

# An Agent-based Model of Alcohol Use and Abuse: SimARC

Francois Lamy

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

Pascal Perez

SMART-IF  
Uni. of Wollongong  
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-component; agent-based model, ontology, alcohol, social simulation, public health**

## Introduction

World Health Organization (WHO), in its «Global Status Report on Alcohol and Health 2011» 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]. Similarly, a recent report from the Independent Scientific Committee on Drugs (ISCD) shows that alcohol, in term of social cost, is more dangerous than heroin and crack [2]. Moreover, Collins and Lapsley have estimated at 15.3 billions AUS\$ (11.6 billions €) the social cost of alcohol [3].

Furthermore, both in Australia and Europe, heavy drinking session, known as "binge drinking" is on the increase leading to greater chances of individual harms (i.e.

falls, pedestrian/car accidents) as well as greater risks of committing crimes (i.e brawl, degradation, violent assaults) [4][5].

Alcohol, through its legality and large availability, is now become a major health problem for governments [6][7] who generally attempt to resolve this social problem by applying different public policies, i.e. alcohol taxation, prevention campaigns and/or reduction in alcohol availability [8][9]. However, net revenues associated with alcohol consumption largely make up for subsequent expenditures in the Australian federal budget [3].

Therefore, alcohol-related social harm constitutes a difficult research topic [10]: alcohol 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.

## Alcohol Use: a Multi-Factorial System

Inside these three levels five components interact together: Alcohol, Individual, Network, Context and Society. We consider the interaction between the alcohol and individual components constitutes the *prima causa* of alcohol-related harms.

### Micro-level: Alcohol/Individual

Alcohol is a potent psychoactive and highly addictive substance. 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].

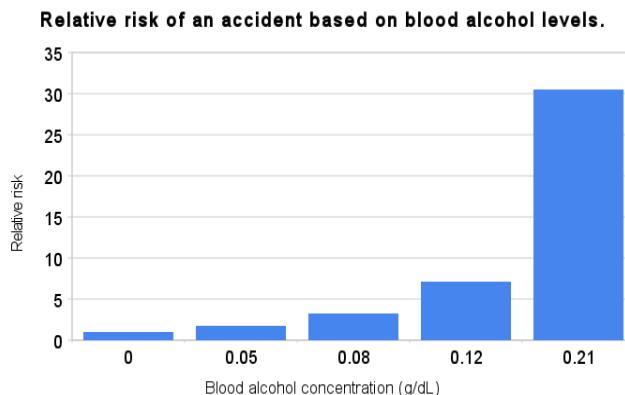


Figure 1. Risk of traffic accident/BAC

To understand alcohol-fuelled violence, BAC is not sufficient. Looking at the neurobiology of alcohol will give us good indications on the way to model these behaviors. Ethanol, the active principle of alcohol, impacts on several neurotransmitters, but Dopamine, Gamma-Amino-Butyric Acid (GABA), Glutamate and Serotonin (5-HT) are strongly related to behavioral changes [15]. As most of the other drugs, alcohol induces release of Dopamine, the neurotransmitter of reward and pleasure: dopamine increases self-confidence, and happiness. However, it is also considered as one of the key-factors that trigger craving, addiction as well as schizophrenia [16].

Ethanol also acts on GABA, the main inhibitory neurotransmitter in the brain. In normal concentration, GABA has a relaxant effect, but higher doses cause drowsiness and motor impairments [17]. GABA has also the function of balancing the excitatory action of Glutamate. This neurotransmitter, at standard dose is implied in learning and memorization [18]; inversely, a high concentration of Glutamate in the brain can generate "excitotoxicity", impairing or killing neurons.

Finally, alcohol interacts strongly with Serotonin. Also known as 5-HydroxyTryptamine (5-HT), this neurotransmitter is responsible for mood regulation, sleeping cycles and thermoregulation. Mild dose leads to euphoria and a general sentiment of happiness. Conversely, depleted serotonin level is correlated with 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 disinhibition. 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 involved in a car or pedestrian accident.

Once the organism slowly gets rid of the alcohol, "stocks" of neurotransmitters have been largely depleted. The "calm down" period starts and users sense the different side effects following their consumption. Individuals, due to dopamine and serotonin depletions, will start to feel depressed, get moody and/or exhibit violent behaviors. Moreover, neuronal connections might be damaged, consequence of high glutamate concentration (excitotoxicity) [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.

### 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 comportments: 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.

### **Macro-level: Social Environment/all components**

According to 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.

### **Alcohol-related Social Simulation**

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 this 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 the computer sciences definition of ontology: “*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).

### **Overall Architecture of SimARC**

Like any Multi-Agent System (MAS), SimARC is constituted by the following elements [45]:

- 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.

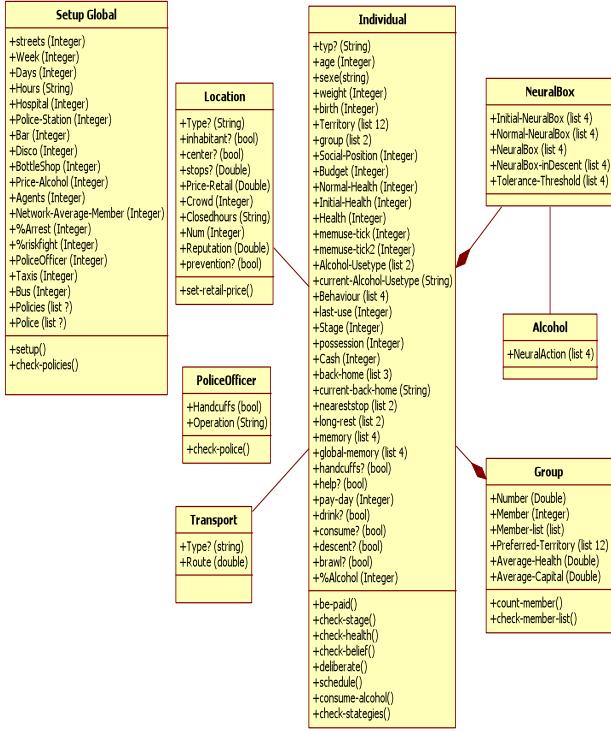


Figure 2. SimARC Class Diagram

Figure 2 presents the UML-based Class diagram of SimARC's structure which has been subsequently implemented in NetLogo v4.1.3 [46]. SimARC's 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.

SimARC users can select different scenarios: Alcohol Price, Police Operations and Public Policies. Using sliders, users can also select the probability for a constable to arrest a 'problematic' user and the probability of occurrence of alcohol-fuelled brawls.

In the following sections, we are describing the different components of SimARC and their dynamics. The last section provides some preliminary results.

## 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.

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.).

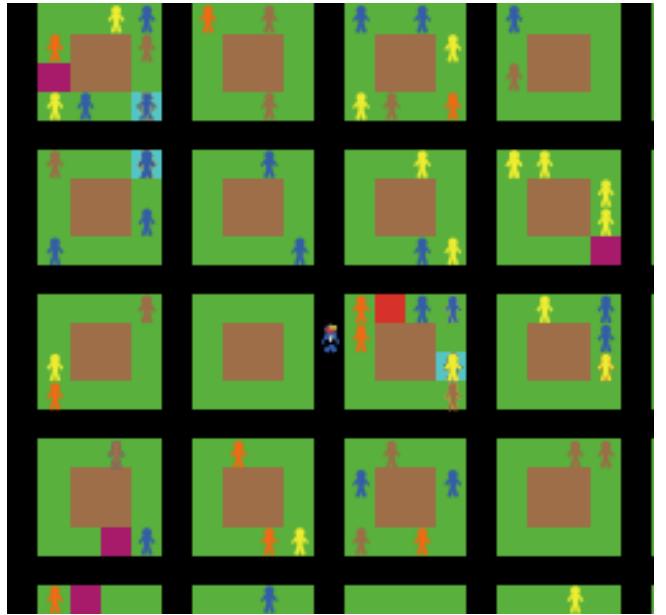


Figure 3. SimARC Urban Grid

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.

## 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 follow a "daily-routine": 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*). Agent salary equals ten times their Income (normal-distribution) every fortnight (randomly predefined). This salary constitutes "pocket money" for non-essential expenses, and is exclusively spent on alcohol. The average amount of this pocket money is equal to 200 (in a range of 50 to 360).

Alcohol consumption varies according to each agent's *Alcohol-routine* and *Stage*. If some agents will just have a few drinks in their favorite pub on a Saturday night, some others can have several binge-drinking sessions at their dwelling all along the week. Others agents are just staying home and sober the whole week, resulting in no individual harm or social trouble. Agents drinking large quantities of alcohol can exhibit violent or hazardous behaviors (brawl, accident and having been sick are counted and memorized) and these "binge-drinking" sessions decrease their *Health* attribute.

One unit of alcohol represents one Standard Drink (10 g of alcohol) and each "Drink" increases male agent BAC by  $10/(\text{Weight} \times 0.7)$  and by  $10/(\text{Weight} \times 0.6)$  for female agent. BAC is decreased by 0.15 every step (2 hours time). Each SD modifies the levels of neurotransmitters and the relative balance of neurotransmitters changes individual patterns. Based on their cumulative experience of negative consequences (personal or witnessed) during or after successive night-outs, agents may change their mind and representation about alcohol. In turn, these updated beliefs might modify an agent's *Alcohol-routine* and *Stage*.

Moreover, agents can interact through physical colocation in the spatial environment or through messages amongst friendship networks. Thus, agents of a same group have night-out all together in network *Favorite Venues*. Those *Friends* of the group can also "enjoin" an agent with a low *Health* or frequent dangerous behaviors to "slow down": if this agent consents, it will not go out nor drink for one to two weeks, restoring its *Health* from its previous consumptions; otherwise, it will leave from its primary network to find new drinking mates.

## Preliminary Results

This section will present some preliminary results from SimARC. This simulation hasn't been entirely calibrated yet; therefore, these experiments *in-silico* intend to test the internal consistency of the model. To do so, we examine the consequences of alcohol taxation policy. Six different price

scaling factors have been tested: x1, x5, x8, x10, x12 and x15 (relative to the average \$200 available to agents).

In this experiment, the virtual population is composed of 750 agents and 2 constables. The latter have 1% chance to arrest users with a BAC  $> 0.05$  g/dL. In order to initiate the simulation, we arbitrarily consider the following distribution: 70% of the agents start at Stage 1, 15% at Stage 2, 10% at Stage 3 and 5% at Stage 4.

We have run 50 replicates of each simulation running over 4367 time steps (equivalent to one year in total). Quantities of alcohol consumed for each scenario has been measured as well as the evolution of the population distribution across the different *Stages*.

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

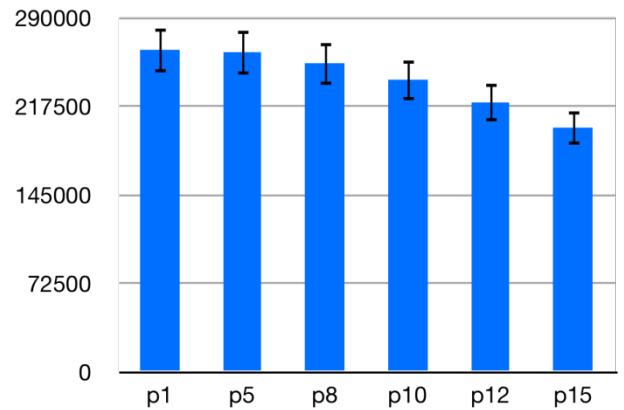


Figure 4. Alcohol Consumption (SD) / Price of Alcohol

As shown in figure 4, alcohol consumption level decreases with an augmentation of the taxation rate: a 3.4% decrease between P5 and P8, 8.6% between P5 and P10, 15.7% between P5 and P12, and, finally, 23.6% between P5 and P15. These results are consistent with other studies [33]. The lack of response between P1 and P5 can be attributed partly to the "social control" operated by peers and partly to the evolution of individual opinions in response to bad experiences during heavy drinking sessions (see above).

An essential mechanism involved in intoxication is the neurological *tolerance* of each individual to alcohol and its evolution. Tolerance reflects the adaptation of both brain and body to any drug. A major consequence of tolerance mechanisms is that individuals need to increase the dosage and/or frequency of intake to achieve the effects as before.

SimARC neurologic 'motor' works as follow: each drink increases neurotransmitters concentration, if any concentration reaches the "*Tolerance-Threshold*" of its related neurotransmitters, agent will exhibit specific behaviours (i.e. if the Tolerance-Threshold of Dopamine is

reached, an agent's *Behavior* will shift to "Happy"). The *Tolerance-Threshold* increases after each drinking session (and decreases while agents are resting). Therefore, agents need to consume more alcohol to obtain positive effects. Once the frequency of SD by week hits a pre-defined level, agents change their *Stage* as well as their *Alcohol Routine*. We have implemented 7 Stages to simulate the evolution of an agent's *tolerance* to alcohol through time from *Stage 1*, assuming a consumption of 2 or 3 SD/week, to *Stage 7*, assuming a severe addiction equivalent to 25 SD/day.

Our experiment gives the following results (cf. figure 5):



Figure 5. Population Repartition (Stages) / Price of Alcohol

The number of agents at higher Stages (5, 6 and 7) decreases with an augmentation of prices. Meanwhile, the number of agents at Stage 2, 3 and 4 increases. Our results seem to indicate that alcohol taxation has a positive impact by limiting heavy use and so, harmful individual consumption.

## Conclusion and Further Works

Through our short review of different factors involved in alcohol use, we have underscored the necessity of a multidisciplinary perspective to understand the complexity of alcohol abuse related consequences. This complexity leads us to consider an ontological approach to capture alcohol consumption and alcohol-related social problems. The resulting model is implemented and simulated with NetLogo in order to run multiple simulations and to test hypothetical public policies.

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 conduct 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]. Next step will consist in integrating real urban information (GIS) in order to display an accurate geographical context and to provide a more realistic 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 Realease 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. Kypri 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 Bratingham 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. Haworth. 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