PATTERN OF ALCOHOL USE AMONG PATIENTS WITH TRAUMATIC BRAIN INJURY IN MALAYSIA'S URBAN HOSPITAL

DR.LIM SZE HUEI (MGC140018)

DISSERTATION SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF PSYCHOLOGICALMEDICINE

DEPARTMENT OF PSYCHOLOGICAL MEDICINE UNIVERSITY OF MALAYA KUALA LUMPUR

2018

PATTERN OF ALCOHOL USE AMONG PATIENTS WITH TRAUMATIC BRAIN INJURY IN MALAYSIA'S URBAN HOSPITAL

Dr. LIM SZE HUEI (MGC NO.140018) MASTER OF PSYCHOLOGICAL MEDICINE (MPM) 2017/2018

SUPERVISOR: ASSOCIATE PROFESSOR DR. AMER SIDDIQ BIN AMER NORDIN ASSOCIATE PROFESSOR DR. SIA SHEAU FUNG

> CO-SUPERVISOR: DR. ABDUL RAZAK BIN OTHMAN

DEPARTMENT OF PSYCHOLOGICAL MEDICINE FACULTY OF MEDICINE UNIVERSITY OF MALAYA, KUALA LUMPUR

UNIVERSITY OF MALAYA ORIGINAL LITERARY WORK DECLARATION

Name of candidate: LIM SZE HUEIRegistration/Matric No.: MGC 140018Name of degree: MASTER OF PSYCHOLOGICAL MEDICINETitle of Project Paper/Research Report/Dissertation/Thesis ("this Work"):

PATTERN OF ALCOHOL USE AMONG PATIENTS WITH TRAUMATIC BRAIN INJURY IN MALAYSIA'S URBAN HOSPITAL

Field of Stud: PSYCHIATRY

I do solemnly and sincerely declare that:

- 1) I am the sole author/writer of the Work;
- 2) This Work is original;
- 3) Any use of work in which copyright exists was done by way of fair dealing and for permitted purposes and any excerpt or extract from, or reference to or reproduction of any copyrighted work has been disclosed expressly and sufficiently and the title of the Work and its authorship have been acknowledged in this Work;
- 4) I do not have any actual knowledge nor do I ought reasonably to know that the making of this work constitutes an infringement of any copyrighted work;
- 5) I hereby assign all and every right in the copyright to this Work to the University of Malaya (UM), who henceforth shall be owner of the copyright in this Work and that any reproduction or use in any form or by any means whatsoever is prohibited without the written consent of UM having been first had and obtained;
- 6) I am fully aware that if in the course of making this Work I have infringed any copyright whether intentionally or otherwise, I may be subject to legal action or any other action as may be determined by UM.

Candidate's Signature

Date

Date

Subscribed and solemnly declared before,

Witness's Signature

Name:

Designation:

3

CERTIFICATION

This is to certify that the candidate, DR. LIM SZE HUEI, had carried out this dissertation project. To the best of my knowledge, this dissertation is entirely his own work.

ASSOCIATE PROF. DR. AMER SIDDIQ BIN AMER NORDIN MBChB, MPM (MALAYA) Department of Psychological Medicine, Faculty of Medicine, 50603, University of Malaya ASSOCIATE PROF. DR. SIA SHEAU FUNG MD, PhD MRCS, AFRCS, MS (MALAYA) Division of Neurosurgery, Department of Surgery Faculty of Medicine, 50603, University of Malaya

ABSTRACT

Pattern of alcohol use among patients with traumatic brain injury in Malaysia's urban hospital

Objective:

To date, there is limited literature on the alcohol use among the patients with Traumatic Brain Injury (TBI) in Malaysia. Hence, the primary objective of the study is to determine the pattern of alcohol use among patients with traumatic brain injury in an urban hospital in Malaysia. The secondary objective is to investigate the association of alcohol use in the traumatic brain injured subjects with socio-demographic characteristics, duration of head injury and severity of head injury, history of alcohol use/substance use, psychological wellbeing, and cognitive function.

Method:

This is a cross sectional study conducted in the UMMC Rehabilitation Clinic, Neurosurgical clinic and ward, HKL Neurosurgical clinic and ward, the observation ward, Emergency Department. Data collection occurred from November 2017 till January 2018. A convenient sampling method was used. The patients were selected based on the selection criteria. The socio-demographic and clinical information were gathered after obtaining the informed consent from the patients or their guardians. The participants were each given a booklet of questionnaires to be filled which include their demographic particulars, alcohol use pre-or post TBI particulars, head injury particulars, AUDIT form, GHQ-12 form and MOCA form. All the questions were mainly in the Malay language and the researcher assisted the patients if the subjects had difficulties to understand the questions. Results:

Out of the 60 patients approached, only 47 patients were included in the study. The mean age of the participants was 36.8 years- (SD \pm 14.8). In 83% of the patients were male (n=39) and females constituted 17% (n=8). The majority of the patients were Malays (63.8%) followed by Indian (23.4%) and Chinese (10.6%). In 51.1% of the participants were married and 48.9% were single or divorced. About two third of patients attained at least secondary education. Almost half of the patients (44.7%) were unemployed. Their average salary was RM 1429.79 (SD \pm 2340.28)

A quarter of the subjects had used alcohol prior to their injury. There were significant associations between post-TBI alcohol use, an AUDIT score of 8 or more (OR 30, 95%CI 3.06-294.56) and alcohol use history (OR 1.31, 95%CI 1.03-1.67). A significant relationship was also noted between pre-TBI alcohol use and safety measure taken (OR 9.6, 95% CI 1.27-72.53). However, no significant associations were found between pre-TBI alcohol use with Modified Rankin Scale (mRS), neurosurgical intervention and GCS severity. Similarly, there was no significant associations were found between post-TBI alcohol use with age group, gender, marital status, educational status, occupation status, head injury duration, family history of alcohol use, pre TBI nicotine use, GHQ-12 score and MoCA score.

Conclusion:

The association with alcohol use and occurrence TBI is a complicated yet significant situation. Alcohol use affect incidence and subsequent recuperation from a TBI episode. The findings from the study, support the associations found in numerous literature; that the patients' alcohol use pre-injury may contribute to their recovery and post-TBI usage. The findings of the study add evidence to the need to monitor alcohol level in all patients presenting with traumatic head injury.

Keywords: Alcohol use, prevalence, TBI, head injury, Malaysia

ABSTRAK

Kelaziman penggunaan alkohol di kalangan pesakit yang mengalami kecederaan kepala di hospital bandar di Malaysia

Objektif:

Sehingga hari ini, tidak banyak artikel berkenaan penggunaan alkohol di kalangan pesakit yang mengalami kecederaan kepala di Malaysia. Oleh yang demikian, objektif utama kajian adalah untuk mengkaji corak penggunaan alkohol di kalangan pesakit yang mengalami kecederaan kepala di hospital bandar di Malaysia. Objektif sekunder adalah untuk menyelidik hubungan antara penggunaan alkohol selepas kecederaan kepala dengan data peribadi pesakit, jangka masa kecederaan kepala, tahap kecederaan kepala, sejarah penggunaan alkohol atau barang terlarang, tahap kesejahteraan mental pesakit dan fungsi kognitif.

Kaedah:

Kajian ini merupakan kajian keratan rentas yang dijalankan di Klinik Rehabilitasi PPUM, Klinik dan Wad Neurosurgikal PPUM, Klinik dan Wad Neurosurgikal HKL, Wad pemerhatiaan kecemasan HKL dari November 2017 hingga Januari 2018. Kaedah pengambilan sample mengikut keselesaan penyelidik dibuat bergantung kepada kriteria pemilihan. Soalan berkenaan data peribadi and data klinikal dikumpulkan setelah keizinan daripada pesakit atau penjaga diperoleh. Pesakit akan diminta untuk mengisikan borang penyelidikan yang disediakan termasuk data peribadi, soalan berkenaan penggunaan alkohol sebelum dan selepas kecederaan kepala, soalan berkenaan kecederaan kepala, soalan AUDIT, soalan GHQ-12 dan soalan MOCA. Semua soalan yang diberikan adalah dalam Bahasa Melayu dan penyelidik akan membantu pesakit sekiranya terdapat soalan yang sukar difahami.

Keputusan:

Dari 60 pesakit yang ditemuramah, hanya 47 pesakit sahaja yang diambil untuk kajian ini. Umur purata pesakit adalah 36.8 tahun (SD±14.8). 83% daripada pesakit adalah lelaki (n=39) dan 17% adalah perempuan (n=8). Majoriti pesakit adalah dari bangsa Melayu (63.8%), diikuti dengan bangsa India (23.4%) dan Cina (10.6%). 51.1% daripada pesakit telah berkahwin dan 48.9% adalah bujang atau bercerai. Lebih kurang dua pertiga daripada pesakit mendapat sekurang-kurangnya pendidikan tahap menengah. 44.7% daripada pesakit tidak bekerja. Pendapatan bulanan secara purata adalah RM 1429.79 (SD±2340.28)

Terdapat hubungan yang ketara diperoleh melalui kajian ini dari segi penggunaan alkohol selepas kecederaan kepala dan skor AUDIT lebih dari 8 (OR 30, 95% CI 3.06-294.56). Hubungan ketara juga diperoleh dengan penggunaan alkohol selepas kecederaan kepala dan sejarah penggunaan alkohol (OR 1.31, 95% CI 1.03-1.67). Selain itu, hubungan ketara juga diperoleh daripada penggunaan alkohol sebelum kemalangan dan penggunaan alatan keselamatan (tali pinggang keselamatan, topi keledar)(OR 9.6, 95% CI 1.27-72.53). Walaupun begitu, kajian ini tidak menemui sebarang hubungan ketara berkenaan penggunaan alkohol sebelum kecederaan kepala dan Modified Rankin Scale (mRS), intervensi neurosurgical dan tahap kecederaan kepala (GCS Score). Selain itu, tiada hubungan ketara yang diperoleh berkenaan penggunaan alkohol selepas kecederaan kepala dengan umur pesakit, jantina pesakit, taraf perkahwinan, taraf pembelajaran, taraf pekerjaan, jangka masa kecederaan kepala, sejarah keluarga berkenaan penggunaan alkohol, penggunaan tembakau sebelum kecederaan kepala, skor GHQ-12 dan skor MOCA.

Kesimpulan:

Penggunaan alkohol dan kecederaan kepala adalah masalah yang rumit dimana keduadua faktor ini akan memberi kesan antara satu sama lain biarpun sebelum kecederaan kepala atau selepas kecederaan kepala. Selain itu, masalah ini juga merumitkan penjagaan pesakit selepas itu. Daripada kajian ini, kami dapat menghubungkan beberapa factor yang menyumbang kepada penggunaan alkohol selepas kecederaan kepala iaitu sejarah penggunaan alkohol dan penggunaan alkohol pada tahap bahaya sebelum kecederaan kepala. Kedua-dua faktor ini boleh digunakan oleh doktor yang merawat sebagai indikasi untuk masalah penggunaan alkohol di waktu kelak.

Kata kunci: Penggunaan alcohol, kelaziman, kecederaan kepala, Malaysia

ACKNOWLEDGEMENT

First of all, I would like to express my utmost gratitude to my supervisor, Associate Professor Dr. Amer Siddiq bin Amer Nordin for his brilliant idea in this topic and subsequently advise and support throughout my preparation for this dissertation. Thank you for spending your time to discuss and guide me when I was facing difficulties halfway in sample collection. Your guidance gave me a clear direction on the things that I needed to be done.

Besides this, I would also need to thank my other supervisor, Associate Professor Dr. Sia Sheau Fung. Thank you for spending your time and making effort to help when I am stuck with the write up. He is tireless in giving constructive comment and supporting me along the way for the completion of the thesis.

Other than them, would appreciate the effort given by my co-supervisor Dr. Abdul Razak bin Othman (Psychiatry Department HKL). He has been constantly reminding me about the dateline and supporting me as I did my thesis.

Apart from my supervisors and co-supervisors, I would also like to thank everyone who had helped me towards my completion of the thesis. Thank you to Associate Professor Mazlina binti Mazlan from Neuro-rehabilitation UM for allowing me to gather my sample at the neurorehabilitation clinic. Thank you for my head of department in UMMC and HKL who are supportive for giving adequate time for me to complete my thesis.

Special thanks to Associate Professor. Dr.Aili Hanim binti Hashim to always be there to help out even though she was busy with her other work. Her dedication towards her students were much respected and appreciated. Her critics help to guide me through the completion of the thesis. Thank you for the hardship during the time of need. Additional thanks to Associate Professor. Dr. Jesjeet Singh Gill A/L Jeswant Singh for believing in me and is supportive for my work towards the completion of the thesis.

Besides this, I would also like to thank all my friends and colleagues who had helped me with their advice and beneficial suggestion for my thesis. Their willingness to extend a helping hand and their understanding in covering me to allow me time to write the thesis.

Finally, I would like to thank my family especially my parents, wife and son for their understanding and support throughout the completion of my thesis.

11

TABLE OF CONTENTS

CONTENTS	Page
Certification	4
Abstract	5-9
Acknowledgement	10-11
Table of Contents	12
List of Appendices	13
Abbreviations	14
Chapter 1: Introduction and Literature Review	15-36
Chapter 2: Rationale, Objectives of study	37
Chapter 3: Methodology	38-42
Chapter 4: Result	43-70
Chapter 5: Discussion, Limitation and Conclusion	71-83
References	84-93
Appendices	94-105

LIST OF APPENDICES

Appendix	Title	Pages
А	Ethics Approval	94-96
В	Socio-demographic and Clinical Profile Questionnaires	97-100
С	AUDIT questionnaire (Malay version)	101
D	GHQ-12 questionnaire (English and Malay version)	102-103
E	MoCA questionnaire (English and Malay version)	104-105

ABBREVIATIONS

- ATS Amphetamine Type Stimulant
- AUD Alcohol Use Disorder
- AUDIT Alcohol Use Disorder Identification Test
- BAC Blood Alcohol Concentration
- BAL Blood Alcohol Level
- CI Confidence Interval
- CT Computed Tomography
- DALY Disability Adjusted Life Years
- GCS Glasgow Coma Scale
- GHQ-12 General Health Questionnaire 12
- HKL Hospital Kuala Lumpur
- MOCA Montreal Cognitive Assessment
- MTBI Mild Traumatic Brain Injury
- NMRR National Medical Research Registry
- OR Odds Ratio
- PPUM Pusat Perubatan Universiti Malaya
- SD Standard Deviation
- TBI Traumatic Brain Injury
- UMMC University Malaya Medical Centre
- WHO World Health Organization

1) Introduction.

The frequent and chronic use of alcohol has emerged as an important health and social problem worldwide (Rehm et al., 2007; Rehm et al., 2009; World Health Organisation, 2014; American Psychiatric Association, 2013). In many countries, alcohol has emerged as the foremost risk factor for various non-communicable diseases (Parry et al., 2011; Shield et al., 2012; Shield et al., 2014).

Numerous studies have shown alcohol ingestion to be a leading cause of injury (Rehm et al., 2003; Rehm et al., 2009; Parry et al., 2011; World Health Organisation, 2014), including head injury. It is without questionable the brain is the most vulnerable human organ affected by the chronic and large amounts of alcohol consumption (Oscar-Berman, 2000; Oscar-Berman and Marinkovic, 2003; Planas-Ballvé et al., 2017).

Several studies suggest a high prevalence of substance use problems in people presenting to the hospital and treated for a TBI, particularly alcohol. There are countless studies indicating traumatic injury from accidents were more seen in alcohol-impaired drivers (Bernier and Hillary, 2016; Bird et al., 2009; Iverson, 2006). The studies indicate in patients with alcohol-positive traumatic injury have more serious presentation and are likely to stay longer (Iverson, 2005; Green et al., 2015).

1.1) Alcohol and burden on health

Alcohol is an addictive substance (World Health Organisation, 2014; American Psychiatric Association, 2013). Regular use of alcohol has emerged as a key health and social problem globally (Rehm et al., 2007; Rehm et al., 2009; American Psychiatric Association, 2013; World Health Organisation, 2014).

Excessive and unregulated alcohol usage is tantamount to the terms burden and

harmful use (Rehm et al., 2007). Globally, its use causes approximately 3.3 million deaths every year with 5.1% of global burden of diseases attributable to its use (World Health Organisation, 2014; Mokdad et al., 2004). In 2000, Mokdad et al. (2004) identified alcohol was the second highest cause of death in the United States after tobacco.

Thus, it is not surprising alcohol has emerged and identified as a leading risk factor for death and disability globally (Fenoglio et al., 1997; Parry et al., 2011; World Health Organisation, 2014). Alcohol-related deaths currently make up approximately 4% of all global mortality (World Health Organization, 2004). Rehm et al. (2009) stated even in middle-income and high-income countries alcohol related factors are a leading cause of death. Rehm et al. (2009) and the World Health Organisation (2014) related alcohol for 3.8% of death and 4.6% of disability adjusted life years (DALYs) lost in 2004. Additionally, alcohol consumption is recognized as a vital risk factor for chronic disease and injury (Rehm et al., 2009; Rehm et al., 2003; Parry et al., 2011; World Health Organisation, 2014).

The American Psychiatric Association (2013) characterized excessive or harmful alcohol consumption as the intake of 40-60g/day of alcohol in females and 60-100 g/day in males. Alcohol consumption constitutes intoxication, binge drinking, abuse and dependency (American Psychiatric Association, 2013). The impact of excessive alcohol consumption on the society is well-documented (McIntosh and Chick, 2004; Kim et al., 2006). Excessive alcohol consumption adds burden to the society including psychosocial problems such as increase in utilization of healthcare by admission, alcohol related disease and injuries (Kim et al., 2006; McIntosh and Chick, 2004; World Health Organisation, 2014; Planas-Ballvé et al., 2017). The burden associated with alcohol consumption stems from heavy and regular drinking (Rehm, 2011; Rehm et al., 2012).

The constant and significant amount of alcohol consumption affects multiple neurotransmitter systems in the brain (Oscar-Berman and Marinkovic, 2003), among which is the brain dopamine functioning (Volkow et al., 2003). Several ways alcohol affects the brain are via the dopaminergic neurotransmission (Loheswaran et al., 2016), which is altered by the acute alcohol consumption and dependence. The individual's brain dopamine function is markedly decreased after chronic abuse and during the withdrawal state, and the decrease is related to the dysfunction of the prefrontal regions (Planas-Ballvé et al., 2017). The alteration in the dopaminergic neurotransmission is a vital mediator to the effect of alcohol on the neuroplasticity of the brain (Loheswaran et al., 2016). Neuroplasticity is the change in neural structure and function in response to experience or environmental stimuli (Blugeot et al., 2011; Kays et al., 2012).

Oscar-Berman (2000) and Oscar-Berman and Marinkovic (2003) state a person's susceptibility to alcoholism–related damage is related to the person's

- age,
- gender,
- drinking history,
- blood alcohol level,
- nutrition state, and
- the vulnerability of the specific brain regions.

Rehm et al. (1996) added that an individual's drinking patterns as well play an important role.

Scientists remarked the relationship between alcohol consumption and health and social outcomes is complex and multidimensional (Rehm et al., 1996; Yue et al., 2017). Babor and Grant (1992) and Rehm et al. (2006) proposed the acute and long-term health and social consequences linked to alcohol is via three intermediate mechanisms:

- 1. toxic and beneficial biochemical effects,
- 2. intoxication, and
- 3. dependence.

The brain is particularly susceptible to injury from the constant and sizeable amount of alcohol consumption (Oscar-Berman, 2000; Planas-Ballvé et al., 2017). White (2003), reported among the cognitive functions affected are difficulty walking, blurred vision, slurred speech, slowed reaction times, and impaired memory.

The most important disease conditions in alcohol use usage is the alcohol use disorders (AUDs), which include alcohol dependence and harmful use or alcohol abuse (American Psychiatric Association, 2013; World Health Organization, 1992). Though the AUDs are less fatal than the other chronic disease conditions related to alcohol usage, these conditions are linked to considerable disability (White, 2003; Samokhvalov et al., 2010; World Health Organization, 1992; Rehm, 2011; American Psychiatric Association, 2013). Especially among males, the alcohol-use disorders is among the most disabling disease recognized for its global burden of disease (World Health Organization, 2004; World Health Organisation, 2002).

The existence of the disability associated with alcohol usage constitute a large part of this burden related to the AUDs (Samokhvalov et al., 2010). The health complication of alcohol can be divided into direct or indirect consequences (Rehm et al., 2009). Direct cause involves the toxic component of alcohol itself which may cause impairment in the organs. Alcohol usage is associated with a multitude of toxic effects on the different organs (Rehm et al., 2006; Rehm et al., 2013). In addition, accidental or intentional injuries or deaths follow alcohol intoxication (Rehm et al., 2003; Connor et al., 2005; Raj et al., 2015).

The consequences of alcohol on the central nervous system result in the subjective feeling of intoxication, and these effects are felt and can be measured even at light to moderate consumption levels (Eckardt et al., 1998; Connor et al., 2005). Rehm et al. (2006) stated the acute effects of alcohol and cardiovascular outcomes could occur following the patterns of drinking. Accidental and intentional injuries as examples of

18

acute effects of alcohol while coronary heart disease is consequences of cardiovascular outcomes (Rehm et al., 2003; Connor et al., 2005; Raj et al., 2015).

The use of alcohol can as well cause indirect injury which may involve more of social repercussion of alcohol itself whereby leading to impairment of judgement from intoxication and causing motor vehicle accidents, fights, fall (Watt et al., 2004). There is an increasing number of data implying alcohol-related injuries, trauma and deaths (Watt et al., 2004; World Health Organization, 2007).

World Health Organization (2007) stated every year more than 5 million deaths occur from injuries generating close to one-tenth of the global burden of disease. The link between alcohol and almost all kinds of injuries has long been established (Rehm et al., 2013; Rehm et al., 2012). Some researchers divided the injuries into two categories:

- unintentional injuries, including road traffic injuries, drowning, burns, poisoning and falls; and
- 2. intentional injuries, as a result of deliberate acts of violence against oneself or others.

Injury related to alcohol consumption is a worrying situation (Taylor et al., 2010; Taylor and Rehm, 2012). The correlation of alcohol and virtually all types of unintentional injuries has extensively been recognized. Research found a distinctive correlation between alcohol and almost all kinds of unintentional injuries (Taylor et al., 2010; Taylor and Rehm, 2012; Rehm et al., 2009). The most obvious situation are road traffic accidents. The Department of Transportation (2000) reported thirty-nine percent of all traffic-related deaths were alcohol related while Tien et al. (2006) related up to 50% of patients with trauma and hospitalized were intoxicated at the time of injury.

Alcohol-attributable injury can occur even from a single instance of acute alcohol consumption, leading to intoxication and drunkenness (Taylor et al., 2008; Taylor et al., 2010) and thus the accident. Rehm et al. (2008) discovered the lifetime risk of injury

leading to death is 1 in 100 and happens once the consumption levels of about three drinks daily per week for women, and three drinks five times a week for men. Rehm et al. (2008) added engaging in the acute alcohol consumption repeatedly results in a higher risk of injury. Further Zador et al. (2000) discovered being male, and younger drivers are at relative risk of fatal single-vehicle crash injury. The study concluded the elevated risk to both to drivers and to other road users. Taylor et al. (2010) suggested from the systematic review on alcohol and risk of injury there is no safe level of consumption. Taylor et al. (2010) stated even with two standard drinks, the odds of injury almost doubled for most types of injury.

Alcohol consumption affects a person's psychomotor abilities (Rehm, 2011; Taylor et al., 2010). The adverse effects of alcohol are experienced at the Blood Alcohol Concentration (BAC) level approximately at 0.04 to 0.05 percent, which the person achieves after consuming two to three drinks in an hour (Rehm et al., 2012; Rehm, 2011). Eckardt et al. (1998) proposed at these levels alcohol in the system disrupts a person's psychomotor functions with increase consequences of injury. Taylor and Rehm (2012) cautioned at all levels of BAC, the odds ratio (OR) of fatal motor vehicle injury was significant whereby the 5 combined studies yielded OR of 1.74 (95%CI 1.43-2.14) for fatal injury every 0.02% raised in BAC.

Van Dyke and Fillmore (2014) disclosed drivers with a history of driving under the influence (DUI) of alcohol report heightened impulsivity and display reckless driving. In laboratory studies, Ogden and Moskowitz (2004) and Liguori (2009) of simulated driving performance demonstrate the presence of alcohol impairs the person's ability to maintain a stable position in the lane, reduces braking time and lessens the person's ability to detect potential hazards on the roads.

1.2) Traumatic brain injury (TBI)

Traumatic brain injury is an increasing concern with falls, motor vehicle crashes, struck by or against events, and assaults identified as leading causes (Langlois et al., 2006; Ahmed et al., 2017). Faul et al. (2010) reported in the 1.7 million TBIs occurring each year in the United States, 80.7% presented as emergency department visits. Subsequently 16.3% required hospitalizations, and 3.0% resulted in deaths.

Faul et al. (2010) added among the people who made hospital visits 4.8% of all injuries seen in emergency department visits were diagnosed with TBIs, with 15. % of all hospitalizations. Of all the injury- related deaths in the United States, TBI was a contributing factor 30.5% of the time.

Traumatic brain injury (TBI) is a non-degenerative, non-congenital insult to the brain (Iverson, 2005; Centers for Disease Control and Prevention, 2017). TBI refers to the brain dysfunction caused by external trauma (Ahmed et al., 2017). Traumatic brain injury (TBI) occurs when a blow or jolt to the head or a penetrating injury results in damage to the brain (Ahmed et al., 2017). The brain dysfunction is characterized by damage to the structure and certainly the function of the brain (Iverson, 2005; Centers for Disease Control and Prevention, 2017). The injuries cause the loss of consciousness (Langlois et al., 2006). While, the ongoing ischemia results in further brain injury and contributes to the overall mortality of TBI (Fabbri et al., 2002). Mild traumatic brain injuries are characterized by immediate physiological changes hypothesized as a multilayered neuro-metabolic force in which affected cells typically recover, although under certain circumstances a small number might degenerate and die (Schretlen and Shapiro, 2003; Iverson, 2005). Iverson (2005) describes during the first week after injury the brain undergoes a dynamic restorative process.

The TBIs can affect anyone at any age and may lead to severe complications and disabilities in the future (Langlois et al., 2006; Hyder et al., 2007; Faul et al., 2010). Even

in a developed country as the United States, the TBIs are a major health problem as the condition contribute to substantial number of deaths and cases of permanent disability (Hyder et al., 2007; Faul et al., 2010; Ahmed et al., 2017). In 7–20% of patients with a TBI presents to the emergency room with bleeding, bruising, or swelling on day-of-injury which can only be seen via computed tomography (Livingston et al., 1991; Iverson et al., 2000).

Researchers such as Langlois et al. (2006) described the situation as a "silent epidemic" as the problems experienced by the individuals with TBI, are often not visible. The TBIs may lead to severe complications and disabilities in the future. There are persistent cognitive sequelae in surviving individuals (Yue et al., 2017). Affected individuals are affected by impairment in their memory or cognition, which are often not noticeable by others (Langlois et al., 2006; Hyder et al., 2007).

Of concern is that, scientists are predicting TBI, will surpass many diseases and appear as the major cause of death and disability by the year 2020 others (Langlois et al., 2006; Hyder et al., 2007). Moreover, TBIs are under-diagnosed (Yue et al., 2017). While the TBIs can affect anyone at any age, Ahmed et al. (2017) found young males were more often hospitalized following injuries resulting in TBI.

Researchers such as Ahmed et al. (2017) and Faul et al. (2010) believed TBI is one of the significant public health burdens. Faul et al. (2010) showed that only about 25% of people achieve long-term functional independence following TBI. Ponsford et al. (2000) and (Carroll et al., 2004) showed only some cognitive symptoms resolve within a few months of the injury. Ponsford et al. (2000) investigating cognitive impairment in 84 adults with mild TBI found mainly the headaches, dizziness, fatigue, and visual disturbance improved a few months after the injury. Ponsford et al. (2000) noted significant levels of psychopathology remained in the majority.

1.3) The health burden related to TBIs

Traumatic brain injury is a leading cause of disability in young people, affecting their capacity for work, leisure and relationships (Bombardier et al., 2003; Rabinowitz and Levin, 2014). Vanderploeg et al. (2005) illustrious the cognitive functioning is affected despite in mild cases of TBI. Belanger et al. (2005) in a meta-analysis based on 39 studies comprising 1463 cases of mild TBI and 1191 control cases, illustrated the neuropsychological impairment improved by 3 months post-injury. However, the cognitive impairment remained, beyond three months. Belanger et al. (2005) noted the impairment may worsened over time. Marsh et al. (2016) observed the impairment occurs across various domains of mental functioning. The domains are attention, verbal and visual memory, visual-spatial construction and other executive functions. Patients with TBI most consistently have difficulties with information processing speed and verbal memory (Skandsen et al., 2010; Spitz et al., 2012).

These executive functions are critical for the persons to carry out complex behaviors in every novel situation involving beginning, goal setting, planning, organizing, judgment, and self-monitoring (Mateer and Sira, 2006; Marsh et al., 2016). Vanderploeg et al. (2005) supported the findings as their study found the subtle yet long-term impact of the multifaceted attention and working memory was impaired even in individuals with mild TBI. Spitz et al. (2012) followed-up 111 individuals with moderate-to-severe TBI assessed on average at 3, 6, and 13 months post-injury. The study noted poorer functional outcomes were in the older age-group, lower levels of education, and more significant days of posttraumatic amnesia. Vanderploeg et al. (2005) concluded these impairments can have adverse long-term neuropsychological outcomes and hampers recovery. The presence of cognitive impairment was associated with future disability (Skandsen et al., 2010; Spitz et al., 2012). Marsh et al. (2016) discovered following 71 individuals with TBI the cognitive impairment was evident across all domains. Interestingly, Marsh et al. (2016) noted while the improvement of cognitive functioning could happen, the recovery of full functioning is unlikely.

1.4) Psychological impact of Traumatic Brain Injury

Thus, it is not surprising psychiatric symptomatology transpires with the affected mental functioning (Hoofien et al., 2001). The TBIs affect the individual's cognitive abilities, vocational status, family integration, social functioning, and independence in daily routines (Olver et al., 1996; Hoofien et al., 2001; Mateer and Sira, 2006). Memory and attentional impairments interfere with virtually every aspect of the person's daily life, including them, returning to work (Hoofien et al., 2001; Mateer and Sira, 2006). Memory impairment following TBI is almost universal and often persistent (Hoofien et al., 2001; Mateer and Sira, 2006).

Mittenberg et al. (1996) reported there are mood symptoms as part of the impairment. These symptoms include complaints of irritability, fatigue, headache, depression, anxiety, light sensitivity, and sound sensitivity. These symptoms may remit spontaneously in some patients, though Levin et al. (1987) suggested in many these symptoms continue for months after sustaining mild head trauma. Interestingly, Alves et al. (1986) revealed patients who experience two or more symptoms at three months after injury was probable to experience a similar number of symptoms after 6-12 months. Not surprisingly, Hoofien et al. (2001) examining 76 participants with severe TBI found the individuals with severe TBI, exhibit psychiatric symptomatology, and faced more family and social struggles post-injury. The individuals exhibited higher scores for hostility, depression and anxiety.

These neuro-behavioral problems of TBI affected the individuals and family functioning with devastating consequences (Hoofien et al., 2001). Having TBI impairs and changes the overall quality of life, interpersonal, occupational, and social functioning. The situation results in psychological distress in their spouse and caregivers (Fann et al., 1995; Hoofien et al., 2001; Harris et al., 2001; Mateer and Sira, 2006).

Major depression is a common psychiatric complication among patients with TBI (Deb et al., 1999; Jorge et al., 2004; Roy et al., 2018). Koponen et al. (2002) evaluated sixty subjects for about 30 years after the traumatic brain injury. The study concluded in some individuals with TBIs, there is an apparent vulnerability to psychiatric illness as the traumatic brain injury caused protracted, lasting impairment. Similarly, Koponen et al. (2002) establish depressive episodes, delusional disorder, and personality disturbances were prevalent in these persons with the brain injury. Fann et al. (1995) followed up fifty patients with traumatic brain injury, a quarter of the patients had current major depression while another quarter had a first-onset major depressive episode after the injury that had resolved. In another quarter of the patients had a present generalized anxiety disorder, and in (8%) reported current substance abuse. Roy et al. (2018) followed-up 103 subjects with first-time TBI. The subjects were assessed within 12 months post-injury and evaluated for the development of new onset depression at 3, 6, and 12 months. Roy et al. (2018) revealed more than half of the subjects developed new onset depression and the risk of depression ensued with the decreased social functioning post-TBI.

The depressed and anxious subjects regarded their injuries and their cognitive functioning more debilitating (Jorge et al., 2004; Fann et al., 1995). Everyday problems, i.e., the disabilities arising from impaired mental functioning are the most handicapping for the depressed and anxious individuals, thus affecting their families. The presence of major depression after the TBI often raises the individuals' and their family's struggles. Similar to people without TBI, there is an increased risk of suicide (Roy et al., 2018).

Several patients with TBI take longer to return to their pre-injury functioning (Rabinowitz and Levin, 2014; Marsh et al., 2016). These patients' recovery can be incomplete and complicated by preexisting comorbid problems such as chronic pain,

depression, substance abuse, life stress, and unemployment, and protracted litigation (Rabinowitz and Levin, 2014; Marsh et al., 2016; Ponsford et al., 2000).

The literature review found evidence stating the leading causes of TBI are falls, motor vehicle crashes, struck by or against events, and assaults (Langlois et al., 2006; Ahmed et al., 2017; Hoofien et al., 2001). Additionally, the literature review showed an enormous volume of the link between alcohol and almost all kinds of unintentional injuries. Ponsford et al. (2007) investigating 121 hospital in-patients with TBI, documenting pre-injury alcohol and drug use, and with 133 demographically similar controls, discovered 31.4% of the TBI group and 29.3% of controls were drinking at hazardous levels. Interestingly, Ponsford et al. (2007) the alcohol and drug use declined in the first year post-injury, but subsequently recurred two years post-injury. Ponsford et al. (2007) added heavy alcohol use post-injury were among the young, male and heavy drinkers pre-injury.

Thus, the literature review revealed a high rate of psychiatric disorders among people with TBI. Many studies emphasize the importance of psychiatric follow-up after traumatic brain injury.

1.5) <u>Relationship of alcohol use and traumatic brain injury (pre or post injury)</u>

Some investigators characterized people with TBI as having: complicated vs. uncomplicated mild traumatic brain injury (MTBI) (Borgaro et al., 2003; Iverson, 2006). Complicated TBIs are those with pre-existing psychiatric problems or substance abuse problems. The patients with complicated MTBIs performed significantly poorer (Borgaro et al., 2003; Iverson, 2006). Borgaro et al. (2003) revealed individuals in the complicated group showed greater cognitive and affective disturbances. In the early 90's Corrigan (1995) discovered and alerted the medical field regarding substance abuse among persons with TBI. Corrigan (1995)'s work established one third to one half of hospitalizations were related to alcohol intoxication. Corrigan (1995)'s work and the subsequent literature that followed alerted the community to substance abuse and specifically regarding alcohol usage and TBI.

Corrigan (1995) in the earlier work on substance use and traumatic brain injury, found half of the persons were intoxicated at the time of injury and hospitalized for the TBI. Corrigan (1995) subsequently disclosed, in 55–66%, of the subjects there was pre-TBI history of alcohol misuse. The study by Hibbard et al. (1998) similarly showed a significant percentage of individuals presented with substance use disorders prior to their TBI, which supported Corrigan (1995)'s findings. Many other studies support Corrigan (1995)'s findings (Kraus et al., 1989; Dikmen et al., 1995; Kreutzer et al., 1996a; Bombardier et al., 2002; Phelan et al., 2002; Connor et al., 2005).

What is the significance of alcohol use and traumatic brain injury? The relationship of alcohol and patients with traumatic brain injury can occurred either before the trauma or after the trauma (Bombardier et al., 2002; Ahmed et al., 2017). Brennan et al. (2015) disclosed in 30–72% civilians admitted to the hospitals with mild TBI were acutely intoxicated. Yue et al. (2017) cautioned the effects of acute intoxication with alcohol on the acute care and long-term outcomes following mild TBI is real. In many instances, it is life-threatening. Remarkably, Yue et al. (2017) showed a direct link between BAL¹ with increased loss of consciousness. Brennan et al. (2015) and Yue et al. (2017) theorized the increase in alcohol level depresses consciousness. The reduced in level of alertness, leads to increased risk of injury resulting from a combination state of decreased inhibition, decreased awareness, and delay in seeking fitting attention following the injury.

Researchers found intoxicated individuals had a more severe injury (Kraus et al., 1989; Gurney et al., 1992; Zink et al., 1993; Zink et al., 1998). Gurney et al. (1992) found individuals with alcohol were more likely to require intubation, develop pneumonia, and had respiratory distress. Zink and Feustel (1995) found in ethanol-treated animals following brain injury, hypoxia, and prolonged apnoea ensues.

Zink et al. (1998) discovered the presence of alcohol significantly shortened survival time, as the presence of the alcohol suppresses ventilation and hyper-capnia respiratory drive following the TBI (Zink and Feustel, 1995).

von Heymann et al. (2002) noticed an increase in posttraumatic infectious complications, Guidot and Hart (2005) showed an increased risk of acute respiratory distress syndrome. Following any trauma, alcohol causes changes in the body's physiological response leading to increased complications and mortality (von Heymann et al., 2002). Acute alcohol intoxication impairs the hemodynamic counter-regulatory response to hemorrhagic shock (Molina, 2005; Bird et al., 2009). Phelan et al. (2002) showed an accentuation of tissue injury in alcohol-intoxicated rats. Bird et al. (2009) further showed evidence supporting Phelan et al. (2002)'s. Bird et al. (2009) discovered alcohol intoxication accentuates the rise in alanine transaminine (Organization and Unit) and base deficit during trauma or hemorrhage. The discovery highlighted the presence of tissue injury resulting from the marked hypotension seen in alcohol-intoxicated animals. Bird et al. (2009) additionally showed pro-inflammatory cytokine response to hemorrhage following alcohol-intoxication. In animal studies, Zink and Feustel (1995) and Katada et al. (2009) found in animal studies, elevated intracranial pressure, acidemia, hypoxia, and decreased respiratory drive occurs in the presence of alcohol, with consequences of increased mortality (Katada et al., 2009).

Salim et al. (2009a) in the largest database review in 38 019 patients with TBI, patients with isolated moderate to severe TBI and with a positive serum ethanol level died

less frequently than their ethanol-negative counterparts. The notion is supported by Bernier and Hillary (2016) review of the trend of alcohol- related TBI over the past two decades. Bernier and Hillary (2016) study disclosed the patients admitted to the emergency department for TBI and who tested positive for alcohol had higher rates of survival. Bernier and Hillary (2016) hypothesized could alcohol have subtle protective effects? Bernier and Hillary (2016) further hypothesized alcohol is a known risk factor for TBI, however, the number of overall injuries would be reduced in the absence of intoxication.

Nevertheless, patients tested positive with alcohol faced increase complications. Several other studies reported the similar observations of increase complications seen in patients tested positive for alcohol (Jurkovich et al., 1993; Li et al., 1997).

Jurkovich et al. (1993) investigated 427 patients admitted to a tertiary referral hospital during a 23-month period. Similar to Salim et al. (2009a)'s findings, Jurkovich et al. (1993) disclosed acute intoxication also did not increase the risk of complications and mortality. However, Jurkovich et al. (1993) concluded chronic and not acute, alcohol abuse adversely affects outcome from trauma. Fabian and Proctor (2002) studying the clinically relevance of levels of acute ethanol and its influence on the cerebral perfusion pressure concluded ethanol after TBI may not affect mortality provided there is cardiopulmonary support. The conclusion is understandable as the deleterious effect of alcohol is greater in TBI with cerebral hemorrhage (Jurkovich et al., 1993; Salim et al., 2009a).

The literature search showed a diversified and curious consequence. Many studies support the hypothesis regarding serum ethanol level and TBI outcome (Luna et al., 1984; Salim et al., 2009a; Opreanu et al., 2010; Brennan et al., 2015). Several determine the impact of alcohol with increased mortality and the likelihood of respiratory complications

(Luna et al., 1984; Kraus et al., 1989; Katada et al., 2009; Yue et al., 2017). Interestingly, Mohseni et al. (2016) discovered among patients admitted between January 2007 and December 2011, and admitted to an academic trauma centre, the patients with positive blood alcohol level (BAL) were significantly younger with less co-morbidities. Additionally, the cohorts exhibited no significant difference in the severity of the intracranial injury with patients with no blood alcohol.

Many reports show the relationship between alcohol and nearly all types of unintentional injuries correlate with the blood alcohol concentration (BAC) (Tien et al., 2006; Taylor et al., 2010; Phillips and Brewer, 2011; Rehm, 2011). It shows an exponential dose response relationship (Taylor et al., 2010). The acute effects of alcohol consumption on injury risk are mediated by how regularly the individual drinks.

Studies have shown that alcohol consumption will lead to road traffic accidents mainly because it impaired the judgement and attitude of the driver which resulted in risk taking maneuvers and the driving because unsafe (Martin et al., 2013; Zhao et al., 2014). It is shown in a study that alcohol consumption causes injury in a dose-response manner and the risk increases non-linearly with increase alcohol consumption (Taylor et al., 2010). In another study done, noted that dependent alcohol drinking and binge drinking were more common among patients with head trauma compared to other types of trauma (Savola et al., 2005).

1.6) Road traffic accident and head injury

Road traffic accidents are part of unintentional injuries (Rehm et al., 2012; Rehm et al., 2013).

These traffic accidents are one of the leading causes of death and disability worldwide (World Health Organisation, 2002; Lopez and Murray, 1998; Krug et al., 2000; Gore et

al., 2011). Its impact is increasingly noted in many developing countries (Nantulya and Reich, 2002; Ameratunga et al., 2006). Road traffic injuries comprise approximately 3% of all global deaths (World Health Organisation, 2002), are the main cause of death in people under 30 (Mayou et al., 1993).

Several reports discovered more than half of patients with mild TBI are survivors of motor vehicle accidents (Tien et al., 2006; Salim et al., 2009a; Ruffolo et al., 1999). Unintentional injuries from road-traffic accidents are the second leading cause of disability-adjusted life years worldwide (World Health Organization, 2007; Gore et al., 2011; Hughes et al., 2015). Thus, the evidence supports road traffic accidents as an important cause of morbidity. The World Health Organization (2007) reported disability rate as a consequence of road traffic accidents range about 6% of total disability-adjusted life years in high- income countries to 16% in both southeast Asia and the eastern Mediterranean. The World Health Organization (2013) reported in half of the world's road traffic deaths occur among motorcyclists (23%), pedestrians (22%), and cyclists (5%). As a matter of fact, Gore et al. (2011) considered road-traffic accidents ranked second among daily-adjusted life years and the fifth leading cause for violence.

Road traffic injuries trigger enormous economic consequences to victims, their families and to society (Odero et al., 1997; Gupta et al., 2015), particularly among adolescents and young adults (Odero et al., 1997; Peden et al., 2004). Added to the worry is that in some developing countries there is a dramatic increase in the number of traffic fatalities (Odero et al., 1997; Nantulya and Reich, 2002; Ameratunga et al., 2006; Peden et al., 2004). Ameratunga et al. (2006) believed the increasing burden of road-traffic injuries further adds to the strain on the countries' medical and mental health services. Kumar et al. (2008) believed the increase in population and the number of motor vehicles on the road, were factors affecting fatalities in vehicular accidents.

Reports demonstrated many individuals involved in the road-traffic accidents tend to be younger (Ruffolo et al., 1999; Keyser-Marcus et al., 2002). Thus, if the symptoms of the TBI, persist returning to living and working is a challenge (Kreutzer et al., 1996a; Ruffolo et al., 1999; Keyser-Marcus et al., 2002). For many victims, not resuming work is a huge concern for themselves, socially and economically (Mayou et al., 1993; Ruffolo et al., 1999; Keyser-Marcus et al., 2002). The presence of psychiatric morbidity aggravates the individuals' post-accident challenges (Mayou et al., 1993). Interestingly, Levin et al. (1987) investigating neuro-behavioral functioning in 57 patients post-minor head-injury revealed nearly all the patients' cognitive or somatic complaints, and emotional malaise, resolve at the three months assessment. Levin et al. (1987) suggested that one uncomplicated minor head injury results in no permanent disability and neurobehavioral impairment in a majority of patients and who are free of preexisting neuropsychiatric disorder and substance use.

Studies shown that alcohol consumption is another factor causing road traffic accidents (Tien et al., 2006; Salim et al., 2009a; Zhao et al., 2014; Yue et al., 2017). Using alcohol while driving under its influence (DUI) is a serious traffic offence (Foster and Dissanaike, 2014; Jones, 1991; Foster et al., 1988). Drunk drivers place themselves and many innocent people at risk (Foster et al., 1988).

The review found mixed results regarding the presence of alcohol and its association with brain injury and the person's morbidity and mortality.

The presence of alcohol depresses an individual's consciousness (Yue et al., 2017; Brennan et al., 2015). Accidents occur mainly because its presence impairs the judgement and attitude of the drivers which resulted in risk taking maneuver's and the driving because unsafe (Martin et al., 2013; Zhao et al., 2014). Drivers driving under the influence of alcohol exhibit reckless driving behaviors seen by the increased rates of vehicle crashes, moving violations, and traffic tickets (Van Dyke and Fillmore, 2014). The poor behavioral self-regulation could also increase sensitivity to the disruptive effects of alcohol on driving performance (Irwin et al., 2017; Mundt and Perrine, 1993).

A number of longitudinal studies have highlighted ongoing cognitive, behavioural and emotional sequelae of post-accident brain injury (Hoofien et al., 2001; Olver et al., 1996). Luna et al. (1984) investigating motor vehicle accidents among motor cyclists found in a quarter of the 134 subjects surveyed, were intoxicated. Luna et al. (1984) established the intoxicated group had a fourfold increased mortality rate, similar to opinions of (Oscar-Berman and Marinkovic, 2003) and Salim et al. (2009b). Savola et al. (2005) studied a group of 345 patients presenting to the hospitals for trauma and to investigate the relationship of different patterns of alcohol intake to various types of trauma. The study revealed dependent alcohol drinking and binge drinking were found to be significantly more common among patients with head trauma than in those with other types of trauma.

Even in Malaysia, researchers noted traumatic head injury is a leading cause of trauma seen in several general and tertiary hospitals (Sethi et al., 2002; Jeng et al., 2008; Ministry of Health Malaysia, 2011; Jamaluddin et al., 2009). Sethi et al. (2002) reported the head injuries were severe and requiring admissions to a tertiary care hospital. Moreover, the research noted the severity of the injury increased the consequences of the person's disability. Major trauma constitutes only 1.2% of the total trauma admissions, yet the extend of its mortality and morbidity causes a key burden to the society (Sethi et al., 2002; Jeng et al., 2008; Ministry of Health Malaysia, 2011; Jamaluddin et al., 2009). Moreover, motor vehicle accidents play a foremost role in the death of our young and productive population (Jamaluddin et al., 2009). The younger age group (15-34years old) consist of 56.6% of the major trauma cases (Ministry of Health Malaysia, 2011; Jamaluddin et al., 2009).

Such is the importance of trauma in Malaysia, the first Malaysian National Trauma Database was launched in May 2006 Sabariah et al. (2008). The study examined five tertiary referral centers and collected data on major traumas. The report aimed to look at the management of severe trauma and improve trauma care. Road traffic injury contributes significantly to major morbidity and mortality in a developing country as Malaysia (Sabariah et al., 2008). Sabariah et al. (2008) found road traffic accident made up close to 73.6% of injuries with 65% involving motorcyclist and pillion rider. The report did not state alcohol-related injury. However, Sabariah et al. (2008) concluded the findings suggested Malaysia has higher death rates from road traffic accident.

1.7) Prevalence of Alcohol use in Malaysia

The actual prevalence and alcohol-related disorders in the Malaysian community is unknown.

Malaysia is a multicultural and ethnicity country. In the early days, abstinence of alcohol is a norm in Malaysia during the sultanate ruling and only the indigenous population of Sarawak and Sabah practice drinking of rice wine. However, during the colonial era, European merchants had introduced alcohol to the workers brought in for mining and plantation which started the boom of alcohol industry in Malaysia. Alcohol use is not banned in Malaysia however is prohibited for the Muslim population in Malaysia (Jernigan and Indran, 1997).

There are few studies on patterns of alcohol usage in Malaysia. Based on the recent National Health and Morbidity Survey in 2015, it is reported that 14.5% of population had consumed alcohol before whereas 8.4% of adults of 18-years and above currently consumed alcoholic beverages (Institute for Public Health, 2015). From the

statistics, it is shown that the risk factor of alcohol consumptions is those from urban areas, males, other Bumiputras, age group of 25-29-years-old, singles, those with tertiary educations and those from higher income group (Institute for Public Health, 2015).

Another study done previously to determine the pattern of alcoholism in inpatient patients General Hospital showed that the prevalence of alcohol abusers/dependents was 10.6% of total patients admitted during the duration of study. However, the prevalence of patients who drank alcohol were 52% (Saroja and Kyaw, 1993)

1.8) Prevalence of alcohol use and mortality from motor vehicle accidents (MVA)

The Royal Malaysian Police (2007) reported in the 2006, the number of road accidents in was 341,232 of which 6,287 were road fatalities. The numbers were considered high for a population of 26,640,000, giving an index of 23.5 road fatalities per 100,000 inhabitants (Royal Malaysian Police, 2007; Abdelfatah, 2016).

Most of the research done in Malaysia and regarding road traffic accidents were among motor-cyclists (Rahman et al., 2015; Ramli et al., 2014; Kulanthayan et al., 2007). Motorcycle fatalities constitute the majority of road traffic deaths in Malaysia (Rahman et al., 2015; Ramli et al., 2014). Moreover, there are a significant number of deaths for both the motor-bike rider and their pillion rider (Rahman et al., 2015; Ramli et al., 2014).

There are no actual studies done in Malaysia to determine the prevalence of alcohol use among patients with traumatic brain injury. However, there are several studies conducted for fatality cases in motor vehicle accidents whereby the blood alcohol concentration was done that showed alcohol intoxication among the patients. From the fatal cases, most common cause of death was due to head injury (Ramli et al., 2014). Mohamed et al. (2012) examining fatal accidents in the Klang Valley found 11% of the fatality tested positive for drugs, which close to a quarter of the drivers were positive for alcohol. Mohamed et al. (2012) found in 2.3% of fatally injured drivers had both alcohol

and drugs in their bodies.

Odero et al. (1997) reviewed the epidemiology of motor vehicle accidents in developing countries and their association with alcohol. Despite the varying definitions, measurement methods and data completeness, the study found the significant association between alcohol and trauma. Odero et al. (1997) men were predominantly at risk than women of injury in crashes. Odero et al. (1997) disclosed males where often drivers in motor-vehicles and even cyclists. Moreover, a high proportion of males involved as pedestrians, and passengers suggesting the co-existence of other social and behavioral factors contributing to their vulnerability.

In a study done whereby a compilation was made for alcohol related traffic injuries and fatality in developing countries, it is noted that the prevalence of had a wide range from 8.5% up to 60% depending on how the alcohol test was being conducted whether by blood alcohol level, breath analyzer or from interview (Odero et al., 1997). One of the studies mentioned in the compilation was from our neighbouring country Singapore, and this study noted the prevalence of alcohol use prior to road traffic accident was around 10% (Wong et al., 1990)

A study showed that pre-injury alcohol use were highly predictive of post-injury alcohol use and problems (Bombardier, Temkin, Machamer, & Dikmen, 2003). It was found out that alcohol or drug use will declined during the 1st year post-injury however the quantity and frequency will increase over time and possibly returned to pre-injury level (Kreutzer, Witol, & Marwitz, 1996). The risk factor for post injury heavy alcohol consumption are those who are young, male, and had history of heavy drinking pre-injury (Ponsford, Whelan-Goodinson, & Bahar-Fuchs, 2007)

2) CHAPTER 2: RATIONALE AND OBJECTIVES OF THE STUDY

36

2.1) The rationale of this study

1. There is **limited information** available in Malaysia regarding the prevalence of alcohol use among traumatic brain injury patients.

2. To evaluate the extent of the problem so that necessary measures could be planned in the future. Early detection of problematic alcohol use can enable earlier intervention to improve outcome.

3. To evaluate the burden of alcohol or any substance related problem among patients with traumatic brain injury.

2.2) The objectives of this study include:

1. To determine the pattern of alcohol use among patients with traumatic brain injury in urban hospital of Malaysia.

2. To investigate the association of alcohol use in traumatic brain injury with:

a) socio-demographic characteristic

b) duration of injury and severity of injury

c) past history of alcohol use/substance use

d) psychological wellbeing

e) cognitive function

3) CHAPTER 3: METHODOLOGY

3.1) Site and subjects

This is a cross sectional study and it was conducted in 2 hospitals in Kuala Lumpur namely University Malaya Medical Centre and Hospital Kuala Lumpur. Both hospitals are tertiary centre for referrals especially complicated cases of traumatic brain injury which are severe and require urgent interventions. Both centres are government sponsored facilities.

The subjects included in the study consists of mainly outpatient cases which came for follow up in the neurosurgical clinic, neurorehabilitation clinic, emergency department (observation ward), and some stable admitted patients in the neurosurgical ward. The diagnosis of traumatic brain injury will be based on clinical notes that was reviewed during the sample collection day by the researcher.

Patients recruited were mainly based on convenience sampling and depends on both availability of the researcher and attendance of patient in the follow up clinic on that particular day. All patients were included in studies unless patient deemed too ill such acute cases still restless, comatose or those chronic cases but is globally aphasic, or having severe comprehension deficits.

Informed consent was obtained from all subjects or their substitute consent givers after a detailed description of the study.

Sample size calculation

Sample size calculation was based on the KISH formula.

 $n = \frac{t^2 x p(1-p)}{t^2 p(1-p)}$

m²

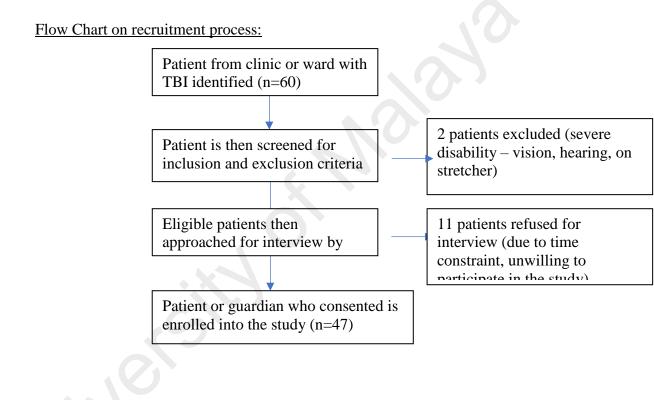
Description:

- n = required sample size
- t = confidence level at 95% (standard value of 1.96)
- p = estimated prevalence of substance use disorder in the area*

• m = margin of error at 5% (standard value of 0.05)

Based on a systematic review study of prevalence of substance use and Traumatic brain injury – it showed the prevalence range between 37-51% prior to the injury (Parry-Jones et al., 2006). Thus, prevalence of 37% is taken for this study.

 $n = (1.96)^2 x (0.37) (1-0.37) / (0.05)^2 = 358$



3.2) Measures

During the interview, data on demographics will be obtained using a standardized questionnaire. Further details regarding the alcohol consumption before and after the trauma as well as the further details of the trauma was taken as well using a standardized questionnaire. Both questionnaires had been given to experts of the field for opinions and validated by them. Questionnaire on alcohol use was reviewed by Associate Professor Dr. Amer Siddiq (Consultant Psychiatrist) and Dr. Abdul Razak (Consultant Psychiatrist).

Questionnairea on the traumatic brain injury part was reviewed by Associate Professor Dr. Sia Sheau Fung (Consultant Neurosurgeon) and Datuk Dr. Hj. Johari Siregar (Consultant Neurosurgeon).

a) Assessment of Alcohol use disorder

Assessment of alcohol use whether is hazardous or dependence using the AUDIT scale (Interviewer version) either in English or Malay language. The AUDIT-M has been validated among alcohol users in Malaysia and showed a significant correlation between AUDIT and AUDIT-M score (Spearman's $\rho = 0.979$, p < 0.01), Cronbach α coefficients for the total AUDIT-M was 0.823 thus is suitable for AUD assessment in Malaysia(Yee et al., 2015).

AUDIT is a free scale developed by World Health Organization (WHO) as a method of screening for excessive drinking and to assist in brief assessment. It consisted of 10 items with each response are scored between 0 to 4 and total maximum score of 40 points. A study done by Saunders et el (1993) showed that 92% of patients with a cutoff point of \geq 8 had hazardous or harmful alcohol use while 94% of non-hazardous drinkers scored < 8 points(Saunders et al., 1993).

b) Assessment of General psychological wellbeing

Assessment of the subject's psychological wellbeing is done by using GHQ-12 (English and Malay version).

GHQ-12 is a 12-item questionnaire which is quick and easy to be conducted. It is also reliable and sensitive and ideal for research studies. It is however use mainly as screening tools rather than a diagnostic tools(Goldberg, 1972). The GHQ-12 (Malay) has been validated among medical students in Malaysia and it showed sensitivity of 81.3% and

specificity of 75.3% at cut-off point of 3 or 4 with a Cronbach α value of 0.85(Yusoff et al.).

c) Assessment of Brain injury severity

Assessment of the severity of the traumatic brain injury done by Glasgow Coma Scale (GCS).

It is a 15-point scale for estimating and categorizing the outcome of brain injury. It is also reliable in trauma patient intoxicated with alcohol. The GCS score information is obtained from the clinical notes of patient during the onset of the traumatic event.

d) Assessment of Cognitive function

Assessment of cognitive function was done using Montreal Cognitive Assessment (MOCA)- English and Bahasa Malaysia version

It is a 30-point scale for assessment of few components of the cognitive functions mainly visuospatial/executive, naming, memory, attention, language, abstraction, delayed recall and orientation. It is can be used for screening of cognitive impairment in TBI patients(Wong et al., 2012).

e) Assessment of physical disability

Assessment of physical disability was done using Modified Rankin Scale (mRS). This scale is commonly used for acute stroke patients and people with neurological deficits to measure the functional outcome(Kasner, 2006). There was a study which showed that the mRS at discharge has strong correlation with signal-intensity abnormality in MRI brain (r=0.772, p <0.01)(Schaefer et al., 2004). It is an observer rated with scoring to measure the physical limitation in the patients. It is rated as:

- 0- No symptoms
- 1- No significant disability. Able to carry out all usual activities despite some symptoms

- 2- Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities.
- 3- Moderate disability. Requires some help but able to walk unassisted.
- 4- Moderately severe disability. Unable to attend to own body needs without assistance and unable to walk unassisted.
- 5- Severe disability. Requires constant nursing care and attention, bedridden, incontinent.
- 6- Dead

A cut off point of MRS 3 or more was taken as having significant physical disability and used in trials as poor outcome(Sulter et al., 1999).

3.3) Data Analysis

All the statistical analyses were performed using SPSS computer software. The socio-demographic data, alcohol questionnaires, head injury questionnaires, AUDIT score, GHQ-12 score and MOCA score were summarized using descriptive statistics. For the continuous data such as age group and salary will be presented in term of mean and standard deviation. For the categorical data, such as gender, race, occupation, and so on will be presented in either a pie chart or bar chart for better visualization. As for the association part, all the continuous data and those with multiple categories are grouped into yes and no categories. The data will be then inserted in 2x2 tables to analyze using cross tabulation with Chi-square technique. Variables and outcome of alcohol use post TBI or risk of pre TBI alcohol were used for the cross-tabulation analysis. Data will be presented in term of significant and odds ratio will be taken. Result were considered significant at p<0.05.

4. CHAPTER 4: RESULT

Throughout the study period November 2017 till January 2018, 60 patients were interviewed for the study and sample were taken mainly from UM Rehabilitation Clinic, UM Neurosurgical clinic and ward, HKL Neurosurgical Clinic and Ward, Emergency department (observation ward). Only 47 patients were included in the study, 2 patients unable to complete the questionnaire as having severe disability (vision, hearing, on stretcher), whereas 11 patients had refused for interview due to time constraint, unwillingness to participate in the study.

Variables		Percentage% (n)	Mean (Std. Dev.)
Age, years: mean			36.8 (±14.8)
Sex	Male	83.0% (39)	
	Female	17.0% (8)	
Race	Malay	63.8% (30)	
	Chinese	10.6% (5)	
	Indian	23.4% (11)	
.0	Others	2.1% (1)	
Marital Status	Single	46.8% (22)	
	Married	51.1% (24)	
\mathbf{S}	Divorced	2.1% (1)	
Education Status	Primary	17.0% (8)	
	Secondary	51.1% (24)	
	Tertiary	31.9% (15)	
	No Education	0% (0)	
Occupation Status	Government sector	10.6% (5)	
	Private Sector	31.9% (15)	

 Table 1: Socio-demographic Data Table

	Self employed	6.4% (3)	
	Student	6.4% (3)	
	Unemployed	44.7% (21)	
Salary, RM/month:			1429.79
mean			(±2340.28)
Past Medical	None	72.3% (34)	
History	Congenital	4.3% (2)	0
	Acquired	23.4% (11)	S.O.

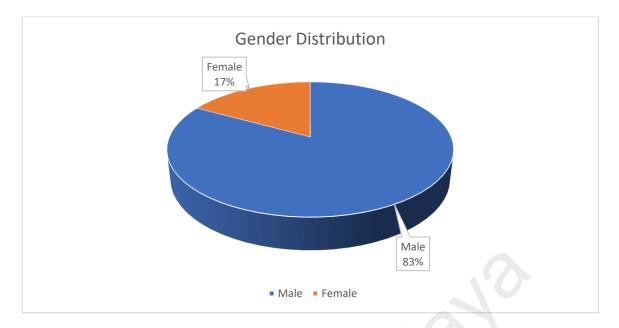
4.1 Questionnaires on Socio-demographic Data

Table 2: Age group.

4.1 Questionnaires on Socio-demographic Data				
Table 2: Age group.				
Age Group	Percentage % (n)			
< 20 years old	2.1% (1)			
20-29 years old	42.1% (20)			
30-39 years old	19.1% (9)			
40-49 years old	17.0% (8)			
50-59 years old	10.6% (5)			
60-69 years old	4.2% (2)			
70-79 years old	4.2% (2)			
Total	100% (47)			

The final study sample was composed of 47 patients. The mean \pm SD age of this group was 36.8 ± 14.8 years, median = 33 years old. The youngest patient was 16 years old whereas the oldest patient was 74 years old.

Figure 1: Gender distribution



83% (39) of patients were male and 17% (8) were females.

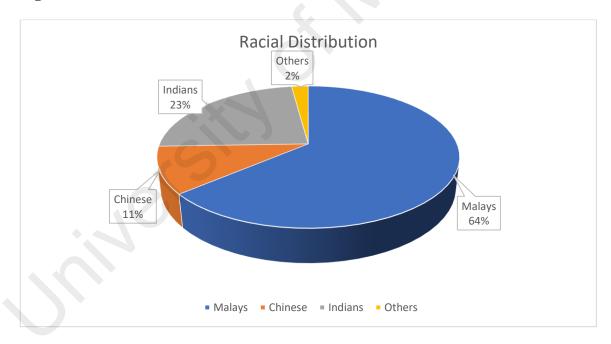
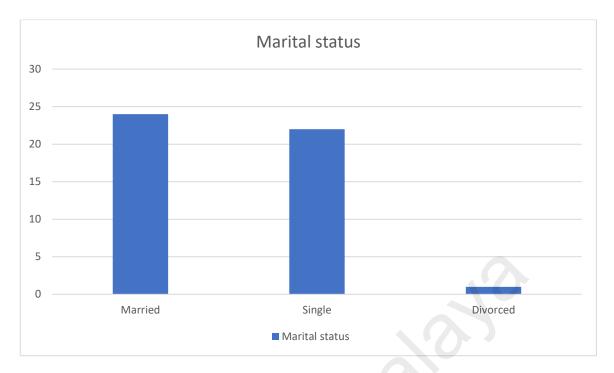


Figure 2: Racial Distribution

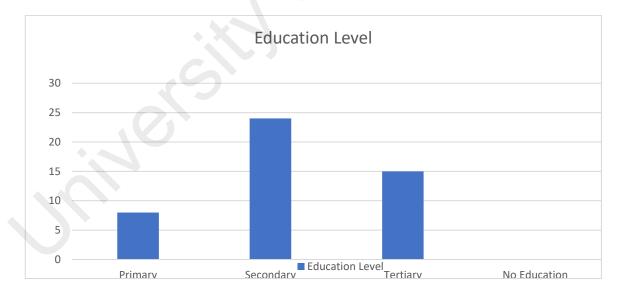
The racial distribution was as follows; Malays 63.8% (30), Chinese 10.6% (5), Indians 23.4% (11) and others 2.1% (1). There was an over representation of Indian patients as compared to the general population.

Figure 3: Marital Status



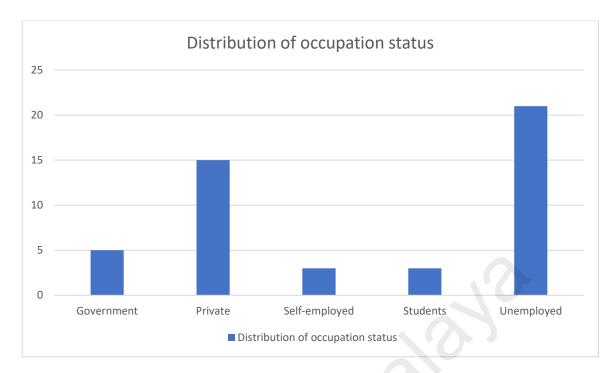
51.1% (24) of the sample were married, 46.8% (22) were single and 2.1% (1) were divorced.

Figure 4: Education status



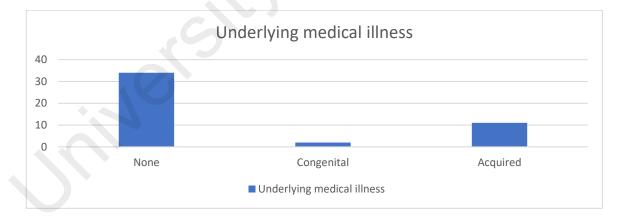
Regarding the education status, 17% (8) were until primary level, 51.1% (24) were until secondary level and 31.9% (15) were until tertiary level. There was no patient without any formal education.

Figure 5: Occupation status



Regarding the occupation, 10.6% (5) worked in the government sector, 31.9% (15) worked in the private sector, 6.4% (3) were self-employed, 6.4% (3) were students and 44.7% (21) were unemployed.





72.3% (34) had no underlying medical illness, 4.3% (2) had congenital medical illness (asthma) and 23.4% (11) had acquired medical illness (such as diabetes mellitus, hypertension, dyslipidaemia)

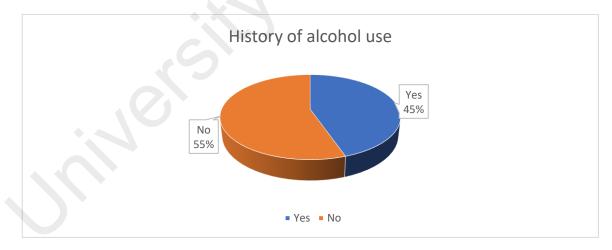
Table 3: Salary

Salary group (per month)	Percentage % (n)
< RM 1000	44.7% (21)
RM 1000-1999	27.7% (13)
RM 2000-2999	12.8% (6)
RM 3000-3999	12.8% (6)
>RM 4000	2.1% (1)
Total	100% (47)

The mean salary for the patients were RM 1429 per month which include those who are not working currently. 20 patients had RM 0 salary whereas there was 1 patient who earns RM 15000 per month.

4.2 Questionnaire on Alcohol use and other substance use (Pre and post TBI)

Figure 7: History of alcohol use (Pre TBI)



44.7% (21) patients had tried on alcohol beverage before the TBI and 55.3% (26) patients never tried on alcohol before the TBI.

The mean age of first time use of alcohol beverage was 20.48 years old.

52.4% (11) out of 21 of the patients had first degree family members using alcohol as well. 95.2% (20) of the patients were males and 4.8% (1) patient was female.

51.1% (24) out of all the patients were using nicotine (smoking cigarette) pre TBI.

66.7% (14) out of 21 patients who tried on alcohol beverage before were using nicotine as well

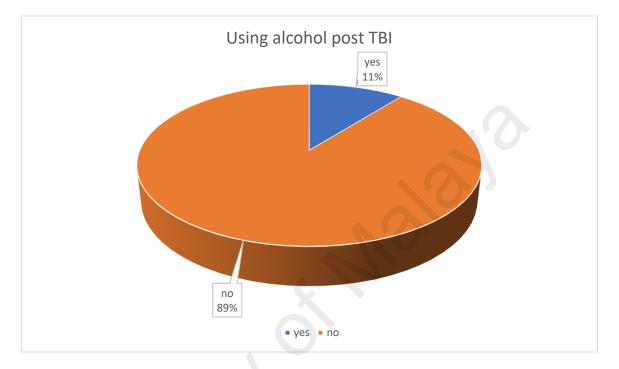


Figure 8: History of alcohol use (Post TBI)

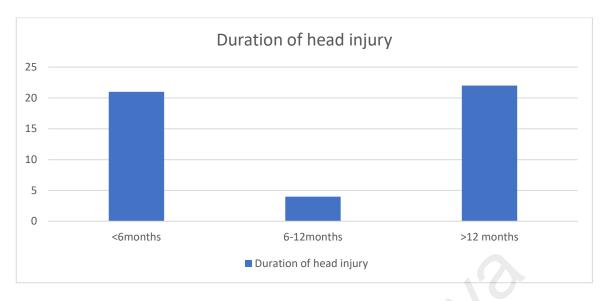
23.8% (5) out of 21 patients who had history of using alcohol pre TBI were still using alcohol post TBI.

10.6% (5) out of all patients were using alcohol post TBI.

62.5% (15) out of 24 patients were still using nicotine post TBI.

4.3 Questionnaires on TBI

Figure 9: Duration of head injury



44.7% (21) had head injury less than 6 months ago, 8.5% (4) had head injury for 6-12 months ago and 46.8% (22) had head injury >12 months ago.

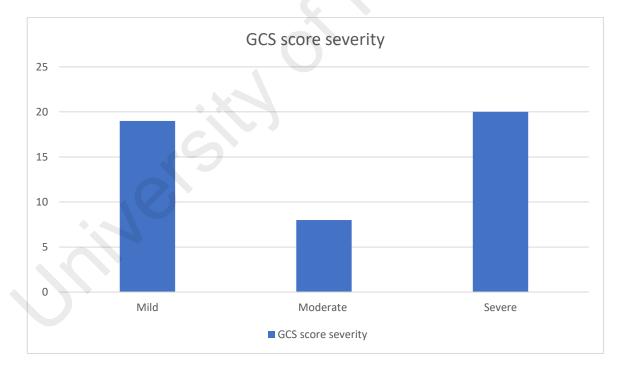
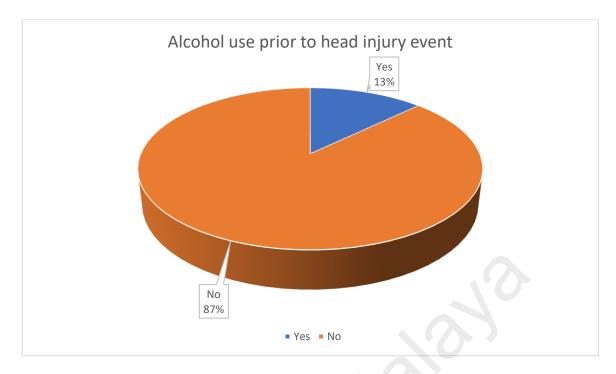


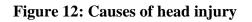
Figure 10: GCS score severity

40.4% (19) had mild GCS score severity, 17.0% (8) had moderate GCS score severity and 42.6% (20) had severe GCS score severity. All of the GCS scoring was made during 1st arrival of patient and documentation in emergency department.

Figure 11: Alcohol use prior to head injury event



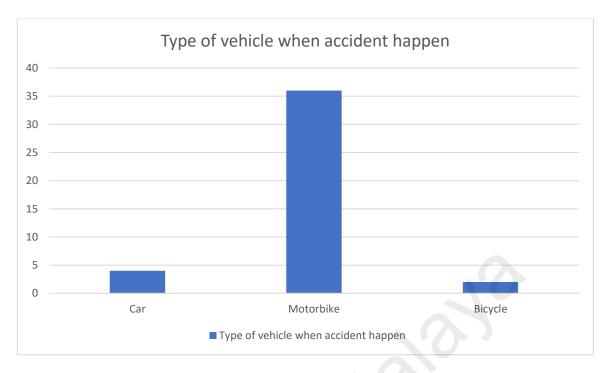
12.8% (6) had use alcohol prior to the head injury event.





Out of the 47 patients, the causes of head injury for 89.4% (42) patients were due to road traffic accidents, 8.5% (4) due to fall and 2.1% (1) due to fights.

Figure 13: Type of vehicle when accident happen



Among the 42 patients involved in road traffic accidents, 9.5% (4) while using a car, 85.7% (36) while riding a motorbike and 4.8% (2) while riding a bicycle.

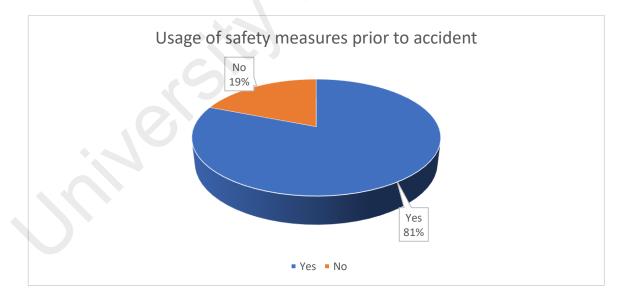
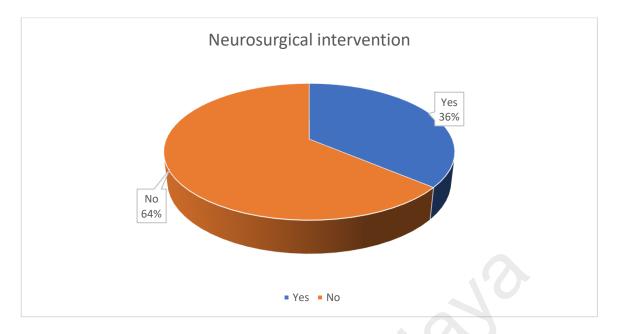


Figure 14: Usage of safety measures prior to accident

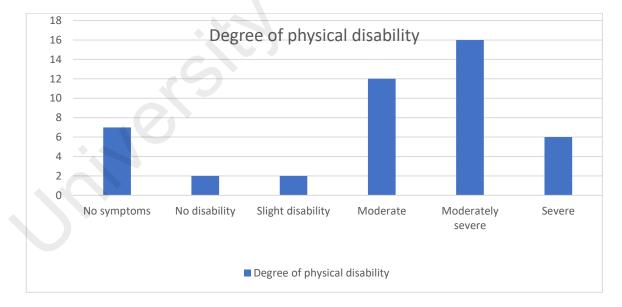
Out of the 42 patients involved in road traffic accident, 81% (34) patients were using safety equipment while driving/riding such as helmet or safety belt and 19% (8) were not using it.

Figure 15: Neurosurgical intervention



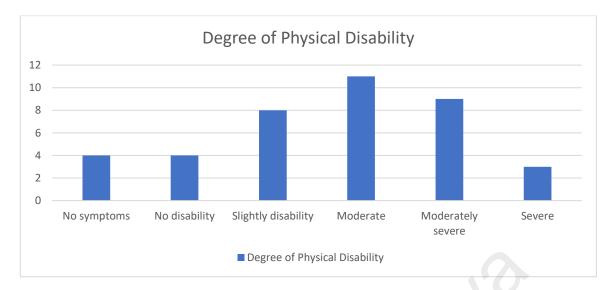
36.2% (17) of patients required neurosurgery procedure (operation) for their head injury and 63.8% (30) were treated conservatively.

Figure 16: Modified Rankin Scale (MRS) – Degree of Physical Disability upon discharge



For MRS on discharge, 45 patients included as 2 patients were not admitted for their head injuries. 26.7% (12) had a score of 3 (moderate disability) and 35.6% (16) had a score of 4 (moderately severe disability)

Figure 17: MRS after 1 month



For MRS after 1 month, 39 patients included as some of the patients just had recent head injury and still in ward during assessment. 28.2% (11) had a score of 3 (moderate disability) and 23.1% (9) had a score of 4 (moderately severe disability)

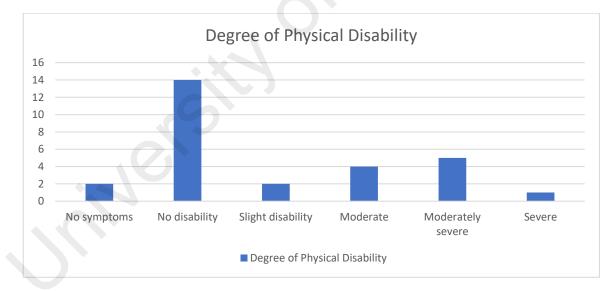


Figure 18: MRS after 6 months

For MRS after 6 months, 28 patients included as some of the patients just had recent head injury and not yet 6 months duration. 50% (14) had a score of 1 (no disability) and 17.9% (5) had a score of 4 (moderately severe disability)

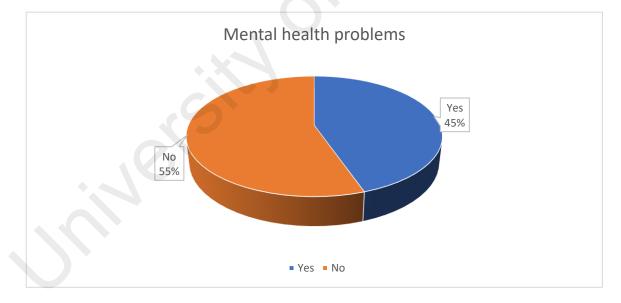
Figure 19: Total AUDIT (Alcohol use disorder identification test) score (cut off point ≥8)



10.6% (5) of patients scored \geq 8 points for AUDIT which indicate hazardous or harmful alcohol use.

Figure 20: Total GHQ-12 (General health questionnaire) score (cut off point \geq 3,

scoring using 0-0-1-1 method)

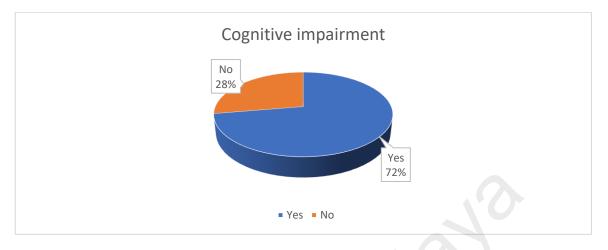


The mean score for GHQ-12 is 2.49. Cut off point of \geq 3 is based on the mean score and previous NHMS 2015 cut off point used.

44.7% (21) of the patients scored \geq 3 indicating presence of mental health problems during the 1-month period during assessment.

Figure 21: Total MOCA (Montreal Cognitive Assessment) score (cut off for normal

\geq 26/30, add 1 point if \leq 12 years education)



The mean MOCA score were 21.6.

72.3% (34) of the patients scored <26 score for MOCA indicating presence of cognitive

impairment.

Figure 22: Language score in MOCA

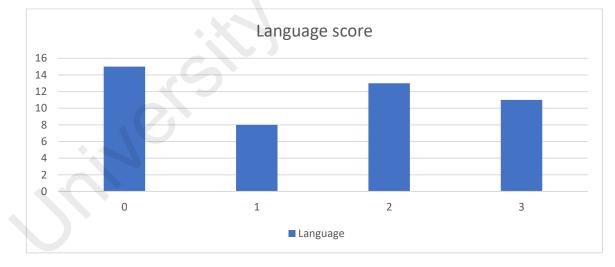
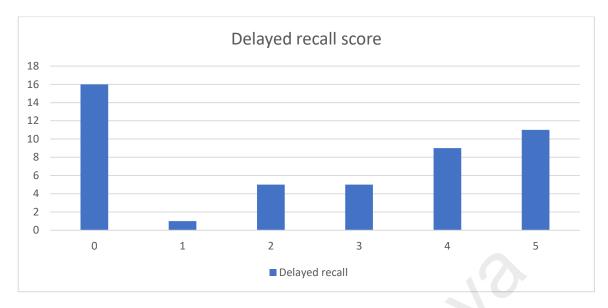


Figure 23: Delayed recall score in MOCA



In term of components of MOCA assessment, 2 main components which patients had difficulties were language and delayed recall. For language, 31.9% (15) scored 0 in the section and for delayed recall 34.0% (16) scored 0 in the section.

		Post TBI alco	ohol use	X ²	Odds Ratio
		Yes	No		
Age 20-29	Yes	3 (15.0%)	17 (85.0%)	0.697**	0.45 (95%
years old	No	2 (7.4%)	25 (92.6%)	_	CI 0.07 –
					3.01)
$\langle \cdot \rangle$					
Race	Malay	2 (6.7%)	28 (93.3%)	1.376**	0.33 (95%
	Non-Malay	3 (17.6%)	14 (82.4%)	_	CI 0.05-
					2.23)
		1			
Gender	Male	4 (10.3%)	35 (89.7%)	0.035**	0.80 (95%
	Female	1 (12.5%	7 (87.5%)	-	CI 0.08-
					8.28)

Table 4: Association of variables and post TBI alcohol use

Marital	Married	1 (4.2%)	23 (95.8%)	2.161**	0.21	(9:
status	Single	4 (17.4%)	19 (82.6%)		CI	0.2
					2.01)	
Education	Tertiary	3 (20.0%)	12 (80.0%)	2.031**	3.75	(95
				2.031		
level	Others	2 (6.2%)	30 (93.8%)		CI 25.33	0.5 3)
				0)	
Occupation	Working	3 (13.0%)	20 (87.0%)	0.274**	1.65	(95
status	Not	2 (8.3%)	22 (91.7%)		CI	0.2
	working				10.91)
		C				
Any alcohol	Yes	5 (23.8%)	16 (76.2%)	6.927#	1.31	(95
use history	No	0 (0.0%)	26 (100.0%)		CI	1.(
					1.67)	
	0					
Family	Yes	3 (25.0%)	9 (75.0%)	3.496**	0.18	(95
history of	No	2 (5.7%)	33 (94.3%)		CI	0.0
alcohol					1.26)	
Nicotine use	Vac	2(12.50/)	21 (97 50/)	0.179**	0.67	(04
	Yes	3 (12.5%)	21 (87.5%)	0.179***	0.67	(95
pre TBI	No	2 (8.7%)	21 (91.3%)		CI 4.41)	0.1
					+.41)	

Head Injury	Less than 1	2 (8.0%)	23 (92.0%)	0.391**	0.55	(95%
duration	year				CI	0.08-
	1 year or	3 (13.6%)	19 (86.4%)	_	3.64)	
	more					
			1	1	J	
Hazardous	Yes	3 (60.0%)	2 (40.0%)	14.341#	0.03	(95%
drinking (8	No	2 (4.8%)	40 (95.2%)		CI	0.00-
or more)				1	0.33)	
	I	I		0)	
Mental	Yes	2 (9.5%)	19 (90.5%)	0.050**	1.24	(95%
health	No	3 (11.5%)	23 (88.5)		CI	0.19-
problem		<u> </u>			8.20)	
(GHQ-12 3		Ċ				
or more)						
Cognitive	Yes	3 (8.8%)	31 (91.2%)	0.426**	1.88	(95%
impairment	No	2 (15.4%)	11 (84.6%)	-	CI	0.28-
(MOCA <					12.78	5)
26)						

*Pearson Chi Square **Fisher's Exact Test #p<0.05

1. <u>Social Demographic characteristic and alcohol use in traumatic brain injury</u> patient.

a) Age group (20-29years old-in view of highest prevalence age group for alcohol use) and Post TBI alcohol use

Table 5: Age (20-29years old) and Post TBI alcohol use (Cross tabulation)

		Post TBI alcohol use		
		No	Yes	Total
Age 20-	No	25 (92.6%)	2 (7.4%)	27
29years old	Yes	17(85.0%)	3 (15.0%)	20
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference in the age group (20-29years old) and post TBI alcohol use. It was found that 15% of patients aged 20-29years old still uses alcohol post TBI and 7.4% of patients not in this age group of 20-29years old still uses alcohol post TBI. The relative odds of patients aged 20-29years old still uses alcohol post TBI compared to other age group is 2.21.

p = 0.638

Odds ratio = 2.21 95%CI (0.33-14.64)

b) Race and Post TBI alcohol use

Table 6: Race and Post TBI alcohol use (Cross tabulation)

	.0	Post TBI al	cohol use	
		No	Yes	Total
Race	Malay	28	2 (6.7%)	30
		(93.3%)		
	Non-Malay	14	3 (17.6%)	17
		(82.4%)		
Total		42	5 (10.6%)	47 (100%)
		(89.4%)		

There was no significant difference in race between Malay and non-Malay for usage of alcohol post TBI. It was found that 6.7% of Malay patients still uses alcohol post TBI and 17.6% of non-Malay patients still uses alcohol post TBI. The relative odds of Malay patients use of alcohol post TBI compared to non-Malay patients is 0.33.

p = 0.336

Odds ratio= 0.33 95%CI (0.05-2.23)

c) Gender and Post TBI alcohol use

		Post TBI alcohol use		
		No	Yes	Total
Gender	Male	35 (89.7%)	4 (10.3%)	39
	Female	7 (87.5%)	1 (12.5%)	8
Total		42 (89.4%)	5 (10.6%)	47 (100%)

Table 7: Gender and Post TBI alcohol use (cross tabulation)

There was no significant difference in gender between patients for usage of alcohol post TBI. 10.3% of male patient still uses alcohol post TBI and 12.5% of female uses alcohol post TBI. The relative odd of male patients still uses alcohol post TBI compared to female is 0.8.

p = 1.00

Odds ratio = 0.80 95% CI (0.08-8.28)

d) Marital status and Post TBI alcohol use

Table 8: Marital status and Post TBI alcohol use (cross tabulation)

		Post TBI alcohol use		
		No	Yes	Total
Marital status	Married	23 (95.8%)	1 (4.2%)	24

	Others	19 (82.6%)	4 (17.4%)	23
Total		42 (89.4%)	5 (10.6%)	47 (100%)

There was no significant difference in marital status between patients for usage of alcohol post TBI. 4.2% of married patients still uses alcohol post TBI and 17.4% of others (single, divorced) uses alcohol post TBI. The relative odds of married patients still use alcohol post TBI compared to others status is 0.21.

p = 0.188

Odds ratio = 0.21 95% CI (0.02-2.01)

e) Education level and Post TBI alcohol use

Table 9: Education level a	and Post	TBI alco.	hol use (cross ta	bulation)

		Post TBI alcohol use		
		No	Yes	Total
Education level	Tertiary	12 (80.0%)	3 (20.0%)	15
	Others	30 (93.8%)	2 (6.2%)	32
Total	S,	42 (89.4%)	5 (10.6%)	47 (100%)

There was no significant difference in education level between patients for usage of alcohol post TBI. 20% of patients with tertiary level of education still uses alcohol post TBI and 6.2% of others (primary, secondary level) uses alcohol post TBI. The relative odds of patients with tertiary level education still uses alcohol post TBI compared to others is 3.75.

p = 0.309

Odds ratio = 3.75 95% CI (0.56-25.33)

f) Occupation status and Post TBI alcohol use

		Post TBI alcohol use		
		No	Yes	Total
Occupation	Working	20 (87.0%)	3 (13.0%)	23
status	Not Working	22 (91.7%)	2 (8.3%)	24
Total		42 (89.4%)	5 (10.6%)	47

 Table 10: Occupation status and Post TBI alcohol use (Cross tabulation)

There was no significant difference in occupation status between patient for usage of alcohol post TBI. 13.0% of patients who are working still uses alcohol post TBI and 8.3% of patients who are not working still uses alcohol post TBI. The relative odds of patient who are working and still uses alcohol post TBI compared to those not working is 1.65.

p = 0.666

Odds ratio =1.65 95% CI (0.25-10.91)

2. Alcohol questionnaire and post TBI alcohol use

g) History of alcohol use pre TBI and Post TBI alcohol use

Table 11: History of alcohol use pre TBI an	d Post TBI alcohol use (cross tabulation)

		Post TBI alcohol use		
		No	Yes	Total
History of	No	26 (100.0%)	0 (0.0%)	26
alcohol use Pre	Yes	16 (76.2%)	5 (23.8%)	21
TBI				
Total	L	42 (89.4%)	5 (10.6%)	47

Patients who had history of alcohol use pre TBI was significantly more likely to use alcohol post TBI compared to those who did not had history of alcohol use pre TBI. 23.8%

of patients with history of alcohol use pre TBI will still use alcohol post TBI whereas 0%

of patients who did not had history of alcohol use pre TBI uses alcohol post TBI.

p = 0.013 (p<0.05)

Odds ratio = 1.31 (95%CI 1.03-1.67)

h) Family history of alcohol use and Post TBI alcohol use

		Post TBI alcohol use		0
		No	Yes	Total
Family history	No	33 (94.3%)	2 (5.7%)	35
of alcohol use	Yes	9 (75.0%)	3 (25.0%)	12
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference in family history of alcohol use between patients who uses alcohol post TBI. 5.7% of patient who uses alcohol post TBI has no family history of alcohol use and 25.0% has family history of alcohol use. The relative odds of patient without family history of alcohol use and uses alcohol post TBI compared to those who had family history of alcohol use is 0.18.

p = 0.097

Odds ratio = 0.18 95% CI (0.03-1.26)

i) Nicotine use pre TBI and Post TBI alcohol use

	Post TBI alcohol use		
	No	Yes	Total
No	21 (91.3%)	2 (8.7%)	23

Nicotine use	Yes	21 (87.5%)	3 (12.5%)	24
pre TBI				
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference in nicotine use pre TBI between patients who uses alcohol post TBI. 8.7% of patients who uses alcohol post TBI did not use nicotine pre TBI and 12.5% of patients who uses alcohol post TBI also used nicotine pre TBI. The relative odds of patients who did not use nicotine pre TBI and alcohol post TBI compared to those who use nicotine pre TBI is 0.67.

p = 1.00

Odd ratio = 0.67 95% CI (0.1-4.41)

3. Head injury questionnaire and Post TBI alcohol use

j) Head injury duration and Post TBI alcohol use

Table 14: Head injury duration and Post TBI alcohol use (Cross tabulation)

5		Post TBI alcoho	l use	
		No	Yes	Total
Head injury duration	Less than 1 year	23 (92.0%)	2 (8.0%)	25
\mathbf{O}^{*}	1 year or more	19 (86.4%)	3 (13.6%)	22
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference between head injury duration of patients and post TBI alcohol use. 8.0% of patients with less than one-year duration of head injury still uses alcohol post TBI and 13.6% of patients with one year or more duration of head injury still uses alcohol post TBI. The relative odd of patients with less than one-year duration of

head injury still uses alcohol post TBI compared to those patients whose duration of head

injury is one year or more is 0.55

p = 0.654

Odds ratio = 0.55 95%CI (0.08-3.64)

4. AUDIT and post TBI alcohol use

k) AUDIT score (≥8)- harmful or hazardous drinking and Post TBI alcohol use

Table 15: AUDIT score and post TBI alcohol use (Cross tabulation)

		Post TBI alcohol use		
		No	Yes	Total
AUDIT score	Less than 8	40 (95.2%)	2 (4.8%)	42
	8 or more	2 (40.0%)	3 (60.0%)	5
Total	•	42 (89.4%)	5 (10.6%)	47

Patients with AUDIT score of 8 or more (hazardous drinking) were significantly more likely to use alcohol Post TBI. 60.0% of patients with AUDIT score of 8 or more still uses alcohol post TBI and 4.8% of patients with AUDIT score of less than 8 still uses alcohol post TBI. The relative odds of patients with AUDIT score of 8 and using alcohol post TBI compared to patients with AUDIT score of less than 8 and still uses alcohol Post TBI is 30.0.

p =0.006 (p<0.05)

Odds ratio = 30.0 95%CI (3.06-294.56)

5. GHQ-12 and post TBI alcohol use

l) GHQ-12 score (>2)- presence of mental health problems and Post TBI alcohol use

 Table 16: GHQ-12 score and post TBI alcohol use (cross tabulation)

Post TBI alcohol use	

		No	Yes	Total
GHQ-12 score	Less than 3	23 (88.5%)	3 (11.5%)	26
	3 or more	19 (90.5%)	2 (9.5%)	21
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference between GHQ-12 score and post TBI alcohol use. 11.5% of patients with GHQ-12 score of less than 3 still uses alcohol post TBI and 9.5% of patients with GHQ-12 score of 3 or more still uses alcohol post TBI. The relative odds of patients with GHQ-12 score of less than 3 and using alcohol post TBI compared to patients with GHQ-12 score of 3 or more is 1.24.

p = 1.00

Odds ratio = 1.24 95% CI (0.19-8.2)

6. MOCA and post TBI alcohol use

m) MOCA score (<26)- presence of cognitive impairment and Post TBI alcohol use

Table 17: MOCA	score and Po	st TBI alcohol use	e (cross tabulation)
----------------	--------------	--------------------	----------------------

		Post TBI alcohol use		
		No	Yes	Total
MOCA score	Less than 26	11 (84.6%)	2 (15.4%)	13
\mathbf{O}^{*}	26 or more	31 (91.2%)	3 (8.8%)	34
Total		42 (89.4%)	5 (10.6%)	47

There was no significant difference between MOCA score and post TBI alcohol use. 15.4% of patients with MOCA score of less than 26 still uses alcohol post TBI and 8.8% of patients with MOCA score of 26 or more still uses alcohol post TBI. The relative odds of patients with MOCA score less than 26 and using alcohol post TBI compared to patients with MOCA score 26 or more is 1.88.

p = 0.607

Odds ratio = 1.88 95% CI (0.28-12.77)

7. Pre TBI alcohol use and outcome

n) Pre TBI alcohol use (prior to accident) and GCS (severity)

 Table 18: Pre TBI alcohol use (prior to accident) and GCS Severity (cross tabulation)

		GCS severity		
		Mild and	Severe (<9)	Total
		Moderate		
Pre TBI	No	23 (56.1%)	18 (43.9%)	41
alcohol use	Yes	4 (66.7%)	2 (33.3%)	6
Total		27 (57.4%)	20 (42.6%)	47

There was no significant difference in GCS(severity) between patients who uses alcohol pre TBI. 43.9% of patients with severe GCS score did not use alcohol pre TBI and 33.3% of patient with severe GCS score uses alcohol pre TBI. The relative odds of patient who did not use alcohol pre TBI and having severe GCS score compared to those who uses alcohol pre TBI is 1.57.

p = 1.00

Odd ratio = 1.57 95% CI (0.26-9.53)

o) Pre TBI alcohol use and safety measures (seat belts, helmets)

 Table 19: Pre TBI alcohol use and safety measures (cross tabulation)

Safety measure	

		No	Yes	Total
Pre TBI	No	5 (13.5%)	32 (86.5%)	37
alcohol use	Yes	3 (60.0%)	2 (40.0%)	5
Total		8 (19.0%)	34 (81.0%)	42

Patients with pre TBI alcohol use were significantly more likely not using safety measures during road traffic accidents compared to those who was not using alcohol pre TBI. 60% of patients with pre TBI alcohol use did not use safety measures and 13.5% of patients with no pre TBI alcohol use did no use safety measures. The relative odds of patients with pre TBI alcohol use and not using safety measure compared to those without pre TBI alcohol use and not using safety measure significantly measures is 9.6.

p = 0.04 (< 0.05)

Odds ratio = 9.6 95%CI (1.27-72.53)

p) Pre TBI alcohol use (prior to accident) and neurosurgical intervention

 Table 20: Pre TBI alcohol use (prior to accident) and neurosurgical intervention

 (Cross tabulation)

		Neurosurgical intervention		
		No	Yes	Total
Pre TBI	No	25 (61.0%)	16 (39.0%)	41
alcohol use	Yes	5 (83.3%)	1 (16.7%)	6
Total		30 (63.8%)	17 (36.2%)	47

There was no significant difference between patients with pre TBI alcohol use for neurosurgical intervention. 39.0% of patients who did not use alcohol pre TBI had neurosurgical intervention done and 16.7% of patients with pre TBI alcohol use had neurosurgical intervention. The relative odd of patients with no pre TBI alcohol use and neurosurgical intervention compared to patient with alcohol use pre TBI is 3.2.

p = 0.396

Odd ratio =3.2 95%CI (0.34-29.96)

q) Pre TBI alcohol use (prior to accident) and Modified Rankin Scale (Physical disability)
Table 21: Pre TBI alcohol use (prior to accident) and Modified Rankin Scale (physical disability)(Cross tabulation)

		Modified Rankin Scale		3
		Less than 3	3 or more	Total
Pre TBI	No	10 (24.4%)	31 (75.6%)	41
alcohol use	Yes	3 (50.0%)	3 (50.0%)	6
Total		13 (27.7%)	34 (72.3%)	47

There was no significant difference between patients with pre TBI alcohol use for physical disability using Modified Rankin Scale. 75.6% of patients who did not use alcohol pre TBI had score of 3 or more and 50% of patients who had pre TBI alcohol use scored 3 or more for the disability scale. The relative odd of patients with no pre TBI alcohol use and score 3 or more in disability scale compared to patient with alcohol use pre TBI is 3.1.

p = 0.326

Odds ratio = 3.1 95%CI (0.54-17.87)

5. CHAPTER 5: DISCUSSION

The present study attempted to investigate patterns of alcohol use among 60 patients with TBI. The patients were hospital based-individuals, either as out-patients attending the Rehabilitation Clinic, or Neurosurgical clinic or in the Neurosurgical ward or Emergency department. In 60 patients, approximately a quarter of the subjects (N=11, 23%) were from the Indian race, and predominantly males (N=39, 83%) and younger age group (<40 years).

The research found among the subjects presenting with TBI, 12.8% used alcohol before their injury. In subjects who had pre-TBI alcohol use were more likely not to use safety measures while driving. Additionally, in the subjects who had a history of alcohol use before TBI and in those with an AUDIT (hazardous, harmful drinking) scored of \geq 8 were more likely to re-use alcohol post TBI.

The literature review found alcohol use as a common factor among persons with brain injury, and its role to both the cause of injury and post-injury adjustment (Kreutzer et al., 1996a). The study showed the prevalence of alcohol use before the TBI is 12.8%. The prevalence of the pre-TBI alcohol use is lower compared to other studies done previously. The review found several papers stating 30% to 50% of all patients persisting with trauma were intoxicated at the time of injury (Tien et al., 2006; Salim et al., 2009a). Oscar-Berman (2000) strongly believed alcohol is a major risk factor for injury and mortality.

Corrigan (1995) earliest work revealed, in 55–66% of the subjects surveyed, there was a pre-TBI history of alcohol. Hibbard et al. (1998) using the DSM-IV, to assess axis I psychiatric disorders in 100 community-residing persons with TBI, found 40% of the sample met DSM-IV criteria for substance abuse or dependence before the injury. The other studies reported rates of alcohol use:

- 1. Bombardier et al. (2003) 50% 203 consecutive inpatients with recent TBI,
- 2. Dikmen et al. (1995) reported pre-injury alcohol abuse in 42% of the subjects.

71

In a systemic review paper, Parry-Jones et al. (2006) discovered an estimate of 37–51% of the subjects were intoxicated at the time of injury and in 36–51%, had pre-TBI history of using alcohol. Ponsford et al. (2007) in a study among 121 hospital inpatients with TBI, close to 32% of the TBI group and 29% of the controls were drinking at a hazardous level. Savola et al. (2005) studied a group of 345 patients presenting to the hospitals for trauma discovered on admission, 51% of the patients had alcohol in their blood. The study also revealed binge drinking was the predominant in 78% of the subject. Thus, occurrence of head injury increased sharply with increasing BAC, the commonest causes for such injuries from accidents, falls and assaults.

Salim et al. (2009a) in a review of 38 019 patients and using the National Trauma Data Bank of patients injured between 2000 and 2005, discovered 38% tested positive for alcohol. Salim et al. (2009a) concluded serum ethanol is independently associated with decreased mortality in the patients with moderate to severe head injuries.

In a study done for alcohol-related traffic injuries and fatalities in developing countries. The prevalence of the pre-TBI alcohol use using interview method range 9-16% (Odero et al., 1997). Dikmen et al. (1995) investigated and followed-up patients with TBI. Dikmen et al. (1995) discovered 42% of the subjects were intoxicated while in the ED. Their alcohol use and associated problems decreased following the TBI. However, alcohol usage raised again by one year after injury. Dikmen et al. (1995) concluded:

- 1. patients with more severe head injuries were likely to decrease their drinking more than did those with less severe head injuries.
- 2. blood alcohol levels in the ED were a good indicator of the magnitude of their preinjury alcohol problems.

Meanwhile, the prevalence of alcohol use post-TBI is 10.6 %. The prevalence of alcohol use post-TBI is lower than pre-injury alcohol use and correspond with other

studies. In a prospective study done by Kreutzer et al. (1996a). The study followed-up 87 patients periodically after their head injury. Kreutzer et al. (1996a) discovered heavy drinkers were likely to return to drinking even at their second follow-up visits. Ironically, their alcohol use patterns were similar to pre-injury, for the heavy to moderate drinkers. Interestingly, several authors have suggested the presence of mood disorders pre and post-injury along with other complications of the trauma may contribute to the return in drinking (Dikmen et al., 1995; Jorge et al., 2004; Coetzer, 2004; Jorge et al., 2005; Ahmed et al., 2017).

The prevalence of alcohol use post-TBI is also higher compared to the Malaysian epidemiology data on prevalence of current drinker among 18-years-old and above which is 8.4% (Institute for Public Health, 2015). However, as it is a cross-sectional-studies thus the study is unable to show an increasing pattern of alcohol usage with duration.

Association (significant findings)

1. History of alcohol use pre-TBI, AUDIT score and post TBI alcohol use

Hibbard et al. (1998) suggested drinking problems may not continue after TBI. However, another has suggested otherwise. This study found in patients who had a history of alcohol use before the TBI and whom scored ≥ 8 in the AUDIT (hazardous, harmful drinking) were significantly more likely to use alcohol post-TBI. This is similar to other studies done which showed that history of alcohol abuse is a risk factor to return to alcohol usage post-TBI (Kreutzer et al., 1996a; Kreutzer et al., 1996b; Bombardier et al., 2003; Corrigan and Karelina, 2015; Weil et al., 2016). Besides, only 25% of people achieve long-term functional independence following TBI (Faul et al., 2010; Ahmed et al., 2017).

The present work showed the importance of getting a history of alcohol use preand post-TBI. As the literature and result of study showed the use of alcohol use can complicate the presentation and rehabilitation of patients. Bernier and Hillary (2016) examining the trend of how alcohol- related TBI over the past two decades found the situation and association of alcohol- related TBI has not changed through the years. However, in the age sub-group, there seemed to be a decreasing trend of alcohol use and related injury in the 7-years and older cluster.

What is the significance of the knowledge? Many pieces of evidence suggested intoxicated drivers and involved in road traffic accidents had an additional history of driving under the influence or driving while impaired (Green et al., 2015; LaBrie et al., 2007; Rauch et al., 2010). (Green et al., 2015), Rauch et al. (2010) and LaBrie et al. (2007) are amount many authors who believe these individuals are more likely to be involved a subsequent alcohol-related accident or trauma. Kreutzer et al. (1990) recruited 87 brain injury persons from the outpatient clinic and followed them an average of 48 months post-injury. Kreutzer et al. (1990) discovered the number of moderate to heavy drinkers seemed to decline by two thirds from before to after TBI. Kreutzer et al. (1996b) in a cross-sectional study and following 322 young individuals post-TBI, a pattern of increasing alcohol consumption was noted. In the younger persons, a return to drinking was noted and with a higher level of consumption. Additionally, Kreutzer et al. (1996b) discovered lower consumption rates were found among subjects with higher levels of disability.

Thus, this present study and other literatures supports alcohol use pre-injury remains a major factor predicting alcohol usage post-injury.

2. Pre-TBI alcohol use and safety measures

The present study found in patients who had pre-TBI alcohol use were significantly more likely not to use safety measures such as seat-beat or helmet while driving (N=8, 19%). The finding is similar to another study done whereby it is found that patients with positive

blood alcohol concentration (BAC) were more likely not to use their safety belt compared to those with negative BAC (Stoduto et al., 1993; Fabbri et al., 2002; Friedman, 2014). Many studies indicate the adverse consequences of acute and chronic alcohol ingestion on psychological functions, safety behaviours, including safety measures while driving (Stoduto et al., 1993; Fabbri et al., 2002; Legrand et al., 2012; Friedman, 2014; Green et al., 2015; Blomberg et al., 2009). Blomberg et al. (2009) investigating 2,871 crashes along Long Beach and Fort Lauderdale, in the United States reported of the total crashes, 603 fled the scene of their crash. From the total individuals involved in the crash, the study managed to interview only 83% (the rest fled or refused) and 81.3% provided usable breath specimens.

The literature review has indicated alcohol affects the individuals' self-regulation, sensitivity and attitude thus consequences on their driving performance (Irwin et al., 2017; Mundt and Perrine, 1993; Zhao et al., 2014; Martin et al., 2013). Interesting, Zhao et al. (2014) studying the effects of alcohol on drivers and their driving performance disclosed it was easier to discriminate a driver with a higher BAC level from normal driving. The study identified the drivers' average speed, speed standard deviation, and lane position standard. The differences were significantly higher when the individuals were under the influence of alcohol. The risk of any accidents and fatal injury increases as the blood alcohol concentration levels surge (Taylor and Rehm, 2012; Taylor et al., 2010; Tien et al., 2006; Green et al., 2015). The evidence suggest drivers with blood alcohol level are likely to make poor driving decisions or engage in a physical altercation (Taylor et al., 2010). In fact, several authors such as Taylor et al. (2010) and Taylor and Rehm (2012) believed the consequences rapidly rises after a driver's BAC exceeds 50 mg/dL compared to unimpaired drivers.

Legrand et al. (2012) in a study comparing the prevalence of alcohol and illicit drugs in seriously injured drivers in Belgium (BE) and the Netherlands (NL), found alcohol was the predominant substance use in both countries. The study concluded alcohol is still the most prevalent substance used among the injured drivers. The authors as well concluded the alcohol use trend among injured drivers has been consistent since the last 15 years.

These studies indicated alcohol and/or drugs are frequently detected in injured drivers, more frequently than in the general driving population. In this study, there was no formal investigation or test done to assess the presence and amount of alcohol usage prior to the accident. The reason for no blood alcohol level documented in this study is because it is not the current practice in Malaysia to conduct blood alcohol concentration for TBI patients at the emergency department.

Association (no significant findings)

The study examined other factors, however, the factors did not show any significant association with alcohol use and in the traumatic brain injured patients surveyed.

1. Socio-demographic data and post-TBI alcohol use

This study found that socio-demographic data such as age, race, gender, education level, employment, marital status, salary, past medical history was not associated with post TBI alcohol use.

Oscar-Berman (2000) revealed there were more males drinking, and females and males metabolize alcohol differently. Moreover, the female brain functioning is more vulnerable to alcohol than the males. In Bernier and Hillary (2016)'s review of alcohol-related TBI over the past two decades, found males out-numbered females in alcohol related-TBI. The review as well showed that men, alcohol usage and between the ages of 18–30 have the highest incidence of TBI overall (65%).

In a local finding on alcohol usage among Malaysian population, it was found subjects between the ages of 20-29-years-old contribute to the highest percentage of current alcohol drinkers, which is 21.3% (Institute for Public Health, 2015). Similarly, Rehm et al. (2013) revealed males and at the ages of 15 to 44-years were likely to use alcohol and increased mortality for high-risk alcohol users. However, from this study, age and gender did not seem significantly affecting the post-TBI alcohol use. In a study by Horner et al. (2005) he found that younger age group was a risk factor for heavy alcohol use after the TBI episode. The similar findings Ahmed et al. (2017) found young adults ages 15–24, and males were more at risk for TBI.

Abdel-Aty and Abdelwahab (2000) as well showed the younger age group of 25– 34 age group experience the highest rate of alcohol or drug involvement in accidents. The rates decline with the increase in the age of the drivers (Abdel-Aty and Abdelwahab, 2000; Ahmed et al., 2017). Interestingly some investigators indicate gender and age plays a role in the effect of blood alcohol level and recovery from the TBI (Kaplan and Corrigan, 1992). Kaplan and Corrigan (1992) found females had lower blood alcohol levels than males but they experience longer length of post-traumatic amnesia and admission.

From this study, the majority of the patient with TBI were Malay which coincides with Malaysian demographic data. However, for it was found that patients of Indian race were much higher compared to Chinese patients. There was no association found between races and post-TBI alcohol use. Saroja and Kyaw (1993) did a survey on the prevalence of alcohol use among in-patients admitted to the General Hospital, Kuala Lumpur. All races including the Malay race were represented in 535 patients surveyed. The Malay race made up 20% i.e. the lowest race, while the Indians made up 36% of the group surveyed.

Studies done in Malaysia found drinkers from all race in Malaysia though individuals of Indian race were more likely to have dependence and abuse of alcohol (MI and AS, 2014; Saroja and Kyaw, 1993). Meanwhile, for the Malay race, even though alcohol is considered illegal in religious views, it was noted that among binge drinker was the highest in the Malay race (MI and AS, 2014).

For gender, the present study also did not find any significant association between gender and post-TBI alcohol use. Kreutzer et al. (1996a) showed despite the number of moderate to heavy drinkers declined from before to after TBI, a history of pre-injury heavy drinking and males are at greatest risk for long-term alcohol abuse post-injury.

Similarly Horner et al. (2005) disclosed the male gender is considered one of the risk factor for post-TBI alcohol use. Horner et al. (2005) interviewed 1606 adults oneyear post-injury. The study revealed risk factors of post-injury alcohol use include male gender, younger age, history of substance abuse prior to TBI, and being depression since the injury.

In the present study, there was also no significant association with the education level and post-TBI alcohol use. Even though, based on population study done in Malaysia, it was found that the highest number of current drinkers more than 18 years old were from tertiary education level. From this study, only 31% of the patients had tertiary education level and most of the patients were from secondary education level.

In the study, there was no significant in employment type and status with post TBI alcohol use. Most of the patients were unemployed which consisted of around 44% of the total patients. TBI is a major cause of long-term disability and this affects both the patients themselves, caregivers as well as the countries itself (Hyder et al., 2007). It was said to be the third largest contributor to the disability and global burden of disease after heart disease and depression according to WHO (Thornhill et al., 2000). According to a study, there was no accurate prediction of patient's ability to return to work as even mild TBI can cause lasting problem in sustained attention thus the need for more comprehensive rehabilitation programme tailored to each patient personally (Shames et al., 2007).

For marital status, this study found no significant in marital status and post TBI alcohol use. However, in another study done, noted that heavy drinkers are more likely to be single and infrequent/abstainer were mainly those who were married(Horner et al., 2005).

Thus, the study and review of the literature identified alcohol use as a common factor among persons with brain injury, relevant to both cause of injury and post-injury adjustment.

2. Other factors and post TBI alcohol use.

The other factors that possibly influence post TBI alcohol use such as family history of alcohol use, other substance usage, head injury duration, mental health of patients and cognitive problem were analyze as well however was found to be not significant in this study.

Family history of alcohol use was a well-known risk factor for a person to develop into problematic alcohol use in the future (Grant, 1998). However, in this study, there was no significant for family history of alcohol use and post TBI alcohol.

From this study, there was no significant between other substance usage (nicotine) and post TBI alcohol use. Post-TBI it was found that the usage of alcohol or other substance will increase overtime and the risk was mainly from past history of that particular substance use (Ponsford et al., 2007). Another possible reason is nicotine was the more preferred substance post TBI because it is more accepted among the culture in Malaysia generally.

Head injury duration was found to be one of the risk factor of increasing usage of post TBI alcohol in a few studies. It was found that patient will reduced intake of alcohol right after the head trauma but eventually the intake will increase overtime (Bombardier et al., 2003; Ponsford et al., 2007). However, in this study, there was no significant found between head injury duration and post TBI alcohol use.

In a study done following head injury for mental health disorder, it was found that 65% of patients had some psychiatry diagnosis mainly depression followed by anxiety disorder and then substance use disorder (Whelan-Goodinson et al., 2009). Another study noted a lower percentage of patient with mental illness post TBI of 21.7% (Deb et al., 1999) However, in this study no formal diagnostic test was done for patient but a screening test for mental well-being was conducted and showed 45% of the patients had mental health problems. There were however no significant findings between the mental health problem and post TBI alcohol use.

Alcohol intake pre TBI was found to be a contributing cause for cognitive impairment in TBI patient and the blood alcohol concentration was predictive of poorer delayed verbal memory and poorer visuospatial functioning (S. Tate et al., 1999). The influence of blood alcohol level is most prominent to cause cognitive impairment within the one month of the injury but may persist in some areas beyond the one month duration (Bombardier and Thurber, 1998). In this study, there was no significant between cognitive impairment and post TBI alcohol use.

LIMITATION, STRENGTH AND RECOMMENDATION

There were several limitations in this study. First and foremost is the small sample size collected from the study below the expected the numbers. The researcher initially started collecting the sample from a single site in UMMC which is Neurosurgical clinic and ward however when facing difficulties in getting the sample, more site was introduced half way into the research by getting permission to get samples from neuro-rehabilitation clinic UMMC and approval to collect in another urban hospital where the

researcher was posted which is Hospital Kuala Lumpur. Even though, more location for data collection was established, the approval took some time and restricted the duration of data collection in the new area. Also, to make things worse, all the clinics (neurorehabilitation UMMC, neurosurgical clinic UMMC, neurosurgical clinic HKL) for cases of head injury were placed on Monday and restricted the researcher on collecting the sample due to different location.

Apart from that, the researcher also faced difficulties to approach patients in a hectic clinic and occasionally some of the patients will be missed out as unable to wait for the interview sessions. Besides that, as the research topic involved alcohol which is considered slightly sensitive to our culture and especially for patients who suffered head injury, there were patients/ family members who were not keen to participate in the study.

The study uses convenient sampling method and depends on the availability of the researcher at that time thus may introduced selection bias. Convenient sampling is used to get as much samples as possible during the limited duration of data collection. The researcher tends to take more patients from certain location which were easier to get and thus the sample collected demographic may be skewed depending on the location of the sample taken. For this study, most of the sample taken were mainly from neurorehabilitation clinic in UMMC.

The questionnaires given to patient were mainly in Malay language as it is the national language of Malaysia thus more receptive for the patients. The average time taken to answer the questionnaires were around 20-30minutes. However, patients with TBI had some difficulties in concentration and attention, some also had some language barrier and not familiar with certain words in the questionnaire thus the researcher will clarify the questions again with the patients. Apart from that, as the main questions were about alcohol use, there may be some restriction in giving the true answer of certain

questions by the patients. Thus, the understanding of the questions and sensitivity of the questions may affect the outcome of the data in some way.

The other limitations of the research include difficulties in obtaining consent and consent validity especially in patients with traumatic brain injury in view of impairment in cognitive functions. For this study, no formal assessment tools were done prior to getting consent however in patients with gross difficulties to understand and comprehend, the consent was taken from guardians or family members of patients.

This study only uses GHQ-12 as screening for psychological problem affecting the patients with traumatic brain injury and it was unable to ascertain regarding certain psychiatric diagnosis or other social issues faced by the patients.

STRENGTH

Although TBI and alcohol use relationship had been studied extensively throughout the world however, there were still very limited local data available in Malaysia. This study will help established a foundation and data regarding our local scenario of the situation. Hopefully from the study, certain significant findings were able to be use as a guidance for further research on this issue.

RECOMMENDATION

There is much improvement needed for this study and could be recommended for future undertakings. The main issue that needed to overcome is to increase data collections and by increasing the sample size, the study is more robust and give a better finding which is statistically more representative of the population.

Regarding the difficulties to obtain consent and whether the consent will be valid as patients with traumatic brain injury are considered vulnerable group, thus for future research it is suggested to use an assessment tools to assess ability of patients to give consent prior to the be included in the study.

In future studies, possibly the research could proceed with prospective study which then can establish the causality link between the factors and the outcome. Apart from that, possibly to use other screening or diagnostic tools to ascertain regarding other psychological or psychosocial problems which may affect patients with traumatic brain injury such as personality changes, depression and others.

CONCLUSION

The present study supports the numerous literature on MVA related-TBI with a significant association of alcohol use. The results can add knowledge to alcohol use and traumatic injury. It is possible that interventions to increase public awareness about the danger of operating a vehicle while intoxicated. Additionally, stricter identification of people's driving state may contribute to the general safety of the population.

Alcohol use and TBI has been a complicated matter whereby both the factors can affect each other either pre-TBI or post-TBI and it does disrupt the subsequent management of the patients. From this study, we were able to see some associations that may contribute towards patients' alcohol use after TBI which were having history of alcohol use and harmful alcohol use before TBI. Both of these factors need to be monitored by treating physician and be used as a indicator of subsequent alcohol use in the future.

The study also gave an overview of the current situation of TBI patients in these 2 urban hospitals (UMMC and HKL). Besides the main outcome of the research regarding alcohol, it provides additional information regarding other substance use of patients post TBI such as nicotine, the mental health wellbeing of TBI patients and the cognitive impairment among the TBI patients.

References:

- Abdel-Aty MA and Abdelwahab HT. (2000) Exploring the relationship between alcohol and the driver characteristics in motor vehicle accidents. *Accident Analysis and Prevention* 32: 473-482.
- Abdelfatah A. (2016) Traffic fatality causes and trends in Malaysia. *Malaysia* Sustainable Cities Program, Working Paper Series.
- Ahmed S, Venigalla H, Mekala HM, et al. (2017) Traumatic brain injury and neuropsychiatric complications. *Indian Journal of Psychological Medicine* 39: 114-121.
- Alves WM, Colohan AR, O'leary TJ, et al. (1986) Understanding posttraumatic symptoms after minor head injury. *The Journal of Head Trauma Rehabilitation* 1: 1-12.
- Ameratunga S, Hijar M and Norton R. (2006) Road-traffic injuries: confronting disparities to address a global-health problem. *The Lancet* 367: 1533-1540.
- American Psychiatric Association. (2013) *Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5),* Arlington: American Psychiatric Publishing.
- Babor TF and Grant M. (1992) Project on Identification and management of alcoholrelated problems: Report on Phase II—a randomized clinical trial of brief interventions in primary health care. Geneva: World Health Organization, Program on Substance Abuse.
- Belanger HG, Curtiss G, Demery JA, et al. (2005) Factors moderating neuropsychological outcomes following mild traumatic brain injury: A metaanalysis. *Journal of the International Neuropsychological Society* 11: 215-227.
- Bernier RA and Hillary FG. (2016) Trends in alcohol use during moderate and severe traumatic brain injury: 18 years of neurotrauma in Pennsylvania. *Brain Injury* 30: 414-421.
- Bird MD, Choudhry MA, Molina PE, et al. (2009) Alcohol and trauma: A summary of the Satellite Symposium at the 30th Annual Meeting of the Shock Society. *Alcohol* 43: 247-252.
- Blomberg RD, Peck RC, Moskowitz H, et al. (2009) The long beach/fort lauderdale relative risk study. *Journal of Safety Research* 40: 285-292.
- Blugeot A, Rivat C, Bouvier E, et al. (2011) Vulnerability to depression: from brain neuroplasticity to identification of biomarkers. *Journal of Neuroscience* 31: 12889-12899.
- Bombardier CH, Rimmele CT and Zintel H. (2002) The magnitude and correlates of alcohol and drug use before traumatic brain injury. *Archives of Physical Medicine and Rehabilitation* 83: 1765-1773.
- Bombardier CH, Temkin NR, Machamer J, et al. (2003) The natural history of drinking and alcohol-related problems after traumatic brain injury. *Archives of physical medicine and rehabilitation* 84: 185-191.
- Bombardier CH and Thurber CA. (1998) Blood alcohol level and early cognitive status after traumatic brain injury. *Brain Injury* 12: 725-734.
- Borgaro SR, Prigatano GP, Kwasnica C, et al. (2003) Cognitive and affective sequelae in complicated and uncomplicated mild traumatic brain injury. *Brain Injury* 17: 189-198.
- Brennan JH, Bernard S, Cameron PA, et al. (2015) Ethanol and isolated traumatic brain injury. *Journal of Clinical Neuroscience* 22: 1375-1381.
- Carroll L, Cassidy JD, Peloso P, et al. (2004) Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine* 36: 84-105.

Centers for Disease Control and Prevention. (2017) *Traumatic brain injury & concussion*.

- Coetzer BR. (2004) Grief, self-awareness, and psychotherapy following brain injury. *Illness, Crisis and Loss* 12: 171-186.
- Connor J, Broad J, Rehm J, et al. (2005) The burden of death, disease, and disability due to alcohol in New Zealand. *The New Zealand Medical Journal (Online)* 118.
- Corrigan JD. (1995) Substance abuse as a mediating factor in outcome from traumatic brain injury. *Archives of Physical Medicine and Rehabilitation* 76: 302-309.
- Corrigan ZMWJD and Karelina K. (2015) Alcohol abuse after traumatic brain injury: Experimental. *Journal of Neuroscience Research* 30: 8285-8295.
- Deb S, Lyons I, Koutzoukis C, et al. (1999) Rate of psychiatric illness 1 year after traumatic brain injury. *American Journal of Psychiatry* 156: 374-378.
- Department of Transportation NHTSA. (2000) The economic impact of motor vehicle crashes Washington,DC.
- Dikmen SS, Machamer JE, Donovan DM, et al. (1995) Alcohol use before and after traumatic head injury. *Annals of Emergency Medicine* 26: 167-176.
- Eckardt MJ, File SE, Gessa GL, et al. (1998) Effects of moderate alcohol consumption on the central nervous system. *Alcoholism: Clinical and Experimental Research* 22: 998-1040.
- Fabbri A, Marchesini G, Morselli-Labate AM, et al. (2002) Positive blood alcohol concentration and road accidents. A prospective study in an Italian emergency department. *Emergency Medicine Journal* 19: 210-214.
- Fabian MJ and Proctor KG. (2002) Hemodynamic actions of acute ethanol after resuscitation from traumatic brain injury. *Journal of Trauma and Acute Care Surgery* 53: 864-875.
- Fann JR, Katon WJ, Uomoto JM, et al. (1995) Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *The American Journal of Psychiatry* 152: 14931499.
- Faul M, Xu L, ., Wald MM, et al. (2010) Traumatic brain injury in the United States. Atlanta, GA: National Center for injury Prevention and Control, Centers for disease Control and Prevention.
- Fenoglio P, Parel V and Kopp P. (1997) The social cost of alcohol, tobacco and illicit drugs in France, 1997. *European Addiction Research* 9: 18-28.
- Foster CA and Dissanaike SD. (2014) Prevalence and consequences of positive blood alcohol levels among patients injured at work. *Journal of Emergencies, Trauma, and Shock* 7: 268.
- Foster GR, Dunbar JA, Whittet D, et al. (1988) Contribution of alcohol to deaths in road traffic accidents in Tayside 1982-6. *British Medical Journal (Clinical research ed.)* 296(6634), p.1430.: 1430-1432.
- Friedman LS. (2014) Complications associated with blood alcohol concentration following injury. *Alcohol* 48: 391-400.
- Goldberg DP. (1972) The detection of psychiatric illness by questionnaire: A technique for the identification and assessment of non-psychotic illness: Oxford University Press.
- Gore FM, Bloem PJ, Patton GC, et al. (2011) Global burden of disease in young people aged 10–24 years: A systematic analysis. *The Lancet* 377: 2093-2102.
- Grant BF. (1998) The impact of a family history of alcoholism on the relationship between age at onset of alcohol use and DSM-IV alcohol dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. *Alcohol Research and Health* 22: 144.

- Green RS, Kureshi N and Erdogan M. (2015) Legal consequences for alcohol-impaired drivers injured in motor vehicle collisions: A systematic review. *Accident Analysis and Prevention* 80: 106-116.
- Guidot DM and Hart MC. (2005) Alcohol abuse and acute lung injury: epidemiology and pathophysiology of a recently recognized association. *Journal of Investigative Medicine* 53: 235-246.
- Gupta M, Menon GR, Devkar G, et al. (2015) Regulatory and road engineering interventions for preventing road traffic injuries and fatalities among vulnerable (non-motorised and motorised two-wheel) road users in low-and middle-income countries. *The Cochrane Library*.
- Gurney JG, Rivara FP, Mueller BA, et al. (1992) The effects of alcohol intoxication on the initial treatment and hospital course of patients with acute brain injury. *The Journal of Trauma* 33: 709-713.
- Harris JK, Godfrey HP, Partridge FM, et al. (2001) Caregiver depression following traumatic brain injury (TBI): A consequence of adverse effects on family members? *Brain Injury* 15: 223-238.
- Hibbard MR, Uysal S, Kepler K, et al. (1998) Axis I psychopathology in individuals with traumatic brain injury. *The Journal of Head Trauma Rehabilitation* 13: 24-39.
- Hoofien D, Gilboa A, Vakil E, et al. (2001) Traumatic brain injury (TBI) 10? 20 years later: A comprehensive outcome study of psychiatric symptomatology, cognitive abilities and psychosocial functioning. *Brain Injury* 15: 189-209.
- Horner MD, Ferguson PL, Selassie AW, et al. (2005) Patterns of alcohol use 1 year after traumatic brain injury: a population-based, epidemiological study. *Journal of the International Neuropsychological Society* 11: 322-330.
- Hughes BP, Newstead S, Anund A, et al. (2015) A review of models relevant to road safety. *Accident Analysis and Prevention* 74: 250-270.
- Hyder AA, Wunderlich CA, Puvanachandra P, et al. (2007) The impact of traumatic brain injuries: A global perspective. *NeuroRehabilitation* 22: 341-353.
- Institute for Public Health I. (2015) National Health and Morbidity Survey 2015: Noncommunicable diseases, risk factors and other health problems. Kuala Lumpur: Institute for Public Health, National Institutes of Health, Ministry of Health, Malaysia.
- Irwin C, Iudakhina E, Desbrow B, et al. (2017) Effects of acute alcohol consumption on measures of simulated driving: A systematic review and meta-analysis. *Accident Analysis & Prevention* 102: 248-266.
- Iverson GL. (2005) Outcome from mild traumatic brain injury. *Current Opinion in Psychiatry* 18: 301-317.
- Iverson GL. (2006) Complicated vs uncomplicated mild traumatic brain injury: acute neuropsychological outcome. *Brain Injury* 20: 1335-1344.
- Iverson GL, Lovell MR, Smith S, et al. (2000) Prevalence of abnormal CT-scans following mild head injury. *Brain Injury* 14: 1057–1061.
- Jamaluddin SF, Wahab MA, Mohamed FL, et al. (2009) National trauma database January to December 2007–second report. National Trauma Database and Clinical Research Centre, Ministry of Health.
- Jeng TC, Haspani MSM, Adnan JS, et al. (2008) Delayed traumatic intracranial haemorrhage and progressive traumatic brain injury in a major referral centre based in a developing country. *The Malaysian Journal of Medical Sciences* (*MJMS*) 15: 56-67.
- Jernigan DH and Indran SK. (1997) Alcohol use patterns, problems and policies in Malaysia. *Drug and Alcohol Review* 16: 401-409.

- Jones AW. (1991) Top ten defence challenges among drinking drivers in Sweden. *Medicine, Science and the Law* 31: 229-238.
- Jorge RE, Robinson RG, Moser D, et al. (2004) Major depression following traumatic brain injury. *Archives of General Psychiatry* 61: 42-50.
- Jorge RE, Starkstein SE, Arndt S, et al. (2005) Alcohol misuse and mood disorders following traumatic brain injury. *Archives of General Psychiatry* 62: 742-749.
- Jurkovich GJ, Rivara FP, Gurney JG, et al. (1993) The effect of acute alcohol intoxication and chronic alcohol abuse on outcome from trauma. *JAMA* 270: 51-56.
- Kaplan CP and Corrigan JD. (1992) Effect of blood alcohol level on recovery from severe closed head injury *Brain Injury* 6: 337-349.
- Kasner SE. (2006) Clinical interpretation and use of stroke scales. *The Lancet Neurology* 5: 603-612.
- Katada R, Nishitani Y, Honmou O, et al. (2009) Prior ethanol injection promotes brain edema after traumatic brain injury. *Journal of Neurotrauma* 26: 2015-2025.
- Kays JL, Hurley RA and Taber KH. (2012) The dynamic brain: Neuroplasticity and mental health. *The Journal of Neuropsychiatry and Clinical Neurosciences* 24: 118-124.
- Keyser-Marcus LA, Bricout JC, Wehman P, et al. (2002) Acute predictors of return to employment after traumatic brain injury: A longitudinal follow-up. *Archives of Physical Medicine and Rehabilitation* 83: 635-641.
- Kim JH, Lee S, Chow J, et al. (2006) Prevalence and the factors associated with binge drinking, alcohol abuse, and alcohol dependence: A population-based study of Chinese adults in Hong Kong. *Alcohol and Alcoholism* 43: 360-370.
- Koponen S, Taiminen T, Portin R, et al. (2002) Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. *American Journal of Psychiatry* 159: 1315-1321.
- Kraus JF, Morgenstern H, Fife D, et al. (1989) Blood alcohol tests, prevalence of involvement, and outcomes following brain injury. *American Journal of Public Health* 79: 294-299.
- Kreutzer JS, Doherty KR, Harris JA, et al. (1990) Alcohol use among persons with traumatic brain injury. *The Journal of Head Trauma Rehabilitation* 5: 9-20.
- Kreutzer JS, Witol AD and Marwitz JH. (1996a) Alcohol and drug use among young persons with traumatic brain injury. *Journal of Learning Disabilities* 29: 643-651.
- Kreutzer JS, Wltol AD, Sander AM, et al. (1996b) A prospective longitudinal multicenter analysis of alcohol use patterns among persons with traumatic brain injury. *The Journal of Head Trauma Rehabilitation* 11: 58-69.
- Krug EG, Sharma GK and Lozano R. (2000) The global burden of injuries. *American Journal of Public Health* 90: 523-526.
- Kulanthayan S, Phang WK and Hayati KS. (2007) Traffic light violation among motorists in Malaysia. *IATSS Research* 31: 67-73.
- Kumar A, Lalwani S, Agrawal D, et al. (2008) Fatal road traffic accidents and their relationship with head injuries: An epidemiological survey of five years. *Indian Journal of Neurotrauma* 5: 63-67.
- LaBrie RA, Kidman RC, Albanese M, et al. (2007) Criminality and continued DUI offense: Criminal typologies and recidivism among repeat offenders. *Behavioral Sciences and the Law* 25: 603-614.
- Langlois JA, Rutland-Brown W and Wald MM. (2006) The epidemiology and impact of traumatic brain injury: A brief overview. *The Journal of Head Trauma Rehabilitation* 21: 375-378.

- Legrand SA, Houwing S, Hagenzieker M, et al. (2012) Prevalence of alcohol and other psychoactive substances in injured drivers: Comparison between Belgium and the Netherlands. *Forensic Science International* 220: 224-231.
- Levin HS, Mattis S, Ruff RM, et al. (1987) Neurobehavioral outcome following minor head injury: A three-center study. *Journal of Neurosurgery* 66: 234-243.
- Li G, Keyl PM, Smith GS, et al. (1997) Alcohol and injury severity: Reappraisal of the continuing controversy. *Journal of Trauma and Acute Care Surgery* 42: 562-569.
- Liguori A. (2009) Simulator studies of drug-induced driving impairment. *Drugs, driving and traffic safety*: 75–82.
- Livingston DH, Loder PA, Koziol J, et al. (1991) The use of CT scanning to triage patients requiring admission following minimal head injury. *Journal of Trauma* 31: 487–489.
- Loheswaran G, Barr MS, Rajji TK, et al. (2016) Brain stimulation in alcohol use disorders: Investigational and therapeutic tools. *Cognitive Neuroscience and Neuroimaging* 1: 5-13.
- Lopez AD and Murray CC. (1998) The global burden of disease, 1990–2020. *Nature Medicine* 4: 1241.
- Luna GK, Maier RV, Sowder L, et al. (1984) The influence of ethanol intoxication on outcome of injured motorcyclists. *The Journal of Trauma* 24: 695-700.
- Marsh NV, Ludbrook MR and Gaffaney LC. (2016) Cognitive functioning following traumatic brain injury: A five-year follow-up. *NeuroRehabilitation* 38: 71-78.
- Martin TL, Solbeck PA, Mayers DJ, et al. (2013) A review of alcohol-impaired driving: The role of blood alcohol concentration and complexity of the driving task. *Journal of Forensic Sciences* 58: 1238-1250.
- Mateer CA and Sira CS. (2006) Cognitive and emotional consequences of TBI: Intervention strategies for vocational rehabilitation. *NeuroRehabilitation* 21: 315-326.
- Mayou R, Bryant B and Duthie R. (1993) Psychiatric consequences of road traffic accidents. *BMJ* 307: 647-651.
- McIntosh C and Chick J. (2004) Alcohol and the nervous system. *Neurosurgery and Psychiatry* 75: iii16-iii21.
- MI MF and AS AN. (2014) Alcohol harm in Malaysia: always the right time to discuss. *Malaysian Journal of Psychiatry* 23: 101-104.
- Ministry of Health Malaysia. (2011) National trauma database January 2009 to December 2009 Fourth Report In: Jamaluddin SF, Wahab MA, Wahab MYA, et al. (eds). Kuala Lumpur: National Trauma Database and Clinical Research Centre, Ministry of Health.
- Mittenberg W, Tremont G, Zielinski RE, et al. (1996) Cognitive-behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology* 11: 139-145.
- Mohamed N, Batcha WA, Abdullah NK, et al. (2012) Alcohol and drug use among fatally injured drivers in urban area of Kuala Lumpur (No. MRR 02/2012).
- Mohseni S, Bellander BM, Riddez L, et al. (2016) Positive blood alcohol level in severe traumatic brain injury is associated with better long-term functional outcome. *Brain Injury* 30: 1256-1260.
- Mokdad AH, Marks JS, Stroup DF, et al. (2004) Actual causes of death in the United States, 2000. 291 10: 1238-1245.
- Molina PE. (2005) Neurobiology of the stress response: contribution of the sympathetic nervous system to the neuroimmune axis in traumatic injury. *Shock* 24: .3-10.

- Mundt JC and Perrine MW. (1993) Measured BAC vs. estimated BAC, rated intoxication, and driving ability. *International Conference on Alcohol, Drugs and Traffic Safety*.
- Nantulya VM and Reich MR. (2002) The neglected epidemic: road traffic injuries in developing countries. *BMJ* 324: 1139-1141.
- Odero W, Garner P and Zwi A. (1997) Road traffic injuries in developing countries: a comprehensive review of epidemiological studies. *Tropical Medicine and International Health* 2: 445-460.
- Ogden EJD and Moskowitz H. (2004) Effects of alcohol and other drugs on driver performance. *Traffic Injury Prevention* 5: 185–198.
- Olver JH, Ponsford JL and Curran CA. (1996) Outcome following traumatic brain injury: a comparison between 2 and 5 years after injury. *Brain Injury* 10: 841-848.
- Opreanu RC, Kuhn D and Basson MD. (2010) Influence of alcohol on mortality in traumatic brain injury. *Journal of the American College of Surgeons* 210: 997-1007.
- Organization WH and Unit WHOMoSA. (2014) *Global status report on alcohol and health, 2014*: World Health Organization.
- Oscar-Berman M. (2000) Neuropsychological vulnerabilities in chronic alcoholism. *Review of NIAAA's Neuroscience and Behavioral Research Portfolio* 34: 149-158.
- Oscar-Berman M and Marinkovic K. (2003) Alcoholism and the brain: An overview. *Alcohol Research and Health* 27: 125-133.
- Parry-Jones BL, Vaughan FL and Miles Cox W. (2006) Traumatic brain injury and substance misuse: A systematic review of prevalence and outcomes research *Neuropsychological Rehabilitation* 16: 537-560.
- Parry CD, Patra J and Rehm J. (2011) Alcohol consumption and non-communicable diseases: Epidemiology and policy implications. *Addiction* 106: 1718-1724.
- Peden M, Scurfield R, Sleet D, et al. (2004) World report on road traffic injury prevention. Geneva: World Health Organization.
- Phelan H, Stahls P, Hunt J, et al. (2002) Impact of alcohol intoxication on hemodynamic, metabolic, and cytokine responses to hemorrhagic shock. *Journal* of Trauma and Acute Care Surgery 52: 675-682.
- Phillips DP and Brewer KM. (2011) The relationship between serious injury and blood alcohol concentration (BAC) in fatal motor vehicle accidents: BAC= 0.01% is associated with significantly more dangerous accidents than BAC= 0.00%. *Addiction* 106: 1614-1622.
- Planas-Ballvé A, Grau-López L, Morillas RM, et al. (2017) Neurological manifestations of excessive alcohol consumption. *Gastroenterología y Hepatología (English Edition)* 40: 709-717.
- Ponsford J, Whelan-Goodinson R and Bahar-Fuchs A. (2007) Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury* 21: 1385-1392.
- Ponsford J, Willmott C, Rothwell A, et al. (2000) Factors influencing outcome following mild traumatic brain injury in adults. *Journal of the International Neuropsychological Society* 6: 568-579.
- Rabinowitz AR and Levin HS. (2014) Cognitive sequelae of traumatic brain injury. *The Psychiatric Clinics of North America* 37.
- Rahman NHN, Baharuddin KA and Mohamad SMS. (2015) Burden of motorcyclerelated injury in Malaysia. *International Journal of Emergency Medicine* 8: 71.

- Raj R, Skrifvars MB, Kivisaari R, et al. (2015) Acute alcohol intoxication and longterm outcome in patients with traumatic brain injury. *Journal of Neurotrauma* 32: 95-100.
- Ramli R, Oxley J, Noor FM, et al. (2014) Fatal injuries among motorcyclists in Klang Valley, Malaysia. *Journal of Forensic and Legal Medicine* 26: 39-45.
- Rauch WJ, Zador PL, Ahlin EM, et al. (2010) Risk of alcohol-impaired driving recidivism among first offenders and multiple offenders. *American Journal of Public Health* 100: 919-924.
- Rehm J. (2011) The risks associated with alcohol use and alcoholism. *Alcohol Research and Health* 34: 135-143.
- Rehm J, Ashley MJ, Room R, et al. (1996) On the emerging paradigm of drinking patterns and their social and health consequences. *Addiction* 91: 1615-1621.
- Rehm J, Dan Chisholm D, Room R, et al. (2006) Epidemiology of alcohol use and alcohol-related disease conditions. *Disease control priorities in developing countries*. World Bank Publications, 887-906.
- Rehm J, Gnam W, Popova S, et al. (2007) The costs of alcohol, illegal drugs, and tobacco in Canada, 2002. *Journal of Studies on Alcohol And Drugs* 68: 886-895.
- Rehm J, Mathers C, Popova S, et al. (2009) Global burden of disease and injury and economic cost attributable to alcohol use and alcohol use disorders. *Lancet* 373: 2223–22233.
- Rehm J, Room R, Graham K, et al. (2003) The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: An overview. *Addiction* 98: 1209-1228.
- Rehm J, Room R, Monteiro M, et al. (2013) Alcohol as a risk factor for global burden of disease. *European Addiction Research* 9: 157-164.
- Rehm J, Room R and Taylor B. (2008) Method for moderation: measuring lifetime risk of alcohol-attributable mortality as a basis for drinking guidelines. *International Journal of Methods in Psychiatric Research* 17: 141-151.
- Rehm J, Shield KD, Rehm MX, et al. (2012) Alcohol consumption, alcohol dependence and attributable burden of disease in Europe. *Centre for Addiction and Mental Health.* 16: 11-19.
- Roy D, Koliatsos V, Vaishnavi S, et al. (2018) Risk factors for new-onset depression after first-time traumatic brain injury. *Psychosomatics* 59: 47-57.
- Royal Malaysian Police. (2007) Statistical report road accidents Malaysia: 2006. Traffic Branch, Bukit Aman, Kuala Lumpur.
- Ruffolo CF, Friedland JF, Dawson DR, et al. (1999) Mild traumatic brain injury from motor vehicle accidents: factors associated with return to work. *Archives of Physical Medicine and Rehabilitation* 80: 392-398.
- S. Tate P, David MF, Charles HB, et al. (1999) Traumatic brain injury: influence of blood alcohol level on post-acute cognitive function. *Brain Injury* 13: 767-784.
- Sabariah FJ, Ramesh N and Mahathar AW. (2008) National Trauma Database (NTrD)– improving trauma care: First year report. *Medical Journal of Malaysia* 63: 45-49.
- Salim A, Ley EJ, Cryer HG, et al. (2009a) Positive serum ethanol level and mortality in moderate to severe traumatic brain injury. *Archives of Surgery* 144: 865-871.
- Salim A, Teixeira P, Ley EJ, et al. (2009b) Serum ethanol levels: predictor of survival after severe traumatic brain injury. *Journal of Trauma and Acute Care Surgery* 67: 697-703.
- Samokhvalov AV, Popova S, Room R, et al. (2010) Disability associated with alcohol abuse and dependence. *Alcoholism: Clinical and Experimental Research* 34: 1871-1878.

- Saroja K and Kyaw O. (1993) Pattern of alcoholism in the General Hospital, Kuala Lumpur. *The Medical Journal of Malaysia* 48: 129-133.
- Saunders JB, Aasland OG, Babor TF, et al. (1993) Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. *Addiction* 88: 791-804.
- Savola O, Niemelä O and Hillbom M. (2005) Alcohol intake and the pattern of trauma in young adults and working aged people admitted after trauma. *Alcohol and Alcoholism* 40: 269-273.
- Schaefer PW, Huisman TA, Sorensen AG, et al. (2004) Diffusion-weighted MR imaging in closed head injury: high correlation with initial glasgow coma scale score and score on modified Rankin scale at discharge. *Radiology* 233: 58-66.
- Schretlen DJ and Shapiro AM. (2003) A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry* 15: .341-349.
- Sethi D, Aljunid S, Saperi SB, et al. (2002) Comparison of the effectiveness of major trauma services provided by tertiary and secondary hospitals in Malaysia. *Journal of Trauma and Acute Care Surgery* 53: 508-516.
- Shames J, Treger I, Ring H, et al. (2007) Return to work following traumatic brain injury: trends and challenges. *Disability and rehabilitation* 29: 1387-1395.
- Shield KD, Parry C and Rehm J. (2014) Chronic diseases and conditions related to alcohol use. *Alcohol Research: Current Reviews* 35: 155-171.
- Shield KD, Taylor B, Kehoe T, et al. (2012) Mortality and potential years of life lost attributable to alcohol consumption in Canada in 2005. *BMC Public Health* 21: 91.
- Skandsen T, Finnanger TG, Andersson S, et al. (2010) Cognitive impairment 3 months after moderate and severe traumatic brain injury: A prospective follow-up study. *Archives of Physical Medicine and Rehabilitation* 91: 1904-1913.
- Spitz G, Ponsford JL, Rudzki D, et al. (2012) Association between cognitive performance and functional outcome following traumatic brain injury: A longitudinal multilevel examination *Neuropsychology* 26: 604-612.
- Stoduto G, Vingilis E, Kapur BM, et al. (1993) Alcohol and drug use among motor vehicle collision victims admitted to a regional trauma unit: demographic, injury, and crash characteristics. *Accident Analysis & Prevention* 25: 411-420.
- Sulter G, Steen C and De Keyser J. (1999) Use of the Barthel index and modified Rankin scale in acute stroke trials. *Stroke* 30: 1538-1541.
- Taylor B, Irving HM, Kanteres F, et al. (2010) The more you drink, the harder you fall: a systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. *Drug and Alcohol Dependence* 110: 108-116.
- Taylor B and Rehm J. (2012) The relationship between alcohol consumption and fatal motor vehicle injury: High risk at low alcohol levels. *Alcoholism: Clinical and Experimental Research* 36: 1827-1834.
- Taylor B, Rehm J, Room R, et al. (2008) Determination of lifetime injury mortality risk in Canada in 2002 by drinking amount per occasion and number of occasions. *American Journal of Epidemiology* 168: 1119-1125.
- Thornhill S, Teasdale GM, Murray GD, et al. (2000) Disability in young people and adults one year after head injury: prospective cohort study. *Bmj* 320: 1631-1635.
- Tien HC, Tremblay LN, Rizoli SB, et al. (2006) Association between alcohol and mortality in patients with severe traumatic head injury. *Archives of Surgery* 141: 1185-1191.

- Van Dyke N and Fillmore MT. (2014) Alcohol effects on simulated driving performance and self-perceptions of impairment in DUI offenders. *Experimental and Clinical Psychopharmacology* 22: 484.
- Vanderploeg RD, Curtiss G and Belanger HG. (2005) Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society* 11: 228-236.
- Volkow ND, Fowler JS and Wang GJ. (2003) The addicted human brain: insights from imaging studies. *The Journal of Clinical Investigation* 111: 1444-1451.
- von Heymann C, Langenkamp J, Dubisz N, et al. (2002) Posttraumatic immune modulation in chronic alcoholics is associated with multiple organ dysfunction syndrome. *Journal of Trauma and Acute Care Surgery* 52: 95-103.
- Watt K, Purdie DM, Roche AM, et al. (2004) Risk of injury from acute alcohol consumption and the influence of confounders. *Addiction* 99: 1262-1273.
- Weil ZM, Corrigan JD and Karelina K. (2016) Alcohol abuse after traumatic brain injury: Experimental and clinical evidence. *Neuroscience & Biobehavioral Reviews* 62: 89-99.
- Whelan-Goodinson R, Ponsford J, Johnston L, et al. (2009) Psychiatric disorders following traumatic brain injury: their nature and frequency. *The Journal of head trauma rehabilitation* 24: 324-332.
- White AM. (2003) What happened? Alcohol, memory blackouts, and the brain. *Alcohol Research and Health* 27: 186-196.
- Wong GKC, Lam S, Ngai K, et al. (2012) Evaluation of cognitive impairment by the Montreal cognitive assessment in patients with aneurysmal subarachnoid haemorrhage: prevalence, risk factors and correlations with 3 month outcomes. J Neurol Neurosurg Psychiatry 83: 1112-1117.
- World Health Organisation. (2002) The world health report 2002: reducing risks, promoting healthy life. Geneva: World Health Organization.
- World Health Organisation. (2014) Global status report on alcohol and health 2014. Global Status Report on Alcohol. World Health Organization
- Management of Substance Abuse Unit, World Health Organization 1–392.
- World Health Organization. (1992) The ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines, Geneva, Switzerland.
- World Health Organization. (2004) Global status report on alcohol 2004. In: Abuse DoMHaS (ed). Geneva, Switzerland: World Health Organization.
- World Health Organization. (2007) Alcohol and injury in emergency departments: Summary of the report from the WHO Collaborative Study on Alcohol and Injuries. Department of Mental Health and Substance Abuse Department of Injuries and Violence Prevention.
- World Health Organization. (2013) Pedestrian safety a road safety manual for decison makers and practitioners. Geneva: World Health Organization.
- Yee A, Adlan AS, Rashid RR, et al. (2015) Validation of the alcohol use disorders identification test (AUDIT)–Bahasa Malaysia version among a group of alcohol users. *Journal of Substance Use* 20: 229-233.
- Yue JK, Ngwenya LB, Upadhyayula PS, et al. (2017) Emergency department blood alcohol level associates with injury factors and six-month outcome after uncomplicated mild traumatic brain injury. *Journal of Clinical Neuroscience* 45: 293-298.
- Yusoff MSB, Rahim AFA and Jamil M. THE SENSITIVITY, SPECIFICITY AND RELIABILITY OF THE MALAY VERSION 12-ITEMS GENERAL HEALTH (GHQ-12) IN QUESTIONNAIRE DETECTING DISTRESSED MEDICAL STUDENTS.

- Zador PL, Krawchuk SA and Voas RB. (2000) Alcohol-related relative risk of driver fatalities and driver involvement in fatal crashes in relation to driver age and gender: An update using 1996 data *Journal of Studies on Alcohol* 61: 387-395.
- Zhao X, Zhang X and Rong J. (2014) Study of the effects of alcohol on drivers and driving performance on straight road. *Mathematical Problems in Engineering*.
- Zink BJ and Feustel PJ. (1995) Effects of ethanol on respiratory function in traumatic brain injury. *Effects of ethanol on respiratory function in traumatic brain injury* 82: 822-828.
- Zink BJ, Stern SA, Wang X, et al. (1998) Effects of ethanol in an experimental model of combined traumatic brain injury and hemorrhagic shock. *Academic Emergency Medicine* 5: 9-17.
- Zink BJ, Walsh RF and FeusteL PJ. (1993) Effects of ethanol in traumatic brain injury. *Journal of Neurotrauma* 10: 275-286.

93