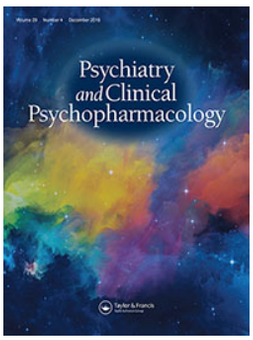

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BRIEF REPORT

Distinctive clinical correlates of hazardous drinking

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ABSTRACT

The present study was conducted to identify clinical correlates of hazardous drinking (HD). The data were derived from the Korean Research for Development of Alcohol Addiction Diagnosis and Assessment System. Variable measurement were personal characteristics, lifetime alcohol use history, Motivational Structure Questionnaire for alcoholics, Alcohol Outcome Expectancies Scale, and Alcohol Dependence Scale. Behavioural, psychiatric, and psychological factors were evaluated by responses to the Rosenberg Self Esteem Scale, Jung Self Rating Depression Scale, Barratt Impulsiveness Scale-11, State Traits Anxiety Inventory, and State Trait Anger Expression Inventory. The valid sample comprised 295 male drinkers, 89 subjects (30.2%) were classified as HD, and 209 (69.8%) were in the non-HD (NHD) group by NIAAA criteria. The results of binary logistic analysis showed that age at the first blackout, coping, and social motives for alcohol use, and non-planning impulsiveness increased the likelihood of HD net of each other's effects, and the final model explained 29.6% (Negelkerke R^2) of the variation in HD.

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KEYWORDS
Clinical correlates; coping motives for alcohol use; hazardous drinking; first black out; non-planning impulsiveness; social motives for alcohol use

Introduction

Hazardous drinking (HD) is an alcohol use pattern with a risk of harmful consequences related to the alcohol consumption. The National Institute of Alcohol Abuse and Alcoholism (NIAAA) defines it as consuming more than 21 standard drinking (SD) per week or 7 SD per day for men, and 14 SD per week or 5 SD per day for women [1]. This measure reflects the level of ethanol intake at which individual's psychomotor and cognitive functions are significantly impaired. It also represents a drinking pattern linked to increased risks of alcohol use disorder (AUD) and many types of alcohol related harm [2]. HD is considered to be the most influential factor for the occurrence of AUD and increase the legal and socioeconomic burdens caused by alcohol use.

The HD etiology, which encompasses a variety of behavioural, environmental, psychiatric, psychological and physiological factors, might be genetically predisposed. Although genetics play an important role to the development of HD, while the other factors are as important as genetics [3]. Recently, some candidate genes that might be associated with alcohol consumption were identified through genome wide association studies. However, genetics offers only limited exploration, and, to be applied, further investigations on single nucleotide polymorphisms and other genetic variants are needed [3]. There also are a number of limitations to identifying and using genetic information associated with HD in the primary health care system.

Recent investigations of etiological factors, including comorbid psychiatric and psychological conditions, indicate a high prevalence of HD. Lifetime drinking history including the age of first alcohol use and first blackout experience were found to predict incidence, maintenance and severity of alcohol related problem during adulthood [4]. Negative psychiatric conditions such as depression, anxiety, and aggression and personality trait such as impulsiveness also significantly related to higher alcohol consumption and HD [5]. Expectation and motivation for alcohol use also were closely related to HD [6], which was exacerbated by negative psychological conditions [7]. Environmental factors, such as family, neighbourhood, and significant other's heavy alcohol consumption also increase the risk of HD [8].

Expect for genetics, various non-genetic etiological factors might influence the development of HD. Identifying the influential clinical factors for the development of HD might be much more useful for screening and interventioning HD in primary health system. The present study was conducted to identify clinical correlates of HD. The data were derived from the Korean Research for Development of Alcohol Addiction Diagnosis and Assessment System (RDADAS). RDADAS was conducted to develop an effective

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diagnostic and intervention system based on the actual situations of Korean alcohol users for implementing it in mobile application. The Study was conducted in 13 hospitals (7 general hospitals and 6 psychiatric hospitals). The subjects were recruited through public advertising in January 2016 through November 2017. The data comprise a variety of information that might be relevant to alcohol use among Korean adult male.

Material and methods

Subjects

This study used data drawn from RDADAS in Korea. In this study, inclusion criteria were: (1) male (2) alcohol drinker, and (3) age ≥18 years and ≥60 years old. Our exclusion criteria were respondents that: (1) were diagnosed as or being treated for AUD, (2) had mental disorder (e.g. mental impairment, ongoing memory disorders, or dementia), (3) had physical disease (e.g. cirrhosis) or head injuries, (4) did not understand the survey contents. The valid sample comprised of 295 subjects.

Screening hazardous drinking with drinking record

All of subjects recorded their amounts of alcohol consumption for every drinking instance for three months after participating in this study. Recordings were based on an SD, and the meaning of "SD" was educated to all of the subjects when they agreed to participate in this study. HD was determined by drinking record, and when the subjects found the SD was not clear, the participants recorded the type and amount of alcohol, and computed the SD using the following: alcohol consumption = amount of alcohol (cc) X alcoholic contents (%) X specific gravity of alcohol (0.8). The NIAAA criteria were applied to indicate HD (consuming more than 21 SD per week or 7 SD per day for men) [1].

Variable measurement

Data in the subjects' personal characteristics were collected regarding age, marital status, educational attainment, occupation, co-residence, and presence of others' HD. The "others" were defined as the immediate family members or significant others, and the "significant others" were defined as non-family persons with whom the subject spent the most time or those who had the most influence on the persons. Lifetime alcohol use history, such as age at first drinking, first HD, and blackout were collected. The drinking patterns and amount were evaluated using Drinking record and the Alcohol Use Disorder Identification Test (AUDIT) [9].

The factors associated with drinking were evaluated by responses to the Motivational Structure Questionnaire for alcoholics (MCQ-A) [10], Alcohol Outcome Expectancies Scale (AOES) [11], and Alcohol Dependence Scale (ADS) [12]. Behavioural, psychiatric, and psychological factors were evaluated by responses to the Rosenberg Self Esteem Scale (SES) [13], Jung Self rating Depression Scale (SDS) [14], Barratt Impulsiveness Scale-11 (BIS) [15], State Trait Anxiety Inventory (STAI) [16], and State Trait Anger Expression Inventory (STAXI) [17]. To select scales, the emphasis was on measuring the trait rather than the specific state. All of the scales had been previously translated into Korean, and their validity and reliability have been confirmed in Korean populations.

Statistical analysis

Personal and clinical characteristics and the assessment scales' scores were compared between male drinkers categorized as those with and those without HD. Independent *t*-tests assessed statistically significant differences between those group regarding the means of the continuous variables, and the Chi-squared for contingency test ($\chi 2$) was used to assess statistically significant differences in categorical variables. A binary logistic regression analysis was performed to estimate the influence of predictive factors on the likelihood of HD, in this analysis, the dependent variable was HD, and the non-HD group was the reference category. The clinical variable that was statistically significant were tested as covariates. Goodness of fit indices were used to determine and validate the final model. The Statistical significance cut-off value was set at $P < .05$ (two-tailed test) for all tests. All statistical analyses were performed using SPSS 18.0 for Windows (SPSS Inc., Chicago, IL, USA).

Ethnical consideration

All of the subjects voluntarily agreed to participate in the survey and written informed consent was obtained after the study's purpose and methodology were explained to them. This study was conducted after receiving approval from the Institutional Review Board of Bugok National Hospital (BNH IRB No. 5-018). Private information was coded symbolized, and limited to use for purposes other than RDADAS.

Result

Differences in personal characteristics by HD

Table 1 shown that 89 subjects (30.2%) were classified as HD, and 209 (69.8%) were in the non- HD (NHD) group. Mean age and education attainment in the sample were 35.6 and 13.2 years, respectively. The employed rate was 89%, and 75% of the subjects lived with someone else. There were no significant differences between the two groups. Regarding employment

Table 1. Distribution of variable in the sample ($n = 295$) and by hazardous drinking subgroup.

	Total sample ($N = 295$)	Hazardous drinking ($N = 86$)	Non hazardous drinking ($N = 209$)	*Coefficients*	*p-Value*
Age, years, mean (SD)	35.6 (11.3)	37.2(13.4)	34.9 (10.8)	$t = -1.245$	0.218
Marital status, *n* (%)				$\chi^2 = 5.876$	0.053
Married	180 (61.0)	65 (75.5)	115(55.0)		
Unmarried	91 (30.8)	15 (17.4)	76 (36.4)		
Separation	24 (8.1)	6 (7.0)	18 (8.6)		
Employment, *n* (%)				$\chi^2 = 0.101$	0.752
Employed	265 (89.8)	74 (86.0)	191 (91.3)		
Unemployed	30 (10.2)	12 (14.0)	18 (8.7)		
Co-residence, *n* (%)				$\chi^2 = 0.001$	0.982
Yes	222 (75.2)	65 (75.5)	157 (75.1)		
No	73 (24.8)	21 (24.5)	52 (24.9)		
Education, years, mean (SD)	13.2(2.1)	12.3(2.4)	13.5(1.9)	$t = 1.597$	0.116
First drinking, years, mean (SD)	16.8 (2.5)	16.6 (2.1)	17.1 (2.5)	$t = -0.862$	0.390
First HD, years, mean (SD)	18.9 (2.6)	17.6 (2.5)	19.4 (2.6)	$t = -2.312$	0.024*
First blackout, years, mean (SD)	21.3(3.9)	19.3 (3.4)	22.0 (3.9)	$t = -2.498$	0.015*
Other's HD, *n* (%)					
Family member	91 (30.8)	25 (29.1)	63 (30.1)	$\chi^2 = 0.042$	0.838
Significant other	118 (40.0)	57 (66.3)	61 (29.1)	$\chi^2 = 4.810$	0.028*

Note: HD: Hazardous drinking; SD: standard deviation.
*$p < .05$.

or co-residence. The NHD group was more likely than HD group to be unmarried, which was borderline significant ($p = .053$). Ages at first HD, and first blackout were significantly younger for the HD than the NHD group HD ($p = .024$ and $p = .015$, respectively). Age at first drink was younger in the HD than the NHD group, but the difference was not statistically significant. There was no significant difference between the groups regarding a family member with HD, but a significant other with HD was significantly more likely for the HD than the NHD group ($p = .028$).

Differences in assessment scales' scores by HD

Table 2 shows that AUDIT and ADS scores were significantly higher in the HD than the NHD group ($p < .001$ and $p = .008$, respectively). Motives for alcohol use, enhancement, coping, and social motives also were significantly higher in the HD than the NHD group ($p = .011$, $p = .009$, and $p = .015$, respectively), but there was no statistically significant difference in conformity motives. The AOES scores were not significantly different between the two groups, but negative expectations related to alcohol use were lower in the HD group. Regarding impulsiveness, non-planning and motor impulsiveness were significantly higher in the HD ($p < .01$ and $p = .045$, respectively), but there was no significant difference in attentional impulsiveness.

Logistic regression analytical results

The preliminary logistic regression model found that a significant other's HD, age at first HD, age at first blackout, enhancement, coping, social motives for alcohol use, non-planning, and motor impulsiveness significant influenced the likelihood of HD. Forward selection of the model was performed to avoid multicollinearity problems. The Hosmer and Lemeshow test validated the model's goodness of Fit ($\chi^2 = 5.544$, $p = .598$). Table 3 shows that age at the first blackout, coping,

Table 2. Distribution of assessment scale scores in the sample ($n = 295$) and by hazardous drinking subgroup.

	Total sample ($N = 295$)	Hazardous drinking ($N = 86$)	Non hazardous drinking ($N = 209$)	*Coefficients*	*p-Value*
AUDIT, mean (SD)	10.2 (6.9)	16.5 (4.9)	8.2 (5.1)	$t = 10.421$	<0.001
ADS, mean (SD)	5.5(3.7)	7.5 (4.1)	4.7 (3.3)	$t = 2.724$	0.008**
MCQ-A, mean (SD)					
Enhancement	4.8 (3.4)	6.4 (3.7)	4.1 (3.1)	$t = 2.609$	0.011*
Coping	5.2 (3.9)	7.2 (4.4)	4.4 (3.6)	$t = 2.688$	0.009**
Conformity	4.1(2.7)	3.9 (2.3)	4.1 (2.8)	$t = 0.277$	0.726
Social	8.2 (3.7)	10.1 (2.4)	7.3 (3.8)	$t = 2.427$	0.018*
AOES, mean (SD)					
Positive expectancy	58.3 (14.9)	59.4 (15.5)	57.9 (14.8)	$t = 0.354$	0.725
Negative expectancy	46.4 (12.1)	42.8 (12.3)	47.6 (11.9)	$t = -1.660$	0.101
SES, mean (SD)	26.3 (1.6)	25.9 (1.9)	26.4 (1.5)	$t = 0.928$	0.357
SDS, mean (SD)	41.7 (5.4).	41.4 (5.1)	42.9 (6.2)	$t = 0.341$	0.734
BIS, mean (SD)					
Non-planning impulsiveness	19.2 (4.5)	27.5 (4.5)	16.9 (4.8)	$t = 5.175$	<0.001
Motor impulsiveness	16.8 (3.9)	18.1 (4.4)	16.1 (2.9)	$t = 2.053$	0.045*
Attention impulsiveness	17.1 (2.9)	16.1 (3.2)	17.4 (2.8)	$t = 1.487$	0.142
STAI, mean (SD)1	41.7 (6.4)	40.7 (6.9)	41.5 (5.6)	$t = 0.124$	0.901
STAXI, mean (SD)	18.7 (3.7)	20.3 (4.9)	18.1 (6.4)	$t = 1.609$	0.113

Notes: ADS: Alcohol Dependence Scale; AOES: Alcohol Outcome Expectancies Scale; AUDIT: Alcohol Use Disorder Identification Test; BIS: Barratt Impulsiveness Scale; MCQ-A: Motivational Structure Questionnaire for alcoholics; SD: standard deviation; STAI: State Trait Anxiety Inventory; STAXI: State Trait Anger Expression Inventory.
*$p < .05$; **$p < .01$.

Table 3. Result of the logistic regression estimations of the influences of the clinical factors on the odds of hazardous drinking (n = 295).

	B	Standard error	Wals	*p*-Value	Odd ratio	95% CI
Significant other HD	2.079	1.082	3.697	0.055	5.609	0.861–36.629
Age at first HD	−0.143	0.153	0.869	0.159	0.867	0.662–1.170
Age at first blackout	−0.244	0.107	5.184	0.023*	0.783	0.635–0.967
Enhancement (MCQ-A)	0.031	0.154	0.040	0.842	0.970	0.717–1.311
Coping (MCQ-A)	0.251	0.104	5.833	0.016*	1.285	1.048–1.575
Social (MCQ-A)	0.287	0.135	4.488	0.034*	1.332	1.022–1.737
Non-planning impulsiveness (BIS)	0.191	0.071	7.221	0.008**	1.206	1.053–1.391
Motor impulsiveness (BIS)	−0.129	0.075	2.237	0.084	0.879	0.759–1.017

Note: B: Unstandardized regression coefficient, BIS: Barratt Impulsiveness Scale; HD: Hazardous drinking; MCQ-A: Motivational Structure Questionnaire for alcoholics.
*$p < .05$; **$p < .01$.

and social motives for alcohol use, and non-planning impulsiveness increased the likelyhood of HD net of each other's effects, and the final model explained 29.6% (Negelkerke R^2) of the variation in HD.

Discussion

The purpose of this study was to identify clinical correlates of HD, and we found that age at first blackout, coping and social motives for alcohol use, and non-planning impulsiveness were significant factors. The results support the findings of a Swedish male study on blackout experiences in relation to the development of AUD that alcohol induced blackout at age 20 predicted the development, maintenance and severity of binge drinking at age 25 (adjusted odd ratio: 1.47, 1.66, and 1.20, each other) [4]. Those results suggest that experiencing a blackout at relatively young age might be an important indicator of HD development. Some studies have found that blackouts are caused by damage to multiple memory systems in discrete brain regions, and the acute effects of alcohol on learning and memory might be caused by the neuronal change to the hippocampus and related structures on a cellular level. Altered memory functions during intoxication might affect an individual's alcohol expectancy and drinking pattern, which might lead to HD [18]. Therefore, a blackout experience at a relatively young age might be a useful clinical factor for predicting future HD. In light of other findings of genetic or central nervous system vulnerability, effectively managing blackouts might reduce the occurrence of HD.

Many previous studies have found that coping and social motives for alcohol use significantly influenced on HD. The motives for alcohol use have been closely related with alcohol consumption and it's negative consequences including self-harm, violence, and social and interpersonal problem [19]. To explain these results, some studies point out that individuals with stronger social motives for drinking less often used protective strategies, and individuals who had greater coping motives had fewer protective behaviour strategy [20]. Another study proposed that irrational decision making pattern might mediate abnormal motives for drinking and HD [7]. Supporting the previous studies, we found that motives for drinking were the most likely cause of HD in this study.

The relationship between impulsiveness and alcohol consumption has been reported in human and animal research. In humans, impulsiveness has been associated with the initiation of alcohol use, current use, early indicators of alcohol problems and alcohol abuse [21]. Motor and non-planning impulsiveness have been correlated with the number of drinks per drinking occasion, and attentional impulsiveness have been correlated with the duration of drinking occasions. In a community sample of adult men and women, overall and three dimensions of impulsiveness (non-planning, attentional, and motor impulsiveness) independently predicted of alcohol consumption [22]. In our study, motor and non-planning impulsiveness were higher in HD than NHD group. However, only non-planning impulsiveness was a statistically significant factor in the regression analysis. Non-planning impulsiveness refers to a tendency not to plan ahead and is strongly related to non-compliance with norm and rules. Because it might be associated with less treatment compliance and more legal problems, it required further study.

In our previous study, we have demonstrated by the structural equation model that the negative experience of childhood stimulates motives for alcohol use mediate irrational coping mechanism, represented by an impulsive and intuitional decision making style [7]. We plan to conduct follow-up research based on a combination of the results of the two studies to identify the links among impulsiveness, motives for alcohol use and HD. Additional studies are needed on the influence of impulsiveness on age at first HD, first blackout, and other clinical correlates of HD maintenance. In addition, impulsiveness is highly inherited trait, and it might be possible to obtain clinically useful results by analyzing the relationship between individuals' clinical HD related factors and the impulsiveness of the family members.

Despite its value, this study has several limitations. First, the measure of alcohol consumption was derived from the subjects' personal drinking record, and it did not account for biomarkers such as mean cell volume, carbohydrate deficient transferrin or gamma glutamyl transferase that might measure individuals' drinking status. Second, comorbidities, including affective

disorder and anxiety disorder, were not taken into consideration. Third, we did not control for the covariance of attention deficit/Hyperactivity disorder or other personality disorders that might influence impulsiveness. Fourth, changes in the subjects' lives or stress events that might influence their drinking behaviours were not consideration. Fifth, the study's sample comprised of men so gender differences were not analysed. Last, other related problems, such as current smoking, hypochondriasis, and suicidal ideation, which are the risk factors of HD, presented by Park et al. [23], were not part of this study.

Despite these limitations, this study provides a comprehensive contrast of diverse clinical characteristics of Korean men with to those without HD. We found that relatively young age at first blackout experience, coping and social motives for alcohol use, and non-planning impulsiveness were significantly more likely among hazardous than non hazardous drinkers. Thus, screening male drinkers for alcohol abuse might identify men who might benefit from targeted comprehensive therapeutic approaches.

Disclosure statement

No potential conflict of interest was reported by the authors.

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References

[1] Harrison EL, Desai RA, McKee SA. Nondaily smoking and alcohol use, hazardous drinking, and alcohol diagnoses among young adults: findings from the NESARC. Alcohol Clin Exp Res. 2008;32(12):2081–2087.
[2] Syden L, Sidorchuk A, Makela P, et al. The contribution of alcohol use and other behavioural, material and social factors to socio-economic differences in alcohol-related disorders in a Swedish cohort. Addict. 2017;112(11):1920–1930.
[3] Tawa EA, Hall SD, Lohoff FW. Overview of genetics of alcohol use disorder. Alcohol. 2016;51(5):507–514.
[4] Studer J, Gmel G, Bertholet N, et al. Alcohol-induced blackouts at age 20 predict the incidence, maintenance, and severity of alcohol dependence at age 25: a prospective study in a sample of young Swiss men. Addict. 2019; doi:10.1111/add.14647.
[5] Grant BF, Saha TD, Ruan WJ, et al. Epidemiology of DSM-5 drug use disorder: results from the national epidemiologic survey on alcohol and related conditions-III. JAMA. 2016;73(1):39–47.
[6] Hasking P, Lyvers M, Carlopio C, et al. The relationship between coping strategies, alcohol expectancies, drinking motives and drinking behaviour. Addict Behav. 2011;36:479–487.
[7] Jang OJ, Park SC, Kim SG, et al. How early life stress and insecure attachment affect decision making style and motivation to use alcohol: a structural equation model. Psychiat Clin Psych. 2019;29(2):178–181.
[8] Sampson L, Cohen GH, Calabrese JR, et al. Mental health over time in a military sample: the impact of alcohol use disorder on trajectories of psychopathology after deployment. J Trauma Stress. 2015;28(6):547–555.
[9] Saunders JB, Aasland OG, Babor TF, et al. Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. Addict. 1993;88(6):791–804.
[10] Cox WM, Klinger E. A motivational model of alcohol use: determinants of use and change. J Abnorm Psychol. 1988;97:165–180.
[11] Leigh B, Stacy AW. Alcohol Outcome Expectancies: Scale construction and predictive utility in higher order confirmatory models. Psychol Assess. 1993;5(2):216–229.
[12] Skinner HA, Allen BA. Differential assessment of alcoholism. Evaluation of the Alcohol Use Inventory. J Stud Alcohol. 1983;44(5):852–862.
[13] Rosenberg M. Society and the adolescent self-image. Princeton (NJ): Princeton: Princeton University Press; 1996.
[14] Zung WWK. A Self-rating Depression Scale. Arch Gen Psychiatry. 1965;12(1):63–70.
[15] Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt Impulsiveness Scale. J Clin Psychol. 1995;51(6):768–774.
[16] Spielberger CD, Gorsuch RL, Lushene R, et al. Manual for the State-Trait Anxiety Inventory(form Y). Palo Alto (CA): Consulting Psychologists Press; 1983.
[17] Spielberger CD. Manual for the State-Trait Anger Expression Inventory (STAXI). Odesa (FL): Psychological Assessment Resources; 1988.
[18] Lee H, Roh SW, Kim DJ. Alcohol-induced blackout. Int J Environ Res Public Health. 2009;6(11):2783–2792.
[19] Hingson RW, Zha W, Weitzman ER. Magnitude of and trends in alcohol related mortality and morbidity among U.S college students ages 18-24, 1998-2005. J Stud Alcohol Drugs. 2009;16:12–20.
[20] Becker HC, Mulholland PJ. Neurochemical mechanisms of alcohol withdrawal. Handb Clin Neurol. 2014;125:133–156.
[21] Spoelder M, Flores Dourojeanni JP, de Git KCG, et al. Individual differences in voluntary alcohol intake in rats: relationship with impulsivity, decision making and Pavlovian conditioned approach. Psychopharmacology (Berl.). 2017;234(14):2177–2196.
[22] Hamilton KR, Sinha R, Potenza MN. Hazardous drinking and dimensions of impulsivity, behavioral approach, and inhibition in adult men and women. Alcohol Clin Exp Res. 2012;36(6):434–449.
[23] Park SC, Lee SK, Oh HS, et al. Hazardous drinking-related characteristics of depressive disorders in Korea: the CRESCEND study. J Korean Med Sci. 2015;30(1):74–81.