we will discuss the need for new technologies to capture alcohol-related problems, and finally, we will describe the different components of SimARC and show some preliminary results.

II. ALCOHOL USE: A MULTI-FACTORIAL SYSTEM

Our three different levels represent three levels of interactions between five components: alcohol, individual, group, context and society. We consider the relation between the alcohol and individual components constitutes the *prima causa* of alcohol-related harms.

A. Micro-level: Alcohol/Individual

Alcohol is a powerful psychoactive substance highly addictogen. BAC (Blood Alcohol Concentration) is the main indicator of alcohol intoxication and impairment. BAC gives good indications concerning cognitive and motor impairment: the following figure (cf. Figure 1) illustrates the relation between accident and BAC [14].

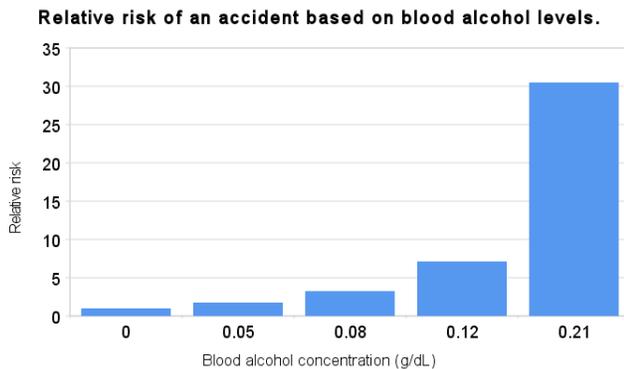


Figure 1. Risk of traffic accident/BAC

However, aggressive behaviors are consequences of the neural action of alcohol on the brain. Ethanol, the active principle of alcohol, has an impact on many neurotransmitters, but Dopamine, GABA, Glutamate and Serotonin (5-HT) are strongly related to behavioral changes [15]. As most of the other drugs, alcohol generates release of Dopamine, the neurotransmitter of reward and pleasure. From behavioral viewpoint, dopamine increases self-confidence, and happiness; however, it is also considered as one of the key-factors that trigger craving, addiction and schizophrenia [16].

Alcohol also acts on GABA (Gamma-Amino-Butyric Acid) the principal inhibitory neurotransmitter in the brain. In standard volume, GABA has a relaxant effect, but higher doses cause drowsiness and motor impairments [17]. Another role of GABA is to balance the excitatory action of Glutamate. At normal dose, this neurotransmitter is implied in learning and memorization [18], inversely, higher concentration of Glutamate in the brain leads to "excitotoxicity", impairing or killing neurons.

Finally, ethanol interacts strongly with Serotonin. Also known as 5-hydroxytryptamine (5-HT), this neurotransmitter is responsible for mood regulation, sleeping cycles and thermoregulation. Mild dose brings euphoria and a sentiment of happiness. Conversely, depleted serotonin level is

generally correlated with feeling of depression and aggressive behaviors [19].

From a neurophysiologic viewpoint, during alcohol consumption the level of Glutamate in the brain decreases with a correlative increase in GABA concentrations, giving a mild relaxant feeling and desinhibition. At the same time, the agonist effect of alcohol on Dopamine and Serotonin neurotransmitters induces euphoria as well as feelings of happiness and self-confidence in the user [20]. Inversely, excessive amounts of GABA and low rates of Glutamate lead to motor impairment, lack of concentration and potentially induce sleep, increasing dramatically the chance of being involve in a car or pedestrian accident.

Once alcohol slowly disappears from the bloodstream and "stocks" of those neurotransmitters have been partially or completely depleted, a "calm down" period starts and users sense the different side effects following their consumption. Individuals will start to feel depress, get moody or exhibit violent behaviors (due to dopamine and serotonin depletions). Furthermore, some neurons may be damaged due to the rise of glutamate (excitotoxicity) after drinking [21].

This short review of the neuro-pharmacology of alcohol gives a partial explanation of behavioral changes but remains insufficient if it is not linked to social reactions and especially peer influence.

B. Meso-level: Individual/Group

Sociology has studied the impact of peer perception and influence regarding alcohol and drug use. Making reference to social learning theory, Kilpatrick et al. [22] and Flay et al. [23] have shown that children witnessing drug consumption from "significant others" (parents, sibling or tutors) have an increased risk of substance abuse. Obviously this influence can be extended to other elements of personal interactions, "peer pressure" has an important influence on experimental alcohol and drug use. On this subject, a vast literature exists about the social influence of friendship groups [24] indicating that individuals are influenced (positively or negatively) by their friends but also select which peers they have to mix with in order to find and use any drugs [25].

If peer-pressure has been the object of many studies as a risk factor, "social control" coming from members of the family, friends or community consist a solid protective factor [26]. Drug users compare their behaviors and consumption to other consumer compartments: irrational or erratic behaviors are generally banned and stigmatized [27]. However, repeated public misbehaviors around a particular location and/or generalization of alcohol-fuelled violence and disorders call to societal and political responses.

C. Macro-level: Social Environment/all components

As pointed by Livingston, the density of alcohol-related venues is directly related to violence in all neighborhood type, but bars and nightclubs are associated with violence in

the inner city while packaged alcohol outlets were associated with violence in suburban zone [28][29]. Similarly, the social capital of neighborhood seems to decrease with the density of alcohol outlet leading to more incivilities and to a possible social segregation [30].

Inside those venues, measures as closing times [31], limitation of crowding and a coordinated staff [32] have significant positive impact on alcohol-related violence.

Alcohol price taxation is accurately associated to alcohol-consumption: in their review of the different studies done on the subject, Chaloupka et al. indicated that increasing the monetary prices of alcoholic beverages reduces significantly alcohol consumption and alcohol-related problems [33].

Having reviewed those different factors, we need to find a robust framework able to encapsulate these components and capture their inter-evolutions over time. Hence, we propose to employ computer simulation to mimic this social phenomenon.

III. AGENT-BASED MODEL AND SOCIAL SIMULATION CONCERNING ALCOHOL

Computer simulation models have attracted an increasing number of researchers and practitioners over the last decade. As a matter of fact, social simulations can be used as artificial social experiments (*in-silico*) to explore the consequences of pre-defined conditions on a range of specific social and environmental indicators. In his seminal book ‘*Generative Social Science*’, Epstein argues that computer simulations provide new tools for integrative and empirical research in social sciences [34].

A particular instance of computer simulation, called Agent-Based Modelling (ABM), allows building artificial societies from the bottom-up; whereby individual autonomous agents interact, communicate and pursue personal goals while societal norms and regulations constrain their freedom [35]. ABM is also very helpful for collecting and making sense of dynamical (spatial movements, time series) or heterogeneous information (qualitative, quantitative, ill-defined or aggregated).

Finally, ABM is largely used in environmental, health or defence studies to explore intervention scenarios with policy makers [36]. According to Liu and Eck, “*crime simulation is [also] an emerging research area that has the potential of revealing hidden processes behind urban crime patterns and criminal justice system operations*” [37]. Again, the analytical value of the approach doesn’t rely on its capacity to describe spatiotemporal dynamics, but – more importantly – on its ability to assess different hypothesis about social causality [38].

In the field of alcohol and other drugs use, ABM has been successfully used to explore mechanisms of drug use initiation [39], and impacts of different policing interventions on street-based illicit drug markets [40]. Agent-based simulations concerning alcohol experiences gossiping amongst student [41], interactions agent-environment [42] or

movement of alcohol user in the city [43] have mainly studied agent/group or agent/environment interactions.

Our aim is to encapsulate both neurologic physiology, impact of the network on decision, geographic data and societal response in a single model. Computer science concept of ontology seems to tally with our objectives. Originally, ontology was a philosophical concept which, a branch of metaphysics: coming from *ontos* (being) and *logos* (discourse), ontology aims to describe general properties of things. For our purpose, we will consider ontology as a *description of a particular domain defined by its objects, concepts, and their properties and relations* [44]. This framework enables the description of the previous data and concepts in a common language, Unified Modeling Language (UML) (cf. Figure 2):

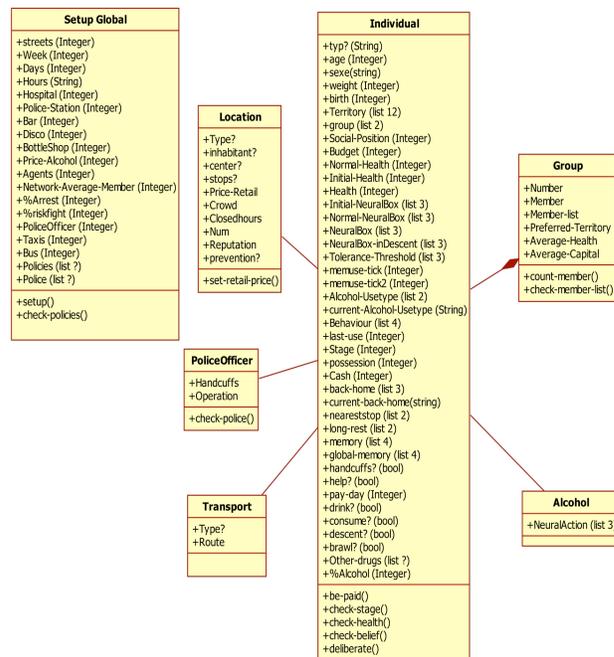


Figure 2. SimARC Class Diagram

IV. SIMARC: GLOBAL FUNCTIONNING

As indicated by Ferber [45], a Multi-Agent System (MAS) comprises of the following elements:

- An environment (E), a space that generally has a volume;
- A set of passive objects (O) which can be perceived, created, destroyed and modified by the agents;
- An assembly of agents (A) representing the active set of objects;
- An assembly of relations (R) that link active or passive agents to each other;
- An assembly of operations (Op) making it possible for the agents of A to act on objects from O.

SimARC aims to encompass all those different components. Besides that some part of this simulation are still under construction, we coded the UML structure in Netlogo 4.1.3 [46]. SimARC interface allows the experimenter to choose the number of Streets, Bar, Disco, Bottle-shop, Hospital and Police station. Sliders help to choose how many Agents and Constables will be created.

Finally, the simulation user can select Alcohol Price, Police Operations and Public Policies (those two latter are pre-implemented by the programmer). He can also select via sliders the percentage for a constable to arrest an alcohol user and the "brawl risk" percentage.

In the two next sections, we will describe the manner in which we have implemented most of our algorithms and the last section will give some preliminary results.

A. SimARC Urban Environment and Interface

The visual interface is a drastic simplification of an urban area, the grid includes the following features: street (here in black), house (green), bar (blue), disco (purple), bottle-shop (orange), police station (red), hospital and a rehab centre.

The Figure 3 gives an outline of the urban environment.

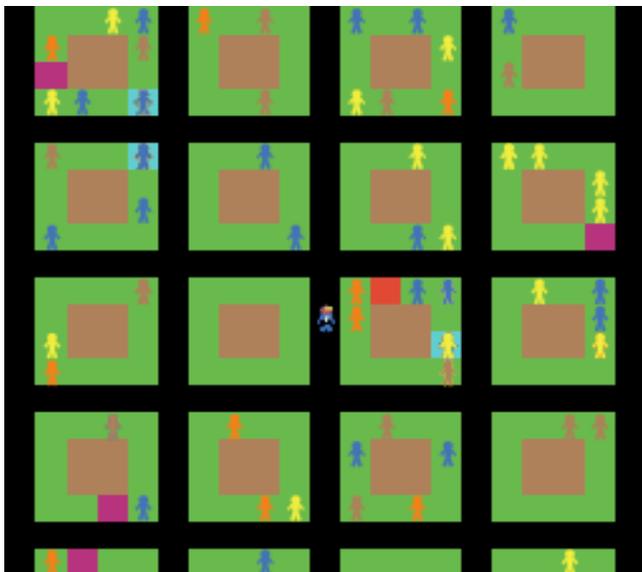


Figure 3. SimARC Urban Grid

Licensed premises (*venues*) have different Retail Prices and characteristics (Happy-Hours, Lock-out, Curfew, Crowding) as well as 'Reputation'. Every step (tick in Netlogo) represents 2 hours time, 12 ticks a real weekday and weekend (Monday, Saturday etc.).

The Retail Price varies according to the type of venues: bottle-shops have their retail price equal to the price chooses by the experimenter; bar sees this price increases by 2 and discos have a retail price multiply by 2. According to the Reputation of the venue, constables may be more incline to patrol in that neighborhood and some agents can just avoid this venue. Actually and according to the implemented

Public Policies, Retail Prices can increase for every alcohol-venues, but Happy-Hours can also be suppressed and/or Curfews or Lock-outs can be imposed. These different policies influence the choice and drinking patterns of the virtual alcohol users.

B. SimARC Agents and Networks

Each agent is characterized by the following attributes:

- Physical attributes (*Health, Age, Gender, BAC*);
- Status of neurotransmitters (*Serotonin, GABA, Glutamate and Dopamine*);
- A *Stage* representing its frequency of alcohol use and a correlated *Alcohol-routine*;
- Behavioral tendencies (aggressive, neutral or elusive)
- Memories of past experiences (past consumption, accidents, violence and sickness);
- Strategy to "get-back-home" once the night-out is over (private or public transport);
- Social characteristics (*Income, Friends, Address, Favorite Venues*).

An agent acts according to a series of heuristics based on an hourly schedule. All agents have a routine "daily-life": they go to work (part of their *Address* data), earn virtual money every fortnight (*Income*), eventually, decide to have a drink and finally, come back home to rest (restore their *Health* and *Status of neurotransmitters*). Agents earn ten times their *Income* (normal-distribution) every fortnight (randomly predefined). This fortnight income constitutes "pocket money" for non-essential expenses. The average amount of this pocket money is equal to 180.

Alcohol consumption varies according to each agent's *Alcohol-routine* and *Stage*: some agents may have a few drinks in their favorite bar during the weekend while others can have several binge-drinking sessions at home during weekdays. Some agents are just staying home and sober the whole week, resulting in no individual harm or social trouble. Other agents consuming large quantities of alcohol can display violent or dangerous comportments (brawl, accident and having been sick are counted and memorized) and these heavy drinking decrease their *Health* attribute.

Each "Drink" represents a Standard Drink (10 g of alcohol) and each intake increases the BAC of male agent by $10/(\text{Weight} \times 0.7)$ and by $10/(\text{Weight} \times 0.6)$ for female agent. BAC is reduced by 0.15 every tick (2 hours time).

Each consumption modifies the levels of neurotransmitters and the relative balance of neurotransmitters governs changes in behavioral patterns. An agent might change his opinion about alcohol consumption based on its cumulative experience of negative consequences (personal or witnessed) during or after successive night-outs. In turn, these updated opinions might change an agent's *Alcohol-routine* and *Stage*.

Furthermore, agents can interact through physical co-location in the spatial environment or through messages amongst friendship networks. Therefore, agents of a same

network move all together in *Favorite Venues*. Those *Friends* of the network can also "ask" an agent with a low *Health* or frequent dangerous behaviors to "slow down": if it accepts, the agent will not drink for some weeks, recovering from its past consumptions, otherwise, it will change its primary network to find new drinking mates.

C. Preliminary Results

In this section, we present some preliminary results from SimARC. However, this simulation hasn't been entirely calibrated yet. Therefore, these experimentations intend to test the internal consistency of the model. To do so, we examine the consequences of alcohol taxation policy. Four different prices have been tested 1, 5, 10 and 15 (relatively to the average 180 "pocket money").

For this experiment, the virtual population is composed of 750 agents and 2 constables. Those latter have 1% chance to arrest users with a $BAC > 0.5$. In order to initiate the simulation, we consider that 70% of the agents start with a Stage = 1, 15% with a Stage 2, 10% with a Stage 3 and 5% with Stage 4 (those data have to be calibrated).

For each scenario, we have run 50 replicates of 4367 time steps (one year simulation time). We have measured quantities of alcohol consumed for each scenario as well as the number of accidents and fatal accidents.

Figure 4 summarizes our results on Standard Drink consumption depending of the price of alcohol:

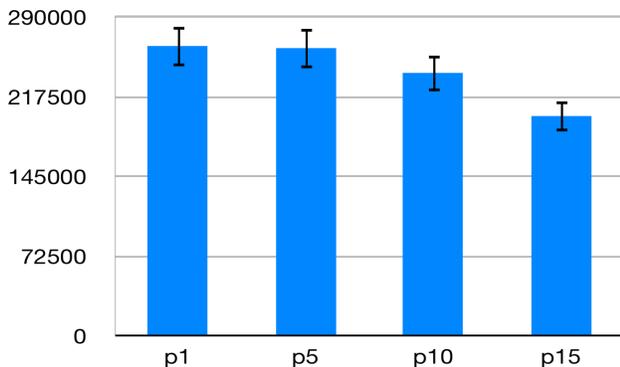


Figure 4. Alcohol Consumption (SD)/Price of Alcohol

Alcohol consumption decreases of 8.6% between P5 and P10, and decreases of 23.6% between P5 and P15. Those results seem to be concordant with economic studies [33]. However, it seems surprising that the amount of alcohol consumed for P1 and P5 are quasi-equivalent: we attribute this proximity of SD consumed to the "social control" operates by peers and to evolution of individual opinions in response to bad experiences during heavy drinking sessions (see IV.B).

Concerning Accidents with have implemented an algorithm matching the relative risk to be involved in an accident shown in Figure 1. Risk increases with BAC according to the following equation:

$$p(\text{crash}|BAC) = 1 / (1 + 0.2 \exp(5 - 2 * \%BAC)).$$

According to MUARC (Monash University Accident Research Centre) on alcohol-related car crashes provoke fatal accidents in approximately 1% of cases, and cause serious wounds in 34,5% and 64,5% constitute minor trauma [47]. Our experiments display the following results (Figure 5):

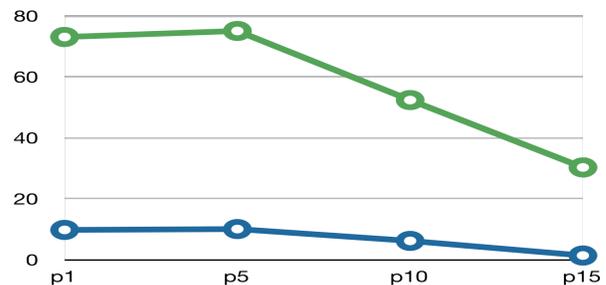


Figure 5. Accidents (green) and Fatal Accidents (blue)/Price of Alcohol

As expected, the number of accidents and related lethal injuries decreases with the increase of alcohol price.

V. CONCLUSION AND FURTHER WORKS

From reviewing different factors involved in alcohol use, we have underscored the necessity of a multidisciplinary perspective to understand the complexity of this phenomenon. This complexity leads us to consider an ontologic ABM as a suitable method to mimic alcohol consumption and alcohol-related social problems. This ontologic model is implemented and simulated with Netlogo in order to run multiple simulations and so achieve public policies testing.

At this stage, most of the algorithms are based on empirical heuristics calibrated against existing quantitative and qualitative data. While the simulation of behavioral patterns linked to alcohol consumption and driven by the neurobiological status of an agent is well advanced, the research team is still seeking complementary information to represent the consequences of these behavioral patterns. Both quantitative and qualitative complementary data are needed.

We plan to realize in-depth interviews with different categories of alcohol users in order to obtain a better understanding of alcohol users behaviors (how their habits change, what are the different reasons for such changes, how users evolve through life...). As proposed by Moore and colleagues, SimARC aims to integrate ethnographical and epidemiological information in an iterative way [48]. Later on we intend to integrate real urban information (GIS) in order to display an accurate geographical context and to give a more accurate representation of public policies implications and results.

REFERENCES

- [1] D.J. Nutt, L.A. King & L.D. Phillips, Drug harms in the UK: a multicriteria decision analysis. *The Lancet*. vol. 376 (9752). 2010.
- [2] WHO, «Global Status Report on Alcohol and Health». 2010.
- [3] D.J Collins & H.M Lapsley, The costs of tobacco, alcohol and illicit drug abuse to Australian society in 2004/2005. 2008.
- [4] Drugs and Crime Prevention Committee, Report on Inquiry into Strategies to Reduce Harmful Alcohol Consumption. Melbourne: Parliament of Victoria. 2006.
- [5] OFDT, Tendances (76), Les niveaux d'usage de drogues en France en 2010. 2011.
- [6] H. Parker, F. Measham & J. Aldridge, *Illegal Leisure: the Normalisation of Adolescent Recreational Drug Use*. Routledge. 1998
- [7] J. Grace, D. Moore & J. Northcote, Alcohol, Risk and Harm Reduction: Drinking Amongst Young Adults in Recreational Settings in Perth, NDRI. 2009.
- [8] WHO, European Alcohol Action Plan 2012-2020. 2011
- [9] Australia Commonwealth, National Alcohol Strategy 2006-2009. Ministerial Council on Drug Strategy. 2006.
- [10] R. Nicholas. Understanding and responding to alcohol-related social harms in Australia. Options for Policing. NDLERF. 2008.
- [11] D.M. Gorman et al. Implications of Systems of Dynamic Models and Control Theory for Environmental Approaches to the Prevention of Alcohol-and other Drug-use related Problems. *Substance Use & Misuse*. vol. 39 (10-12). 2004.
- [12] J. Unger et al. What are the implications of structural/cultural theory for drug abuse prevention? *Sub. Use & Misuse*. vol. 39 (10-12). 2004.
- [13] P. Gruenewald. Why do alcohol outlets matter anyway? A look into the future. *Addiction*. vol. 103. pp. 1585-1587. 2008.
- [14] www.infrastructure.gov.au
- [15] R.M. Julien, C.D. Advokat & J.E. Comaty. *A Primer in Drug Action: a comprehensive guide to the actions, uses, and side effects of psychoactive drugs*. Worth Publishers. 2008.
- [16] H.J. Hanchar, P.D. Dodson, R.W. Olsen, T.S Otis & M.Wallner. Alcohol-induced motor impairment caused by increased extrasynaptic GABA A receptor activity. *Nature Neurosciences*, vol. 8 (3). 2005.
- [17] M.K. Ticku & A.K. Mehta. Effects of alcohol on GABA-mediated Neurotransmission. *Handbook of Experimental Pharmacology*. vol. 114 (6). pp. 103-119. 1995.
- [18] W. McEntee & T. Crook. Glutamate: its role in learning, memory, and the aging brain. *Psychopharmacology* vol. 111 (4). 1993.
- [19] D.M. Lovinger. The Role of Serotonin in Alcohol's Effects on the Brain. *Current Separations*. vol. 18 (1). 1999.
- [20] K. Yoshimoto et al. Alcohol stimulates the Release of Dopamine and Serotonin in the Nucleus Accumbens. *Alcohol*. vol. 9 (1). 1992.
- [21] P.L. Hoffman. Glutamate receptors in Alcohol Withdrawal-Induced Neurotoxicity. *Metabolic Brain Disease*. vol. 10 (1). pp. 73-79. 1995.
- [22] D.G. Kilpatrick, et al. Risk Factors for Adolescent Substance Abuse and Dependence Data from a National Sample. *Journal of Consulting and Clinical Psychology*, vol. 68 (1). 2000.
- [23] B.R. Flay et al. Differential Influence of Parental Smoking and Friends' Smoking on Adolescent Initiation and Escalation and Smoking. *Journal of Health and Social Behavior*, vol. 35(3). 1994.
- [24] M. Pearson & L. Michell. Smoke Rings: social network analysis of friendship groups, smoking and drug-taking. *Drugs: education, prevention and policy*, vol. 7 (1). 2000.
- [25] K.E. Bauman & S.T. Ennet. On the importance of peer influence for adolescent drug use: commonly neglected considerations, *Addiction*, vol. 91 (2). 1996.
- [26] S. Sussman et al. Adolescent peer group identification and characteristics: A review of the literature. *Addictive Behaviors*. vol. 32. pp. 1602-1627. 2007.
- [27] T. Decorte. Drug users' perceptions of 'controlled' and 'uncontrolled' use. *International Journal of Drug Policy*, vol.12, pp.297-320. 2001.
- [28] L. Zhu, D.M. Gorman & S. Horel. Alcohol Outlet Density and Violence: A Geospatial Analysis. *Alcohol & Alcoholism*. vol. 39 (4). pp. 396-375. 2004.
- [29] M. Livingston. A Longitudinal Analysis of alcohol Outlets Density and Assault. *Alcoholism: Clinical and Experimental Research*. vol. 32 (6). 2008.
- [30] K.P. Theall et al. Social Capital and the Neighborhood Alcohol Environment. *Health & Place*. vol. 15. pp. 323-332. 2009.
- [31] K. Kypril et al. Effects of Restricting Pub closing Times on Night-Time Assault in an Australian City. *Addiction*. vol. 106 (2). pp. 303-310. 2011.
- [32] K. Graham et al. Bad Nights or Bad Bars? Multi-level analysis of Environmental Predictors of Aggression on Late-Night Large-Capacity Bars and Clubs. *Addiction*. vol. 101. pp. 1569-1580. 2006.
- [33] F.J. Chaloupka et al. The Effects of Price on Alcohol Consumption and Alcohol-related Problems. National Institute on Alcohol Abuse and Alcoholism. 2002.
- [34] J. Epstein. *Generative Social Science: Studies in Agent-Based Computation*. Princeton University Press. 2007.
- [35] R.K.Sawyer. *Social Emergence: Societies As Complex Systems*. Cambridge University Press. 2005.
- [36] P. Perez & D. Batten. *Complex Science for a Complex World: Exploring Human Ecosystems with Agents*. ANU Press. 2006.
- [37] L. Liu & J. Eck. *Artificial Crime Analysis Systems: Using Computer Simulations and Geographic Information Systems*. Information Science Reference. 2008.
- [38] P.j Brattingham et al. A Statistical Model of Criminal Behavior. *Math. Models and Methods in Applied Sciences* vol. 18. 2008.
- [39] N.H. Agar et al. *Epidemiology or Marketing? The Paradigm Busting Use of Complexity and Ethnography*. Proceedings of Agent. 2004.
- [40] P. Perez et al.. *SimDrug: Exploring the Complexity of Heroin Use in Melbourne*. DPMP. Monograph 11. 2005.
- [41] L.A. Garrison & D.S. Babcock. Alcohol Consumption among College Students: An Agent-based Computational Simulation. *Complexity*. vol. 14 (6). 2009.
- [42] D.M. Gorman et al. ABM of drinking Behavior: A Preliminary Model and Potential Applications to Theory and Practice. *American Journal of Public Health*. vol. 96 (11). pp. 2055-2060. 2007.
- [43] J.E. Rowe & R. Gomez. El Botellon: Modelling the Movement of Crowds in a City. *Complex Systems* vol. 14. pp. 363-370. 2003.
- [44] F. Arvidsson and A. Flycht-Eriksson, *Ontologies I*, Retrieved 26, 2008.
- [45] J. Ferber, *Multi-agent Systems: An Introduction to Distributed Artificial Intelligence*. Addison-Wesley. 1990.
- [46] <http://ccl.northwestern.edu/netlogo/>. U. Wilensky. NetLogo. Center for Connected Learning and Computer-Based Modeling, Northwestern University, Evanston, IL. 1999.
- [47] M. Symmons & N. Hawoth. Safety Attitudes and Behaviours in Work-Related Driving, Stage 1: Analysis of Crash Data. MUARC. Report no. 232. 2005.
- [48] D. Moore et al. Extending drug ethno-epidemiology using agent-based modelling. *Addiction*. vol. 104 (12). pp. 1991-1997. 2009.