EÖTVÖS LORÁND UNIVERSITY
FACULTY OF EDUCATION AND PSYCHOLOGY
DOCTORAL SCHOOL OF PSYCHOLOGY

ZSOLT HORVÁTH

ASSOCIATIONS BETWEEN ALCOHOL USE-RELATED OUTCOMES AND PSYCHOPATHOLOGICAL SYMPTOMS: PERSON- AND VARIABLE-ORIENTED APPROACHES

DOCTORAL SCHOOL OF PSYCHOLOGY
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II. Nyilatkozatok

1. A doktori értekezés szerzőjeként[2]

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[1] A kari hivatal ügyintézője tölti ki.
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Kelt: Budapest, 2021.10.26.

a doktori értekezés szerzőjének aláírása

[3] A doktori értekezés benyújtásával egyidejűleg be kell adni a tudományági doktori tanácshoz a szabadalmi, illetőleg oltalmi bejelentést tanúsító okiratot és a nyilvánosságra hozatal elhalasztása iránti kérelmet.

[4] A doktori értekezés benyújtásával egyidejűleg be kell nyújtani a minősített adatra vonatkozó közokiratot.

[5] A doktori értekezés benyújtásával egyidejűleg be kell nyújtani a mű kiadásáról szóló kiadói szerződést.

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List of abbreviations

α	Cronbach's Alpha internal reliability index
AA	Alcoholics Anonymous
ACC	Anterior Cingulate Cortex
AD	Anxiety Disorder
ADHD	Attention Deficit Hyperactivity Disorder
AIC	Akaike Information Criteria
AN	Anorexia Nervosa
ANX	Anxiety
ASPD	Antisocial Personality Disorder
ASSIST	Alcohol, Smoking and Substance Involvement Screening Test
AUD	Alcohol Use Disorder
AUDIT	Alcohol Use Disorders Identification Test
B	Unstandardized regression coefficient or parameter estimate
β	Standardized regression coefficient or parameter estimate
BCH	Bolck-Croon-Hagenaars [method]
BD	Bipolar Disorder
BDNF	Brain-Derived Neurotrophic Factor
BED	Binge Eating Disorder
BIC	Bayesian Information Criteria
BMI	Body Mass Index
BN	Bulimia Nervosa
BNTS	Bed Nucleus of the Stria Terminalis

BPD	Borderline Personality Disorder
BSI	Brief Symptom Inventory
χ^2	Chi-square statistics
CD	Conduct Disorder
CeA	Central nucleus of amygdala
CEE	Central-Eastern Europe
CES-D	Center of Epidemiological Studies – Depression scale
CFI	Comparative Fit Index
CHRNA4	Cholinergic Receptor Nicotinic Alpha 4
COMT	Catechol-O-Methyltransferase
CRHM2	Muscarinic Acetylcholine Receptor M2
CI	Confidence Interval
CLA	Common Liability to Addiction
CREB	cAMP Response Element-Binding protein
CRF	Corticotropin Releasing Factor
d	Cohen's d effect size measure
DALY	Disability Adjusted Life Years
DEP	Depression
DMQ	Drinking Motives Questionnaire
DMQ-R	Drinking Motivations Questionnaire – Revised
DMQ-SF	Drinking Motives Questionnaire – Short Form
DRD1	Dopamine D1 receptor
DRD2	Dopamine D2 receptor
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders – Fourth edition
DSM-5	Diagnostic and Statistical Manual of Mental Disorders – Fifth edition
ED	Eating Disorder
EMA	Ecological Momentary Assessment

ESPAD	European School Survey Project on Alcohol and Other Drugs
FAD	Food and Alcohol Disturbance
FASD	Fetal Alcohol Spectrum Disorders
FED	Feeding and Eating Disorder
GABA	γ-aminobutyric acid
GAD	Generalized Anxiety Disorder
GD	Gaming Disorder
GS	Global Symptom severity
HBSC	Health Behaviour in School-aged Children Survey
HED	Heavy Episodic Drinking
HiTOP	Hierarchical Taxonomy of Psychopathology
HIV/AIDS	Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome
HOS	Hostility
HPA	Hypothalamic-Pituitary-Adrenal [axis]
I	Intercept
ICD-11	International Classification of Diseases – Eleventh edition
IGD	Internet Gaming Disorder
IGDT-10	Ten-Item Internet Gaming Disorder Test
IRT	Item Response Theory
IS	Interpersonal Sensitivity
λ	Standardized factor loading
LCA	Latent Class Analysis
LCGA	Latent Class Growth Analysis
LMRT	Lo-Mendel-Rubin Adjusted Likelihood Ratio Test

LPHN3	Latrophilin 3
M	Mean
MAO-A	Monoamine Oxidase-A
MDD	Major Depressive Disorder
MDMA	3,4-Methylenedioxy-Methamphetamine
MM	Minnesota Model
N	Number of cases
NESARC	National Epidemiologic Survey on Alcohol and Related Conditions
NPY	Neuropeptide Y
NSAPH	National Survey on Addiction Problems in Hungary
ω	McDonalds's Omega internal reliability index
OC	Obsessive-Compulsive
OCD	Obsessive-Compulsive Disorder
ODD	Oppositional Defiant Disorder
OFC	Orbitofrontal Cortex
OPRMI	Mu-Opioid Receptor gene
OR	Odds Ratio
p	P-value
φ	Phi effect size measure
PAR	Paranoid ideation
PCC	Posterior Cingulate Cortex
PHO	Phobic anxiety
PSY	Psychoticism
PTSD	Posttraumatic stress disorder

r	Pearson’s correlation estimate
RCI	Reliability Change Index
RCT	Randomized Controlled Trial
Ref.	Reference Group
ReHo	Regional Homogeneity
RMSEA	Root Mean Squared Error of Approximation
S	Slope
SAD	Social Anxiety Disorder
SD	Standard Deviation
SE	Standard Error
SEM	Structural Equation Modeling
SOM	Somatization
SSA-BIC	Sample Size Adjusted Bayesian Information Criteria
SUD	Substance Use Disorder
t	Independent-samples T-test statistics
TLI	Tucker-Lewis Index
TSF	Twelve Step Facilitation
VS	Ventral Striatum
VTA	Ventral Tegmental Area
WHO	World Health Organization
WLSMV	Weighted Least Squares Mean and Variance

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List of publications included in the dissertation

Study	Publication	Journal impact factor	Highest Q-rating of the journal
1	Horváth, Zs., Paksi, B., Felvinczi, K., Griffiths, M. D., Demetrovics, Zs., & Urbán, R. (2019). An empirically based typology of alcohol users in a community sample using latent class analysis. *European Addiction Research, 25*(6), 293-302. https://doi.org/10.1159/000501516	2.269	Q1
2	Horváth, Zs., Tremkó, M., Fazekas, Zs., Tóth, A., Petke, Zs., Farkas, J., Griffiths, M. D., Demetrovics, Zs., & Urbán, R. (2020). Patterns and temporal change of psychopathological symptoms among inpatients with alcohol use disorder undergoing a twelve-step based treatment. *Addictive Behaviors Reports, 12*, 100302. https://doi.org/10.1016/j.abrep.2020.100302	N/A	Q1
3	Horváth, Zs., Király, O., Demetrovics, Zs., Németh, Á., Várnai, D., & Urbán, R. (2021). Polysubstance use is positively associated with gaming disorder symptom severity: A latent class analytical study. *European Addiction Research*. https://doi.org/10.1159/000517042	3.015*	Q1*
4	Horváth, Zs., Román, N., Elekes, Zs., Griffiths, M. D., Demetrovics, Zs., & Urbán, R. (2020). Alcohol consumption and risk for feeding and eating disorders in adolescence: The mediating role of drinking motives. *Addictive behaviors, 107*, 106431. https://doi.org/10.1016/j.addbeh.2020.106431	3.913	Q1

Note. Except for Study 3, journal impact factors and highest Q-ratings are presented for each study based on statistics from the year of publication. In the case of Study 3, journal statistics are not available yet for the year of 2021, therefore the latest journal statistics from 2020 are reported (marked with asterisk). Co-authors in each study granted permission for the inclusion of the publications in the dissertation.

I. Preface

Alcohol consumption has a detrimental effect on health: morbidity and mortality due to various disease and injury types can be attributable to alcohol use. Globally, alcohol consumption was responsible for approximately 5% of all deaths (i.e., approximately 3 million alcohol-attributable deaths) and 5% of all disability-adjusted life years lost due to mortality and disability in 2016 (DALYs; more than 130 million alcohol-attributable DALYs) (Griswold et al., 2018; Shield et al., 2020; World Health Organization, 2018a). Alcohol-attributable disease and injury categories differ as a function of whether alcohol consumption is fully or partly responsible for the presence of the negative health-related outcome (Rehm et al., 2017). For example, alcohol use disorder (AUD), alcohol poisoning and fetal alcohol spectrum disorders (FASD) are disease categories that are entirely attributable to alcohol consumption, whereas morbidity and mortality due to infectious diseases (e.g., tuberculosis, HIV/AIDS, pneumonia), various types of cancers (e.g., oral cavity, liver, colon, rectal, breast cancers), neuropsychiatric disorders (e.g., Alzheimer's disease, epilepsy, major depressive disorder), cardiovascular diseases (e.g., ischaemic heart disease, various types of stroke), gastrointestinal diseases (e.g., liver cirrhosis, pancreatitis), injuries (e.g., from traffic accidents) and suicide are party explained by alcohol use (Rehm et al., 2017). Moreover, alcohol use not only has a detrimental effect on the drinkers' health but also contributes to significant health burden to others (e.g., aggression and violence in intimate relationships, lower levels of affected family members' mental health) (Di Sarno et al., 2021; Horváth & Urbán, 2019; Rehm et al., 2017).

Alcohol use contributes to a significant public health problem in Hungary due to the high levels of alcohol consumption, problematic forms of alcohol use and alcohol-attributable health burden. According to the World Health Organization's (WHO) statistics from 2016, the total alcohol consumption per capita was 11.4 liters of pure alcohol (19.1, 4.5 and 9.8 liters among Hungarian males and females and in the WHO European Region on average, respectively), the prevalence rate of heavy episodic drinking (HED) in the past 30 days was 33.5% (53.0% and 16.3% for males and females, respectively), and the 12-month prevalence rate of AUD was 21.2% (36.9%, 7.2% and 8.8% among Hungarian males and females and in the WHO European Region on average, respectively) in the Hungarian population aged 15 years or older (World Health Organization, 2018a). Compared to the average global and European Union estimates from 2016, higher levels

of alcohol-attributable death and DALYs were presented in Hungary (Griswold et al., 2018; Shield et al., 2020). Overall, in line with global trends, Hungarian males showed a higher risk for harmful alcohol consumption-related outcomes and alcohol-attributable burden than females (Griswold et al., 2018; Shield et al., 2020; World Health Organization, 2018a).

Adolescence is considered an important developmental phase in terms of alcohol use, as onset of experimental, regular, and problematic alcohol use can occur in this period. Moreover, adolescent alcohol use can be associated with various forms of negative social and health outcomes, such as injuries, aggressive behaviors, sexual violence, unprotected sexual behavior, impaired brain development, negative consequences on school-related performance and bonding (Chung et al., 2018; Hingson & White, 2014). According to the large cross-national statistics of the Health Behaviour in School-aged Children (HBSC) Study from 2017/2018 and the European School Survey Project on Alcohol and Other Drugs (ESPAD) from 2019, prevalence rates of alcohol use and drunkenness among Hungarian 15-16 years-old adolescents exceeded the European average levels, and in some indicators one of the highest prevalence rates were shown in Hungary (ESPAD Group, 2020; Inchley et al., 2020a). For example, 91% of the Hungarian 15-16 years-old adolescents (compared to the average European level of 79%) reported about lifetime use of alcohol, and the prevalence of alcohol consumption and drunkenness in the past 30 days among Hungarian adolescents was 61% and 21% (compared to the average European levels of 47% and 13%), respectively (ESPAD Group, 2020). Moreover, among 15-19 years-old Hungarians higher levels of alcohol-attributable death and DALYs were demonstrated compared to global average levels (Griswold et al., 2018).

The societal costs of alcohol use highlight the importance of investigating further alcohol consumption patterns in Hungary in order to identify more precisely individuals with risky and harmful alcohol consumption as well as to gain more accurate knowledge on the risk mechanisms underlying adverse alcohol use-related outcomes. To address these issues, the studies performed in the present dissertation examines the co-occurrence of psychopathological symptoms and outcomes of alcohol use from different perspectives. Although there has been extensive research in the international literature on identifying individuals with harmful alcohol consumption patterns as well as on the associations between psychopathological symptoms and alcohol use-related outcomes, less is known on these issues in Hungary (e.g., Bácskai et al., 2011; Gerevich et al., 2006). Considering

the very high levels of alcohol use-related burden in Hungary, it was expected that the studies of the present dissertation can contribute to broaden our epidemiological and psychopathological knowledge of Hungarian alcohol users. Specifically, the present dissertation aimed to (i) identify empirically-based subgroups of alcohol users in clinical and general adult and adolescent samples and to examine their associations with various dimensions of psychopathological symptoms, and (ii) to investigate the role of drinking motives on the relationships between psychopathological symptoms and outcomes of alcohol use. Identifying empirically-based subgroups of alcohol users can allow to differentiate alcohol users with different severity levels more precisely and to describe at-risk and/or problematic alcohol drinker classes in the Hungarian adult and adolescent population. By estimating the prevalence of these at-risk and/or problematic alcohol user subgroups in the Hungarian population, this approach can also be informative for policy makers and for those designing population level intervention and prevention programs. Moreover, the studies which were performed in this dissertation also aimed to contribute to novel and more specific findings on the associations between alcohol use and risk for eating disorders (EDs) and gaming disorder (GD) among adolescents by considering the role of drinking motives and polysubstance use pattern, respectively. It was expected that these findings can help to better understand the background and mechanisms of the co-occurrences between elevated alcohol use and the abovementioned risk behaviors. Finally, the present dissertation attempted to broaden our knowledge on the motivational background and pathways of alcohol use among individuals with clinically diagnosed AUD.

II. General introduction

II/1. Etiological models of AUD

The diagnostic category of AUD denotes a problematic pattern of alcohol use which is characterized by impaired control of use, dependence symptoms and negative psychological and social consequences due to alcohol consumption (American Psychiatric Association, 2013). Multiple and diverse factors are considered in the etiological mechanisms explaining AUD, such as genetic predispositions, environmental, psychological and neurobiological factors. Therefore, the present dissertation only provides a superficial review for some of the etiological models (e.g., theoretical models which are relevant in terms of comorbidity between AUD and psychiatric disorders, drinking motives).

II/1/1. Exploratory models highlighting the functions of psychopathological risk mechanisms and drinking motives in AUD

Various psychoanalytic theories of AUD highlight the emotion regulating function of alcohol use (for review, see: Kun & Demetrovics, 2010). For example, in Khantzian's self-medication hypothesis substance use is conceptualized as a consequence of the individual's difficulties in terms self-regulation, emotion regulation, self-esteem and personality organization. That is, affected individuals consume psychoactive substances in order to compensate these difficulties. In this model the primarily function of substance use is to cope with painful, negative, intense emotions and difficulties in expressing feelings, regulating their self-esteem and relationships. Overall, the self-medication model assumes that different risk profiles (i.e., psychopharmacological characteristics, and intense, painful emotions) can account for the unconscious choice for selecting a specific type of psychoactive substance (e.g., alcohol can be used to alleviate feelings of isolation, emptiness as a result of rigid defense mechanisms) (Khantzian, 1987, 1997; Koob & Le Moal, 2008; Kun & Demetrovics, 2010).

Other models, such as the allostatic hypothesis, also underlines that drinking motives and negative affectivity have a central function in the development of AUD (Koob, 2011; Koob & Le Moal, 2008; Le Moal & Koob, 2007). In the allostatic model's framework, there are cyclical associations between consecutive stages of problematic substance use: (i) preoccupation/anticipation, (ii) intoxication/binging and (iii) withdrawal/negative affect. These stages of addictive problems are characterized by different neurobiological

and motivational processes. In the case of alcohol use, the stage of intoxication/binging represents a more impulsive form of alcohol use: high levels of reward-seeking and enhancement tendencies are characteristic at this stage (e.g., before alcohol use one might experience elevated arousal, whereas after alcohol use one can experience gratification). In the withdrawal/negative affect stage compulsive alcohol consumption tendencies become dominant: drinking's function is to cope with high levels of withdrawal symptoms, such as anxious-depressive and somatic symptoms. That is, there is a shift from positive reinforcement motivational processes to negative reinforcement motivational mechanism between the stages of intoxication/binging and withdrawal/negative affect. Craving can be a characteristic feature of the stage of preoccupation/anticipation. Environmental factors (e.g., stimuli which are associated with alcohol use) as well as positive and negative emotional states can lead to craving which increases in the compulsive stage of AUD and can be a risk factor for relapses (Koob, 2011; Koob & Le Moal, 2008; Le Moal & Koob, 2007).

The allostatic hypothesis also assumes that different neurobiological characteristics are relevant to each stage of AUD. Positive reinforcing mechanisms (e.g., during the binge/intoxication stage) are associated with the activation of the brain reward systems, such as dopaminergic and opioid peptide systems. Activation of specific neurobiological areas were identified which are related to the positive reinforcing effects of alcohol, such as the extended amygdala (e.g., comprising the central nucleus of amygdala [CeA], the bed nucleus of the stria terminalis [BNTS], the shell of the nucleus accumbens), the ventral tegmental area. Moreover, multiple neurotransmitters are included in the positive reinforcing effects of alcohol, such as dopamine, opioid peptides, γ-aminobutyric acid (GABA) and endocannabinoids. Contrary, in the withdrawal/negative affect stage the negative reinforcing mechanisms are represented by decreased activity in the mesocorticolimic dopamine system, and by reduced levels of opioid peptide, GABA and glutamate functions in the nucleus accumbens and in the amygdala. Moreover, the activation of the stress systems (e.g., hypothalamic-pituitary-adrenal [HPA] axis) is also presented in the withdrawal/negative affect stage. That is, increased levels of corticotropin releasing factor (CRF) in the CeA and norepinephrine in the BNTS are associated with negative reinforcing mechanisms. Overall, the chronic nature of AUD can contribute to neuroadaptive alterations in the reward and stress systems which can

lead to an allostatic state regarding reward and motivational dysregulation (i.e., alterations in the hedonic set-point) (Koob & Le Moal, 2008).

It is important to note that, in line with the assumptions of the self-medication and allostatic hypotheses on psychological risk mechanisms accounting for AUD (e.g., low self-control, high negative affectivity and affective dysregulation, high reward seeking), problematic alcohol use can be associated with distinct patterns of personality traits. Namely, meta-analytic findings showed positive associations between negative outcomes of alcohol use and high rates of neuroticism (e.g., affective dysregulation), self-control difficulties (e.g., high rates of impulsivity [e.g., lack of planning, positive and negative urgency], low rates of consciousness [e.g., lack of premeditation, perseverance]) and sensation-seeking (Coskunpinar et al., 2013; Hittner & Swickert, 2006; Kotov et al., 2010; Malouff et al., 2007).

II/1/2. Other relevant cognitive and social risk mechanisms accounting for AUD and elevated alcohol use

In addition to drinking motives, based on the general social-cognitive theory, several theoretical models of problematic alcohol use (e.g., the expectancy theory of alcohol use, the cognitive model of binge drinking, the relapse prevention model) were consistent in highlighting the importance of other alcohol use-related cognitive constructs, such as alcohol outcome expectancies and drinking refusal self-efficacy (Bandura, 1998; Hasking & Oei, 2008; Jones et al., 2001; Marlatt & Donovan, 2008; Oei & Baldwin, 1994; Oei & Morawska, 2004). Alcohol outcome expectancies refer to an individual's beliefs and views on the expected physical, social and psychological effects resulting from alcohol use (Hasking & Oei, 2008; Jones et al., 2001). In the hierarchical structure of outcome expectancies, two broad categories can be discriminated: positive (e.g., tension reduction, positive social consequences) and negative alcohol outcome expectancies (e.g., aggression, negative social consequences, physical symptoms) (Jones et al., 2001; Leigh & Stacy, 2004) The expectancy theory of alcohol use suggested that positive alcohol outcome expectancies can lead to higher engagement in alcohol use behavior (i.e., due to the expected reinforcing effects), whereas negative outcome expectancies can lead to lower rates of alcohol consumption (Hasking & Oei, 2008; Jones et al., 2001). Empirical findings showed that positive outcome expectancies were positively related with higher levels of alcohol quantity and alcohol problems, whereas in the cases of negative outcome expectancies less consistent findings were shown (Bot et al., 2005; Monk & Heim, 2013;

Pabst et al., 2010; Zamboanga et al., 2006). Drinking refusal self-efficacy represents an individual's perceived ability to control the drinking behavior and resist to drinking in different situations (e.g., when there is an opportunity for using alcohol, when someone is experiencing negative affective states) (Hasking & Oei, 2008; Oei et al., 2005). Previous studies consistently reported negative relationships between drinking refusal self-efficacy and levels of alcohol consumption and alcohol problems (Hasking & Oei, 2008). Moreover, the interactive effect of alcohol outcome expectancies and drinking refusal self-efficacy was also proposed in the explanation of outcomes of alcohol use. Namely, higher levels of positive alcohol outcome expectancies in addition to lower rates of refusal self-efficacy can lead to higher levels of alcohol use (Hasking & Oei, 2008).

Alcohol outcome expectancies and drinking refusal self-efficacy can also be integrated in the aforementioned negative reinforcement mechanisms explaining AUD. The extended model of the social-cognitive model of alcohol use highlighted cyclical and reinforcing interrelationships between high rates of maladaptive coping with stress, coping drinking motives, positive alcohol expectancies and low levels of drinking refusal self-efficacy in the explanation of alcohol use outcomes (Hasking & Oei, 2008). For example, someone who shows a preference for using avoidant strategies to cope with anxious-negative affective states and holds positive expectancies regarding the tension-reducing effects of alcohol might drink alcohol in such situations due to the short-term tension-reducing effect of alcohol. This might contribute to the reduce of refusal self-efficacy when this individual experience negative affective states and might develop a pattern of using alcohol to cope with anxious-depressive symptoms due to the lack of ability to use adaptive stress management strategies. It can be expected that this alcohol use pattern might lead to subsequent development of AUD via further reductions in drinking refusal self-efficacy and increase in alcohol outcome expectancies (Hasking & Oei, 2008).

Moreover, based on Bandura's social-cognitive theory, social and environmental influences also can account for engaging in health behaviors (Bandura, 1998; Hasking & Oei, 2008). For example, among adolescents, peer effects are associated with subsequent alcohol use. Two, bidirectionally associated processes are suggested: peer selection refers to a pattern when an alcohol using adolescent subsequently select and seek friends who drink alcohol, whereas peer influence represents that having alcohol using friends can contribute to the adolescent's subsequent alcohol use (Leung et al., 2014). Other models, such as the theory of planned behavior (Ajzen, 1991), underline the functions of

descriptive (e.g., individuals' perception on how many of their friends use alcohol) subjective norms (e.g., individuals' perception on others' approval or disapproval regarding alcohol use) which can be associated with intention to use alcohol and with alcohol using behavior (Cooke et al., 2016; McEachan et al., 2016). Moreover, extensive research examined risk and protective parental and family factors on alcohol consumption among adolescents and young adults. Meta-analytic findings suggested that family history of problematic alcohol use, higher levels of parental provision of alcohol, parental positive attitudes and approval towards alcohol use, parental modelling of drinking, and lower levels of parental monitoring and parent-child relationship quality (e.g., lower levels of perceived support from parents, bonding with parents, quality of communication with parents) were associated with elevated rates of alcohol use-related outcomes (Elliott et al., 2012; Ryan et al., 2010; Yap et al., 2017).

II/1/3. Genetic risks for AUD

However, in the cases of the effects of parental and familial factors on the subsequent problematic alcohol use, not only environmental but genetic factors are also relevant. A meta-analysis of twin and adoption studies reported that the heritability of AUD was 49% (Verhulst et al., 2015). Previous studies which examined the genetics of AUD identified multiple risk factors, including genes encoding alcohol metabolism (e.g., ADH, ADH1A, ADH1B, ADH1C, ALDH2), and genes which have a function in encoding dopaminergic (e.g., DRD2, MAOA, COMT, SLC6A3) serotonergic (e.g., HTR3A, HTR1B, HTR3B, SLC6A4), GABAergic (e.g., GABRA1, GABRA2, GAD1, KCNJ9/GIRK3), glutamatergic (e.g., GRIN2C, GRM8 GIRK1), cholinergic (e.g., CHRM2, CHRNA5), opioid (e.g., OPRK1, OPRM1) and peptide (e.g., NPY) neurotransmission processes (Prom-Wormley et al., 2017; Samochowiec et al., 2014; Tawa et al., 2016). Moreover, other studies reported significant gene-environment interactions (e.g., between GABRA2 gene and marital status), which also can account for the development of AUD (Samochowiec et al., 2014).

II/2. Comorbidity between AUD and different types of psychiatric disorders

The simultaneous or consecutive co-occurrence of AUD and other psychiatric disorders is highly prevalent, which can contribute to significant health burden, for example, by leading to adverse treatment outcomes (e.g., higher rates of relapse) and increased mortality rates (Bradizza et al., 2006; Castillo-Carniglia et al., 2019; Hjorthøj et al., 2015;

Jane-Llopis & Matytsina, 2006). The large taxonomic systems of mental disorders (i.e., Diagnostic and Statistical Manual of Mental Disorders [DSM–5], International Classification of Diseases [ICD-11]) also consider alcohol-induced mental disorders: as a direct result of alcohol intoxication or withdrawal mood, anxiety, and other psychiatric disorders can emerge (American Psychiatric Association, 2013; World Health Organization, 2018b).

II/2/1. Comorbidity with externalizing psychiatric disorders

Extensive cross-sectional and longitudinal findings suggest positive associations and high comorbidity rates between AUD and other externalizing psychiatric disorders, such as substance use disorders (SUDs), attention deficit hyperactivity disorder (ADHD), conduct disorder (CD), oppositional defiant disorder (ODD) and antisocial personality disorder (ASPD) (Castillo-Carniglia et al., 2019; Charach et al., 2011; Glenn et al., 2013; Grant et al., 2015; Groenman et al., 2017; Guy et al., 2018; Helle et al., 2019; Lee et al., 2011; Morales et al., 2020; van Emmerik-van Oortmerssen et al., 2012; Yurasek et al., 2017).

A possible explanation for these comorbidities is that each disorder is related to a broad, higher-order dimension of externalizing disorders, representing a common liability to externalizing behaviors and behavioral dysregulation. In other words, there are shared and non-disorder-specific etiologic and psychopathological factors across these disorders, such as similarities and overlap in genetic correlations, neurobiological characteristics (e.g., areas responsible for behavioral regulation, executive functions, reward mechanisms, stress response; for examples, see: II/Table 1.), environmental risk factors (e.g., child abuse and trauma, interpersonal violence in family, being a member of a peer group with antisocial tendencies), psychological mechanisms (e.g., impulsivity, behavioral dysregulation, sensation seeking) as well as in symptomatic features (e.g., overlapping symptoms between AUD and SUDs, symptoms representing impaired behavioral control across externalizing disorders). Thus, according to these approaches of the externalizing continuum, different levels of AUD are explained by (i) specific effects of alcohol drinking and problems, (ii) a shared risk factor of SUDs which incorporates problems regarding the use of various psychoactive substances and (iii) a broad, transdiagnostic externalizing factor (Clark, 2004; Eaton et al., 2015; Groenman et

al., 2017; Helle et al., 2019; Kotov et al., 2017; Krueger & South, 2009; Lee et al., 2011; Ruiz et al., 2008; Thatcher & Clark, 2008; Vanyukov et al., 2012).

However, it is important to note that other mechanisms also were suggested to explain the comorbidity between AUD and other externalizing disorders. For example, the gateway hypothesis attempts to explain the sequence of psychoactive substance use (e.g. alcohol consumption precedes illicit substance use) (Vanyukov et al., 2012); meta-analytic findings highlighted that childhood ADHD, CD/ODD as well as comorbid ADHD and CD/ODD can predict subsequent AUD (Groenman et al., 2017; Lee et al., 2011); whereas in the case of the comorbidity between AUD and ASPD some findings support bidirectional relationships between the disorders. For example, personality traits which determine ASPD (e.g., impulsivity, sensation seeking) might positively influence the presence of AUD. In contrast, neuropsychological changes due to excessive alcohol use can affect changes on these personality traits) (Helle et al., 2019).

Although behavioral addictions are not included as a part of the externalizing spectrum in contemporary models of psychiatric comorbidity (Eaton et al., 2015; Kotov et al., 2017), it is important to discuss their associations with AUD. The present review only focuses on those forms of behavioral addictions which are considered either by the DSM-5 or the ICD-11; therefore the co-occurrence between AUD and gambling disorder and gaming disorder (GD) are discussed (American Psychiatric Association, 2013; World Health Organization, 2018b). Considering the aims of the present dissertation, it is important to review the main psychopathological characteristics of GD. According to the ICD-11, GD describes a problematic pattern of online or offline gaming behavior which can be characterized by the following symptoms: (i) loss of control over gaming behavior; (ii) giving up other activities and interests due to the increasing dominance of gaming behavior in the individuals' life; (iii) continuation of gaming despite negative consequences (e.g., experiencing psychological distress due to gaming, experiencing negative consequences on personal and social life and functioning due gaming) (World Health Organization, 2018b). That is, in line with Griffiths' component model, overlapping and similar symptomatic characteristics are related to AUD and GD (e.g., loss of control, continuation despite negative consequences) which highlights the possibility of symptomatic-level associations between the two disorders (Griffiths, 2005).

Meta-analytic findings showed high comorbidity levels between gambling disorder and AUD in clinical as well as in community samples (Cowlishaw et al., 2014; Dowling et al., 2015; Lorains et al., 2011). On the other hand, the existing literature were inconclusive on the association between GD and alcohol consumption and problems: significant and positive links between GD and alcohol use-related outcomes were reported in addition to other findings with non-significant associations between the two risk behaviors (Burleigh et al., 2019; Coëffec et al., 2015; Erevik et al., 2019; Estévez et al., 2017; Kotyuk et al., 2020; Männikkö et al., 2020; Marmet, Studer, Wicki, et al., 2019; Ream et al., 2011). However, these studies were heterogeneous in age-related (e.g., using adolescent vs. adult samples) and sampling-related characteristics (e.g., using representative, population-based samples vs. non-representative samples of gamers), in the measurement of alcohol use-related outcomes (e.g., measuring indicators of alcohol consumption vs. problematic symptoms of alcohol use) and in the type of co-occurrence (e.g., concurrent vs. simultaneous use of alcohol and gaming). Future meta-regression analyses are warranted to explore the background of these inconsistent findings between GD and alcohol use-related outcomes.

Bidirectional causal mechanisms might provide explanation for the comorbidity between AUD and gambling disorder, GD. Namely, alcohol consumption before and during gambling and gaming might promote decreased control over gambling and gaming behavior and might lead to negative consequences, in turn, those who experience adverse social and psychological consequences due to gambling and gaming behavior might start to use alcohol in order to dampen stress and negative emotional states due to these gambling or gaming problems (Cowlishaw et al., 2014). Moreover, it is also possible that shared and common genetic, neurobiological (e.g., areas responsible for reward functions, executive functions; for examples, see: II/Table 1.) and psychological precursors (e.g., impulsivity, negative affectivity, maladaptive emotion regulation, coping motivations behind these potentially addictive behaviors) between these disorders can account for the comorbidity between AUD and gambling disorder, GD (Burleigh et al., 2019; Dowling et al., 2015; Estévez et al., 2017; Ioannidis et al., 2019; Jauregui et al., 2016; Kotyuk et al., 2020; Marmet, Studer, Lemoine, et al., 2019; Paulus et al., 2018; Walther et al., 2012).

That is, building on these shared etiological factors between AUD and GD, multiple psychological mechanisms can be assumed which might account for the co-occurrence between AUD and GD. For example, based on the hypothesis of reward deficiency

syndrome, it might be possible that both alcohol use and gaming are used to gain pleasurable and rewarding experiences in order to compensate the hypodopaminergic dysfunction in the reward system (Blum et al., 2000; Kotyuk et al., 2020). In line with this, reward-seeking and enhancement motives are considered as important motivations for both alcohol use and gaming behavior (Cooper et al., 2015; Dong & Potenza, 2014). Higher levels of sensation seeking personality trait can also associated with both problematic alcohol use and gaming (though for GD the relationship is less consistent): those who are characterized with elevated sensation seeking might engage in these risk behaviors to experience pleasurable and arousal increasing stimuli (Gervasi et al., 2017; Hittner & Swickert, 2006; Walther et al., 2012). Moreover, both AUD and GD are characterized by impaired and impulsive decision making tendencies, such as difficulties to perform cognitive-behavioral control over use (e.g., disinhibition), preference of short-term gratifications over long-term pleasures (e.g., using alcohol or gaming despite negative consequences) (Coskunpinar et al., 2013; Dong & Potenza, 2014). Alternatively, similar negative reinforcement pathways were suggested for both AUD and GD which can also account for the co-occurrence between the two addictive disorders. Individuals characterized with higher rates of problematic alcohol use and gaming show elevated levels of emotion dysregulation and symptoms of negative affectivity (Dvorak et al., 2014; Estévez et al., 2017; Marmet, Studer, Lemoine, et al., 2019; Petit et al., 2015; van Rooij et al., 2014). The presence of negative affective and depressive symptoms – in absence of using effective and adaptive emotion regulation strategies – can motive the affected individuals to use alcohol and gaming to regulate and cope with negative emotional states. That is, coping motives are overlapping motivational dimensions for both risk behaviors (Cooper et al., 2015; Demetrovics et al., 2011).

II/Table 1. Examples for shared genetic and neurobiological characteristics between alcohol use disorder (AUD) and psychopathological disorders

	Genetic polymorphisms	**Neurobiological regions and functions**
Shared functions between AUD and attention deficit hyperactivity disorder (ADHD)	Polymorphisms in the ADGRL3 (Latrophilin 3, LPHN3) gene (Luderer et al., 2021)	Decreased dopamine release in limbic brain areas, reduced glutamatergic neurotransmission in the anterior cingulate cortex (ACC) (accounting for difficulties in reward systems) (Luderer et al., 2021)
Shared functions between AUD and substance use disorders (SUDs)	ADH and ALDH genes and group genes of CYP2B6 and CHRNA (Vanyukov et al., 2012)	Dopaminergic neurons in the ventral tegmental area (VTA) which are associated with forebrain regions (e.g., nucleus accumbens, amygdala, frontal cortex, limbic cortex); γ-aminobutyric acid (GABA) systems which can change the release of the dopaminergic neurons in the VTA (Vanyukov et al., 2012)
Shared functions between AUD and antisocial personality disorder (ASPD)	Genetic polymorphisms of monoamine oxidase-A (MAO-A) (Kolla & Wang, 2019)	Deficits in serotonin and dopamine mechanisms; decreased monoamine oxidase-A (MAO-A) level in the orbitofrontal cortex (OFC) and ventral striatum (VS); structural

		changes, such as hippocampal alterations and reduced amygdala volumes (Kolla & Wang, 2019)
Shared functions between AUD and gambling disorder	Polymorphisms of dopamine D2 receptor gene (DRD2), homozygote DdeI and 800T/C alleles of dopamine D1 receptor (DRD1) gene (Connor et al., 2002; Gyollai et al., 2014)	Altered activity in ventral striatum (VS) (explaining reward deficiency) (Romanczuk-Seiferth et al., 2015)
Shared functions between AUD and gaming disorder (GD)	Polymorphisms of the nicotinic acetylcholine receptor gene (CHRNA4) (Jeong et al., 2017)	Increased regional homogeneity (ReHo) in the posterior cingulate cortex (PCC); alterations in the anterior cingulate cortex (ACC) (Kim et al., 2015; Kuss et al., 2018)
Shared functions between AUD and major depressive disorder (MDD)	Variants of the muscarinic acetylcholine receptor M2 (CRHM2) gene (Boden & Fergusson, 2011)	Altered activity in the ventral striatum (VS) and frontostriatal connectivity characteristics (accounting for reward deficiency) (Becker et al., 2017)
Shared functions between AUD and anxiety disorders (ADs)	cAMP response element-binding protein (CREB)-target genes (e.g., the gene encoding neuropeptide Y [NPY]) (Pandey, 2003)	Neuroadaptations in the central amygdala (responsible for regulation of negative affectivity, stress responses) which is associated with regions responsible for executive functions (e.g., medial prefrontal

		cortex), emotion regulation (e.g., paraventricular hypothalamus), reward mechanisms (e.g., nucleus accumbens shell) (Anker & Kushner, 2019)
Shared functions between AUD and eating disorders (EDs)	Polymorphisms of the mu-opioid receptor (OPRMI) gene and the dopamine D2 receptor (DRD2) gene (Schreiber et al., 2013; Schulte et al., 2016)	Increased activation in the orbitofrontal cortex (OFC) as a reaction for alcohol and food-related cues; altered activity of mu opioid receptors (accounting for reward processing); low baseline serotonin level (accounting for craving), changes in glutamate activity in the nucleus accumbens (accounting for controlling alcohol use and eating behavior) (Schreiber et al., 2013; Schulte et al., 2016)
Shared functions between AUD and borderline personality disorder (BPD)	Polymorphisms of the dopamine D2 receptor (DRD2) gene (Nemoda et al., 2010)	Low serotonin levels; altered activity in the prefrontal cortex (accounting for impulsivity, emotional regulations) (Bornovalova et al., 2005)
Shared functions between AUD and bipolar disorder (BD)	Polymorphisms of the serotonin receptor and transporter gene of 5HT2C (Yasseen et al., 2010)	Mesolimbic dopaminergic pathways (e.g., from the ventral tegmental area (VTA) to the nucleus accumbens, prefrontal cortex); left

		frontal cortex (accounting for reward mechanisms) (Alloy et al., 2009)
Shared functions between AUD and schizophrenia	Polymorphisms of the brain-derived neurotrophic factor (BDNF), and specific Val/Val allele of catechol-O-methyltransferase (COMT) (Khokhar et al., 2018)	Impaired development in the associations between the hippocampus, prefrontal cortex and nucleus accumbens; dysfunction regarding dopaminergic activity in the ventral striatum (VS) (responsible for difficulties regarding reward and motivational processes) (Khokhar et al., 2018)

II/2/2. Comorbidity with internalizing psychiatric disorders

Increased rates of comorbidity and significant and positive associations were shown between AUD and various forms of internalizing psychiatric disorders, such as major depressive disorder (MDD) and different types of anxiety disorders (ADs), including generalized anxiety disorder (GAD), social anxiety disorder (SAD), panic disorders, specific phobias, obsessive-compulsive disorder (OCD) and posttraumatic stress disorder (PTSD) (Anker & Kushner, 2019; Boden & Fergusson, 2011; Castillo-Carniglia et al., 2019; Conner et al., 2009; Cuzen et al., 2014; Debell et al., 2014; Groenman et al., 2017; Hawn et al., 2020; Jane-Llopis & Matytsina, 2006; Lai et al., 2015; Morris et al., 2005; Schry & White, 2013; Smith & Randall, 2012; Straus et al., 2018). There is heterogeneity in the strength of the associations between AUD and different types of ADs (e.g., the specific link between AUD and specific phobias, PTSD seems more robust across different studies than considering the general diagnostic category of any ADs in addition to non-significant relationships in some studies between AUD and ADs) which might indicate distinct links between specific ADs and AUD (Castillo-Carniglia et al., 2019; Grant et al., 2015; Groenman et al., 2017).

However, MDD and different types of ADs can covary in a large degree (which is possibly explained by a transdiagnostic, internalizing disorders factor) and similar psychopathological mechanisms and pathways related to AUD were suggested across these internalizing disorders (Anker & Kushner, 2019; Kotov et al., 2017). There is a consensus among researchers that there are two main causal mechanisms which can explain the comorbidity between MDD, various types of ADs and AUD. First, the self-medication hypothesis proposes that symptoms of MDD and ADs have a predictive effect on alcohol use and problems via coping drinking motives. According to the self-medication hypothesis the affected individuals drink alcohol to alleviate and mitigate negative affective states, distress and symptoms related to MDD and ADs. Second, other findings suggested that the AUD can lead to the subsequent presence of MDD and various types of ADs. In the latter case, it was suggested that alcohol use can contribute to neurobiological changes (e.g., higher sensitivity to negative affectivity and stress-related effects due to withdrawal symptoms) and adverse social consequences (e.g., higher levels of experienced stress and internalizing symptoms due difficulties in interpersonal life, family, financial situation, and higher odds for experiencing traumatic events) which can subsequently to lead to the occurrence of internalizing disorders. Therefore, by unifying

these two pathways, bidirectional, circular and exacerbating relationships between AUD and internalizing disorders were suggested by previous research (Anker & Kushner, 2019; Boden & Fergusson, 2011; Conner et al., 2009; Groenman et al., 2017; Hawn et al., 2020; Hussong et al., 2011; Pedrelli et al., 2016; Smith & Randall, 2012; Straus et al., 2018; Turner et al., 2018). Moreover, shared genetic, neurobiological (e.g., functions related to stress response; for examples, see: II/Table 1.), environmental (e.g., early negative life and abusive experiences), and psychological influences (e.g., emotion regulation difficulties, anxiety sensitivity) also can explain the comorbidity between MDD, various types of ADs and AUD (Aldao et al., 2010; Anker & Kushner, 2019; Castillo-Carniglia et al., 2019; Garey et al., 2020; Hussong et al., 2011; Selby et al., 2008; Smith & Randall, 2012; Straus et al., 2018).

Overall, internalizing psychopathological symptoms have a key role in the psychopathology and development of AUD. According to the allostatic model's framework, the development process of more severe forms of AUD (i.e., consecutive stages of intoxication/binging, withdrawal/negative affect and preoccupation) are shaped by neurobiological and motivational changes. First, alcohol use is motivated by positive reinforcement processes (e.g., low level of negative affectivity, high levels of reward-seeking and enhancement tendencies), whereas in subsequent and more severe stages of AUD negative reinforcement mechanisms (i.e., drinking to cope with high levels of withdrawal symptoms, distress) becomes dominant (Anker & Kushner, 2019; Koob, 2011; Le Moal & Koob, 2007).

The broad diagnostic category of eating disorders (EDs) also related to the internalizing psychopathological spectrum (Eaton et al., 2015; Kotov et al., 2017) and incorporates multiple and distinct psychiatric disorders, such as anorexia nervosa (AN), bulimia nervosa (BN) or binge eating disorder (BED). Although these psychiatric disorders all show maladaptive characteristics related to eating behavior and cognitive-behavioral preoccupation with eating and body, different types of EDs are heterogeneous in psychopathological characteristics and mechanisms (e.g., restrictive characteristics vs. symptoms related to loss of control). For example, individuals with AN are characterized by low body mass index (BMI; $\leq$18.5 kg/m^2), high rates of weight loss, the presence of behaviors aiming to avoid weight gain (e.g., restrictive eating patterns, purging behaviors, excessive exercise) as well as by psychological preoccupation with the individual's weight and body (e.g., fear of weight gain, self-evaluation primarily focused on body

shape and weight). In the cases of both BN and BED, the presence of binge eating episodes (i.e., inability to regulate and/or stop eating behavior) and negative psychological consequences due to binge eating (e.g., distress, feelings of guilt) are diagnostic criteria. However, individuals with BN also show compensatory behaviors after episodes of binge eating to avoid weight gain (e.g., vomiting, excessive exercise), whereas these compensatory behaviors are not diagnostic features of BED (World Health Organization, 2018b). The meta-analytic findings showed positive, weak and moderate associations between AUD and different types of EDs, in addition to higher rates of comorbidity with AUD for those types of EDs which are characterized with binge eating and purging behavior compared to other forms of EDs (e.g., AN, EDs with restrictive features) (Bahji et al., 2019; Bogusz et al., 2021; Gadalla & Piran, 2007). A possible explanation for the stronger relationship between AUD and binge eating-related EDs can be that problematic alcohol use and binge eating show some overlap in symptomatic characteristics. Namely, loss of control over the behavior and craving are important diagnostic and psychopathological features for both AUD and binge eating (Ferriter & Ray, 2011; Schulte et al., 2016).

In the case of the comorbidity between AUD and EDs which are characterized by binge eating, common etiological risk mechanisms were identified: shared neurobiological correlates (e.g., areas related to reward processes and behavioral control; for examples, see: II/Table 1.) and psychological and affective characteristics (e.g., both disorders associated with elevated levels of internalizing symptoms, neuroticism, maladaptive emotion regulation, impulsivity as well as similar coping and reward-seeking motivations determine eating and alcohol use) can explain the positive correlation between AUD and EDs which are characterized by binge eating (e.g., BED, BN) (Ferriter & Ray, 2011; Schulte et al., 2016). That is, among those who show concurrent risk for AUD and binge eating it might be possible that both alcohol use and eating are used to gain pleasurable and rewarding experiences (to compensate the dysfunction in their reward system), to regulate and cope with negative affectivity and distressful emotional experiences (in absence of the use of adaptive emotion regulation strategies), whereas it is also possible that both alcohol use and binge eating can emerge due to risky and impulsive decision making and problems related to delaying short-term pleasures (especially in the presence of negative emotions) (Ferriter & Ray, 2011; Schulte et al., 2016). For example, maladaptive emotion regulation processes can suggest circular and bidirectional

associations between alcohol use and binge eating. As these individuals might show a lack of effective skills to regulate distressful emotional experiences, once negative emotional states are presented, they might use alcohol or eating behavior to cope with these emotions, however they might experience significant distress after alcohol use and binge eating behavior (e.g., feelings of guilt due to loss of control) which in turn can motivate for coping with negative emotions by using alcohol or by eating behavior.

Similar to the comorbidity with binge eating, in the case of the co-occurrence of AN and AUD shared psychological etiological factors were reported. For example, higher levels of impulsivity, MDD and borderline personality disorder (BPD) was presented for those individuals who show concurrent presence of AUD and AN (Baker et al., 2013). Bidirectional causal mechanisms were also suggested to explain the association between the two disorders. For example, individuals with AN might use alcohol in order to cope with psychical symptoms and psychological distress related to restrictive behaviors (possibly due to low rates of adaptive emotion regulation strategies), whereas individuals with AUD might show restrictive eating patterns to compensate weight gain due to excessive alcohol use (Baker et al., 2013).

II/2/3. Comorbidity with other psychiatric disorders

According to meta-analytic data, high levels of comorbidity and positive relationships were shown between AUD and borderline personality disorder (BPD) and bipolar disorder (BD) (Di Florio et al., 2014; Guy et al., 2018; Hunt et al., 2016; Messer et al., 2017). Both BPD and BD are characterized by a mix of externalizing and internalizing symptoms; thus, diverse pathways can explain the alcohol use of individuals with these disorders. Similar to the comorbidity models of AUD and ASPD, shared underlying psychological traits (e.g., impulsivity/disinhibition, neuroticism, emotion dysregulation) can account for the emergence of problematic alcohol use and bidirectional links between AUD and BPD (Castillo-Carniglia et al., 2019; Helle et al., 2019). In the case of the link between AUD and BD, existing findings suggested that manic episodes (characterized by high levels of impulsivity) are important risk factors of AUD, whereas self-medication tendencies (e.g., drinking alcohol to alleviate distress and negative affectivity) can also contribute to alcohol use among BD individuals. Common underlying genetic and neurobiological mechanisms were also assumed between AUD and BD (for examples,

see: II/Table 1.) (Balanzá-Martínez et al., 2015; Messer et al., 2017; Rich & Martin, 2014; Turner et al., 2018).

Finally, meta-analytic findings reported high prevalence rates of comorbidity between AUD and thought disorders, such as schizophrenia (Hunt et al., 2018). Multiple models were suggested to explain the comorbidity between AUD and schizophrenia. For example, some theories (i.e., "two-hit" and cumulative risk models) assume the interactive effect of genetic (for examples, see: II/Table 1.) and environmental influences (e.g., social adversities, childhood trauma) on the co-occurrence of AUD and schizophrenia, whereas other concepts (i.e., reward deficiency syndrome) highlight the role of shared neurobiological structures (for examples, see: II/Table 1.) between AUD and schizophrenia-related to reward dysfunction (Archibald et al., 2019; Khokhar et al., 2018).

II/3. Classification models of alcohol use and AUD

Alcohol users and individuals with AUD are highly diverse groups, substantial differences can be captured in both groups in the patterns of alcohol consumption, AUD symptomatology and in other factors which can influence the development and severity of AUD (e.g., family history, age of onset of use or AUD, comorbid SUDs or psychiatric disorders). Therefore, the goal of the alcohol classification models is to establish typologies of alcohol users or AUD. These models usually identify multiple classes of alcohol users, which are ideally characterized by high rates of within-class homogeneity and between-class heterogeneity. Based on these classifications, it is possible to expand our understanding on etiological and prognostic characteristics of problematic alcohol consumption, and to revise treatment and prevention services to become more tailored (Babor & Caetano, 2006; Hesselbrock & Hesselbrock, 2006; Leggio et al., 2009).

II/3/1. Empirically based classification models of AUD and alcohol use

Although the diagnostic taxonomies of DSM-5 and ICD-11 use different approaches to conceptualize AUD, they provide an important basis in determining different subgroups of problematic alcohol users. The DSM-5 uses the broad diagnostic category of AUD, which defines severity-based subtypes of AUD (i.e., mild, moderate, severe) and they differ by the level of total symptom severity and not by distinct types of symptoms (American Psychiatric Association, 2013). The ICD-11 proposes a hierarchical structure of problematic alcohol use: the categories of hazardous use, harmful use and dependence

represent alcohol users with increasing severity levels, but they also show qualitatively different symptom profiles (Saunders, 2017; World Health Organization, 2018b). Moreover, in the DSM-5-based diagnostic classification it is also important to consider "diagnostic orphans" as an at-risk, subthreshold subgroup of problematic alcohol users. These alcohol users present exactly one criteria of AUD; thus, they do not fulfil the criteria to receive the diagnosis of AUD but might show a risk for developing more severe alcohol problems subsequently (Hagman et al., 2014; Saunders et al., 2018). In the diagnostic hierarchy of the ICD-11 for problematic alcohol use it is more likely that the lower order categories can cover subthreshold alcohol users (e.g., it is possible to use the category of harmful use for someone being diagnostic orphan for alcohol dependence) (Saunders et al., 2018).

Similarly, previous empirically based classification models based on AUD symptoms were divergent: different typologies suggested subgroups of alcohol users with quantitatively or qualitatively different symptomatic profiles. On the one hand, numerous studies identified classes of alcohol users which differ in the overall severity level of AUD symptoms in clinical as well as in community samples. These classification solutions usually retained three or four subgroups (e.g., non-symptomatic, moderate symptomatic, moderate-high symptomatic, high symptomatic classes in Ko et al.'s classification model [2010]) with congruous symptomatic profiles indicating that these severity-based classes are distinguished by an increasing overall probability of the presence of AUD symptoms and not by the presence of specific symptoms. These quantitatively different typologies are in accordance with the AUD subtypes of the DSM-5, which can be placed along a severity continuum (Bucholz et al., 1996; Casey et al., 2013; Castaldelli-Maia et al., 2014; Ko et al., 2010; Shireman et al., 2015). On the other hand, some studies also identified classes of AUD, which showed distinct symptomatic profiles with high probabilities to experience specific types of symptoms. For example, some typologies included symptomatic subgroups of AUD, which were characterized primarily by drinking alcohol larger amounts than intended and party by hazardous alcohol use (Castaldelli-Maia et al., 2014; McBride et al., 2011), in addition to subgroups of AUD with disparate patterns of alcohol dependence and harmful consequences (e.g., the presence of dependence symptoms in the absence of harmful consequences) (Rist et al., 2009). That is, these classes showed qualitatively different (and not primarily severity-based) symptomatic profiles compared to other classes (e.g., non-symptomatic, high symptomatic).

However, other classification models proposed that different measures of alcohol consumption (e.g., frequency and quantity of use, HED) should be also considered as indicators of alcohol typologies in addition to symptoms of AUD. Involvement of alcohol consumption indicators in classification can have a capacity to explore subgroups of at-risk alcohol users in a wider range of severity levels, particularly at lower levels of the problematic alcohol use spectrum (e.g., differentiate heavy alcohol drinking groups with and without the presence of AUD symptoms) (Jackson, Bucholz, et al., 2014; Kuvaas et al., 2014; Smith & Shevlin, 2008). Studies with this approach repeatedly discriminated classes of alcohol users with an increasing risk for harmful alcohol consumption, for example, by identifying classes of (i) low-risk (e.g. alcohol users with infrequent and light consumption levels), (ii) moderate-risk (e.g., regular and/or heavy episodic users who experience AUD symptoms with light-medium probabilities) and (iii) high-risk alcohol users (e.g., high levels of alcohol consumption, AUD symptoms and negative consequences) (Jackson, Bucholz, et al., 2014; Sacco et al., 2009; Smith & Shevlin, 2008). However, simultaneous use of classification indicators of alcohol consumption and AUD symptoms can also allow to distinguish at-risk alcohol drinking classes with specific and distinct profiles, such as identifying those who experience negative consequences (but not dependence symptoms) due to heavy alcohol use or even at lower consumption levels (Smith & Shevlin, 2008).

Considering the high levels of alcohol consumption, alcohol use-related problems and burden in the region of Central-Eastern Europe (CEE) and specifically in Hungary (Shield et al., 2020; World Health Organization, 2018a), it would be important to identify latent classes of alcohol users at population level based on indicators of alcohol consumption and alcohol use-related negative consequences (see: Aim 1/a). This approach can more accurately describe subgroups of alcohol users with different severity levels and identify more specifically at-risk and/or problematic alcohol drinker classes in the general adult population, which can be informative for prevention and intervention programs as well as for alcohol-related policies. To the Author's best knowledge, previous studies did not investigate alcohol user typologies in representative adult samples in CEE countries as well as specifically in Hungary. Moreover, considering the high rates of comorbidity between AUD and other psychiatric disorders and its function in alcohol classification models (see further in the next subsection), investigation of the associations between latent classes of alcohol users and various dimensions of psychopathological symptoms

can contribute to describing more precisely characteristics of alcohol drinking subgroups (see: Aim 1/b).

II/3/2. The role of co-occurring psychopathological symptoms and diagnoses in classification models

However, it is important to note that several typologies were not restricted to classify alcohol users based on indicators of alcohol consumption and AUD symptoms, rather considered a wider range of risk characteristics (e.g., family history, age of onset of use or AUD, comorbid SUDs or psychiatric disorders) in addition to alcohol use and problems-related indicators.

Binary classification models suggested that individuals with AUD can be classified into two, severity-based subgroups (Leggio et al., 2009). The less severe subgroups (i.e., Jellineck's Gamma type, Cloninger et al.'s Type I, Babor et al.'s Type A) in these typologies were characterized by lower levels of alcohol use and alcohol use-related problems, the capacity to show temporary abstinence from alcohol use in addition to the inability to control alcohol use during consumption, later onset of AUD, self-medication tendencies, lower levels of family history of AUD and childhood risk factors, high levels of harm-avoidance and reward dependence, lower levels of sensation-seeking and comorbid psychopathology, more optimal treatment outcomes. The more severe subgroups (i.e., Jellineck's Delta type, Cloninger et al.'s Type II, Babor et al.'s Type B) in these typologies showed elevated levels of alcohol use and alcohol use-related problems, the lack of capacity to abstain from alcohol use, earlier onset of AUD, motivation to enhance pleasures by drinking, higher levels of family history of AUD and childhood risk factors, decreased levels of harm-avoidance and reward dependence, higher levels of sensation-seeking and comorbid psychopathology (e.g., antisocial acts) and more adverse treatment outcomes (Babor & Caetano, 2006; Leggio et al., 2009). However, it is important to note that there are considerable differences between these previous binary typologies of AUD and more recent, empirically-based models of AUD and alcohol use. First, the classification models of Jellineck, Cloninger et al., and Babor et al. suggest that different subgroups of AUD show qualitatively distinct symptom and risk profiles, whereas more recent, alcohol use- and AUD symptom-based classification models rather suggested that alcohol problems can be placed along a severity continuum and AUD subgroups show severity-based differences (Bucholz et al., 1996). Second,

these previous binary typologies of AUD used different classification approaches compared to the previously reviewed, empirically-based models of AUD and alcohol use. Some of these previous typologies were derived from clinical observations and intuitions (e.g., Jellineck's classification model), whereas in other empirically tested models the selection of the indicator variables and the categorization of AUD individuals were strongly grounded in theoretical considerations (e.g., Cloninger et al.'s model was derived from a personality theory of AUD individuals, Babor et al.'s classification model built on the concept that differences between AUD individuals can be explained by multiple and interrelated dimensions, such as genetic-, biological-, psychological and sociocultural variables) (Epstein et al., 2002; Jemberie et al., 2020; Leggio et al., 2009). That is, these typologies categorized individuals with AUD based on a larger and more complex set of risk characteristics, whereas several, more recent typologies of alcohol consumption and AUD were less capable to explore the multidimensionality in risk profiles of AUD individuals.

Classification models with more than two subtypes of AUD were also suggested as dichotomous typologies might mask significant differences and fail to capture the complete heterogeneity within the AUD population (Leggio et al., 2009). For example, in Lesch et al.'s classification model four subgroups of AUD were differentiated. Individuals in the Type I class ('model of allergy') shows high levels of withdrawal symptoms and craving, they tend to drink in order to cope with withdrawal symptoms, and there is a presence of family history of AUD. Type II ('model of anxiety or conflict') is characterized by the preference to drink alcohol because of its sedative effects, shows negative behavioral consequences while drinking alcohol. Problematic alcohol users in Type III ('model of depression') can present co-occurring affective disorders and aggressive antisocial acts, their drinking is motivated by the anti-depressant effect of alcohol, and there is a presence of family history of AUD and affective disorders. Finally, Type IV ('model of adaptation') shows pre-morbid neurological difficulties and disorders, behavioral and social difficulties in childhood and adolescence (Leggio et al., 2009). In some other cases, the identified classes of AUD overlapped and showed similar characteristics across the different classification models with usually three-to-five classes. Based on these findings the following subtypes of AUD were distinguished repeatedly: (i) clusters where members showed low-risk alcohol consumption and low-severity of alcohol-related impairment, later onset of AUD, lower severity levels of comorbid

psychopathological symptoms, (ii) chronic severe subtypes which consisted of participants with high-very high levels of AUD symptom endorsement, family history of AUD, high levels of comorbid psychiatric problems, (iii) negative affect classes were characterized by high rates of depressive and anxiety symptoms as well as AUD symptomatic severity; and (iv) antisocial subtypes which had an excessive degree of alcohol consumption, adverse consequences due to drinking and antisocial behavior and externalizing psychopathology, earlier onset of AUD and higher odds for familial AUD (Del Boca & Hesselbrock, 1996; Hesselbrock & Hesselbrock, 2006; Hildebrandt et al., 2017; Leggio et al., 2009; Moss et al., 2007; Windle & Scheidt, 2004).

Overall, the previously reviewed classification solutions highlighted that co-occurring externalizing and internalizing psychopathology can be considered an important and core element of AUD subtypes and can explain the heterogeneity among individuals with AUD (Hildebrandt et al., 2017). In line with this, other classification models attempted to account for the differences in severity levels and distinct constellations of comorbid psychiatric disorders among individuals with AUD. These studies identified subgroups of AUD by considering exclusively co-occurring psychiatric disorder presence (Glass et al., 2014; Müller et al., 2020; Sintov et al., 2010; Urbanoski et al., 2015). However, these classification models were heterogeneous in terms of the applied samples (e.g., treatment-seeking vs. non-treatment-seeking samples of AUD) and indicators (e.g., absence of externalizing disorders in Urbanoski et al.'s classification model). Four classes of AUD were repeatedly identified across the studies: (i) classes which were characterized by overall low probability of the presence of comorbid psychiatric disorders irrespective the internalizing or externalizing nature of the disorders, (ii) classes with moderate-high levels of comorbid internalizing psychiatric disorders (e.g., MDD, phobias, PTSD, GAD) and low levels of comorbid externalizing psychiatric disorders, (iii) subgroups which showed primarily comorbid externalizing psychiatric disorders (e.g., ASPD, SUDs) in addition to low rates of comorbid internalizing psychiatric disorders, and (iv) classes with moderate-high levels of both internalizing and externalizing comorbid psychiatric disorder presence. That is, these findings indicated that some subgroups of AUD were discriminated by distinct and specific profiles of comorbid psychiatric disorders (e.g., qualitative differences in comorbidity patterns between subgroups with predominantly internalizing and predominantly externalizing psychopathology), whereas other subgroups were differed along a continuum of comorbid psychopathological severity

(e.g., severity-based and quantitative differences between not-affected and highly-affected classes) (Glass et al., 2014; Müller et al., 2020; Sintov et al., 2010; Urbanoski et al., 2015). Other classification models which attempted to classify individuals with AUD based on co-occurring psychopathological symptom levels also proposed severity-based discrimination between classes ranging between mild and severe psychopathological symptom levels (Villalobos-Gallegos et al., 2017).

Previous latent class analytic findings revealed that latent classes of AUD might differ in AUD prognosis which might be accounted at least partly for comorbid psychiatric disorders (e.g., higher rates of remaining alcohol dependence and involvement in treatment for a subgroup with high severity of AUD and comorbidity psychiatric disorders) (Moss et al., 2010). However, to the Author's best knowledge, existing studies which aimed to identify subgroups of AUD based on symptomatic levels or presence of comorbid psychiatric disorders used cross-sectional design and did not focus on the changes of comorbid psychiatric disorder severity (e.g., due to attendance in a treatment program) in different latent classes of AUD. Previous meta-analytic findings suggested that combined therapeutic approaches can reduce psychopathological symptoms among individuals with comorbid AUD and other psychiatric disorders (Hobbs et al., 2011; Riper et al., 2014; Roberts et al., 2015). Examination divergences between subgroups of AUD not only in terms of comorbid psychiatric symptom severity and specific symptomatic constellations, but also considering changes in symptomatic levels of comorbid psychiatric disorders can increase theoretical and treatment-related knowledge on subgroups of AUD (e.g., prognosis and recovery of latent classes of AUD with different comorbid psychopathological severity levels, differences in psychopathological change and stability between subgroups of AUD due to treatment attendance) (see: Aim 2/a).

Specifically, regarding to the latter aim of the dissertation, it was aimed to examine differences in terms of psychopathological symptom levels and changes among AUD individuals attending a treatment program of the Minnesota Model (MM). The MM of treatment is a structured, residential, and community-based approach which builds on the twelve-step principles of the Alcoholics Anonymous (AA) as well as on group-psychotherapeutic forms. That is, the MM treatment approach harmonizes professional and AA-based treatment elements (Anderson et al., 1999). This abstinence-centered treatment program strongly advocates AA attendance and twelve-step-related progress of patients both during and after the program (Anderson et al., 1999; Borkman et al., 2007).

Previous studies demonstrated that involvement in MM treatment programs have a positive effect on drinking-related outcomes (Gallagher et al., 2018; Grønbaek & Nielsen, 2007; Stinchfield & Owen, 1998). Moreover, successful attendance in MM programs has been associated with improved rates of mental wellbeing and attenuation of depressive symptoms (Andó et al., 2016; Berglund et al., 2004). Similar to the MM, the Twelve Step Facilitation (TSF) treatment simultaneously relies on professional therapeutic elements (e.g., the use of standardized manuals, treatment process is guided by professional staff) and AA-related twelve step progress (Project Match Research Group, 1998). According to the results of the Project Match study, the TSF treatment showed similar levels of beneficial impact on drinking-related outcomes compared with Motivational Enhancement Therapy and Cognitive Behavioral Therapy (Project Match Research Group, 1998). Overall, existing empirical findings demonstrated that participation in twelve-step-based programs can contribute to improvement in depressive and social anxiety symptoms among patients with AUD and co-occurring psychiatric disorders (Kelly et al., 2012; Timko et al., 2013). Mediational mechanisms were suggested which can explain the role of twelve-step involvement on alcohol use- and mental health-related outcomes: the beneficial effect of the attendance in a twelve-step program on alcohol use-related outcomes is being mediated by the attenuation of psychopathological symptoms, such as depression (Wilcox & Tonigan, 2018).

II/3/3.Classification models of alcohol use among adolescents

Adolescence is considered a critical period for alcohol use as various forms of consumption can develop during this stage: initiation of use, frequent and/or excessive episodic use, and experiencing negative consequences and problems due to use. Therefore, various alcohol consumption patterns can indicate at-risk alcohol use, such as frequent and regular use, HED or the presence of alcohol use-related problems (Bräker et al., 2015; Thompson et al., 2014).

Classification models of adolescent alcohol use can assist in identifying at-risk subgroups of adolescent alcohol users who show risky or hazardous alcohol consumption patterns. Previous empirical adolescent alcohol typologies distinguished various classes of alcohol users along a severity continuum of alcohol involvement ranging between abstainers and problematic alcohol users. These studies showed some consistency in the identified subgroups, as they repeatedly discriminated (i) classes of abstainers who did not use

alcohol, (ii) light or mild drinkers who were characterized by experimental or low-frequency use with low quantity of consumption and low levels of alcohol-related problems, (iii) frequent or moderate alcohol users with regular alcohol consumption patterns, lower rates of HED and very low levels of alcohol-related problems, (iv) heavy drinkers' classes who showed high levels of frequent alcohol drinking and/or HED in addition to low-moderate odds for experiencing negative consequences due to alcohol use, and (v) subgroups of problematic drinkers who were characterized by high levels of frequency and quantity of alcohol use, HED and alcohol-related problems. That is, these alcohol using classes showed an increased risk for adverse alcohol use-related outcomes (Bräker et al., 2015; Dauber et al., 2009; Davoren et al., 2016; Gohari et al., 2020; Jackson, Denny, et al., 2014; Percy & Iwaniec, 2007).

However, several taxonomies were not restricted to classify adolescents solely based on their alcohol consumption patterns, rather included the use of other psychoactive substances as well in the classification models (e.g., identifying subgroups based on alcohol, tobacco and cannabis use) (Halladay et al., 2020; Tomczyk et al., 2016). Studies that used such an approach also discriminated predominantly alcohol user classes with different severity levels (e.g., experimental users, binge drinkers); however, considering a wider range of psychoactive substances can allow to identify subgroups with polysubstance use patterns (e.g., adolescents with concurrent use of alcohol and cannabis or other illicit drugs) (Halladay et al., 2020; Tomczyk et al., 2016). To the best of the Author's knowledge, little is known about the latent classes of alcohol and illicit drug use among adolescents in CEE countries and specifically in Hungary (Bräker et al., 2015; Göbel et al., 2016; Halladay et al., 2020; Tomczyk et al., 2016). Describing classes of polysubstance users and alcohol users with increasing severity levels in a representative adolescent sample might help to identify more accurately subgroups of adolescents with at-risk substance use patterns which can have implications for adolescent substance use-related policies, prevention and intervention programs (see: Aim 3/a).

Previous latent class analytic findings consistently showed that polysubstance users show high levels of externalizing and internalizing psychopathological symptoms and behaviors (e.g., antisocial behaviors, ADHD, depressive symptoms) compared to subgroups with lower levels of substance use (Connell et al., 2010; Cranford et al., 2013; Halladay et al., 2020; Riehman et al., 2009; Shin et al., 2010; Snyder & Smith, 2015; Tomczyk et al., 2016). However, to the best of the Author's knowledge, existing studies

did not examine the association between latent classes of alcohol and illicit drug use and potentially addictive behaviors, such as GD. As discussed earlier, previous studies reported contradictory findings on the association between alcohol use and GD (Burleigh et al., 2019). Therefore, it might be possible that a more accurate interpretation can be obtained on the co-occurrence of alcohol use and GD by comparing levels of GD symptom severity and specific GD criteria between empirically-based subgroups which simultaneously considers alcohol use and illicit drug use. This approach can identify specific and distinct substance use patterns that are characteristic of adolescents with elevated risk for GD. For example, building on that literature which revealed shared underlying neurobiological and psychological risk mechanism between AUD, SUDs and GD (Estévez et al., 2017; Marmet, Studer, Lemoine, et al., 2019; Walther et al., 2012), it might be possible that the membership of a polysubstance users' class is positively associated with higher levels of GD, indicating a cumulation of problem behaviors in a subgroup of at-risk adolescents (see: Aim 3/b).

II/4. The role of drinking motives on the relationships between psychopathological symptoms and alcohol use-related outcomes

II/4/1. The motivational model of alcohol use

The motivational model of alcohol use (Cox & Klinger, 1988) conceptualizes alcohol drinking as a functional behavior: individuals drink alcohol to experience particular expected effects. In this framework, drinking motives (i.e., reasons of alcohol use) are perceived as the most proximal predictors of decision and behavior of alcohol drinking, and they can mediate the effects of other, more distal determinants on alcohol use-related outcomes. These distal antecedents cover a broad range of individual and environmental characteristics (e.g., biochemical responsiveness to alcohol use, personality factors, previous experiences with alcohol drinking, cultural and group-specific norms of alcohol use, increased or limited availability of alcohol use, presence of alcohol drinking friends) and they determine individuals' expectations on psychophysiological and social outcomes of alcohol use (i.e., one's expectations regarding the behavioral, emotional and cognitive consequences of alcohol use). Outcome expectancies of alcohol use provide a foundation for drinking motives in two interrelated dimensions: valence and source of the expected effects. The valence of the expected consequences of alcohol use represents that alcohol use is either motivated by enhancing and approaching positive outcomes or by

avoiding and reducing negative effects. The source of the expected consequences of alcohol use can be internal, self-directed, or external with a focus on the social environment. Four distinct types of drinking motives can be constructed based on these two factors: (i) enhancement (positive valence with an internal source, e.g., drinking in order to experience and enhance pleasurable affective and psychophysiological states), (ii) social (positive valence with an external source, e.g., drinking in order to make a social gathering more pleasurable), (iii) coping (negative valence with an internal source, e.g., drinking in order to alleviate and mitigate negative emotional states) and (iv) conformity motives (negative valence with an external source, e.g., drinking in order to avoid social disapproval or rejection) (Cooper et al., 2015; Kuntsche et al., 2005).

Different drinking motives show specific and distinct links with alcohol consumption-related outcomes (e.g., frequency, quantity) and alcohol use-related problems (Cooper et al., 2015). Meta-analytic findings showed significant and positive bivariate associations between all four drinking motives and outcomes of alcohol consumption and problems; however, the magnitude of the associations differed across different motives (e.g., strongest relationships with enhancement motives and weakest associations with conformity motives) (Bresin & Mekawi, 2021). However, the role of drinking motives with an internal source was highlighted if the simultaneous effect of each factor of drinking motives were controlled (Bresin & Mekawi, 2021; Cooper et al., 2015). Enhancement motives presented the strongest associations with alcohol consumption-related outcomes, whereas alcohol use-related problems were linked to the enhancement and coping motives with the highest levels of magnitude (Bresin & Mekawi, 2021). Meta-analytic findings and literature reviews suggested different indirect mechanisms between internal drinking motives and outcomes of alcohol use: enhancement motives had a positive effect on alcohol use-related problems via increased levels of alcohol use, whereas the positive relationship between coping motives and alcohol use was mediated by alcohol use-related problems (Bresin & Mekawi, 2021; Cooper et al., 2015).

II/4/2. Association between psychopathological symptoms and drinking motives

Existing research also reported that different drinking motives are associated with distinct patterns of distal psychological antecedents (Cooper et al., 2015; Kuntsche et al., 2006b). Several psychopathological symptoms and personality factors (which can be precursors or determinants of psychiatric disorders) showed significant and positive correlations

with drinking motives. These psychopathological variables most consistently were linked to drinking motives with internal source (i.e., implicating that alcohol might be used in order to regulate emotional states in the absence of adaptive emotion regulation means) and with negative reinforcement mechanisms (i.e., implicating that alcohol might be used as a means to avoid negative intra- and interpersonal consequences). Psychopathological variables most consistently were associated with coping, enhancement and conformity motives (Cooper et al., 2015).

For example, higher levels of coping motives were associated with elevated rates of features associated with negative affectivity, affective dysregulation and internalizing symptomatology, such as neuroticism, symptoms of MDD, depressive episode in BD, GAD, PTSD, SAD, OCD and BPD, low self-esteem, anxiety sensitivity, negative urgency; as well as with symptoms of other psychiatric disorders, such as ADHD and problematic eating behaviors (e.g., BN, BED) (Allan et al., 2015; Bakhshaie et al., 2021; Bravo et al., 2018; Cooper et al., 2015; Grazioli et al., 2019; Luce et al., 2007; Meyer et al., 2012; Schry & White, 2013; Tragesser et al., 2007; Vest et al., 2018). Enhancement motives showed positive links with personality characteristics of sensation seeking, reward sensitivity, low self-control, low aggression suppression, positive urgency, in addition to positive associations with psychopathological features with impulsive characteristics, such as ADHD symptoms, manic episode in BD, binge eating, features of cluster B personality disorders (e.g., antisociality, impulsivity, affective instability) (Cooper et al., 2015; Grazioli et al., 2019; Kuntsche et al., 2006b; Meyer et al., 2012; Tragesser et al., 2007; Trojanowski et al., 2019). In the case of conformity motives, significant and positive relationships were shown with personality and psychopathology factors which can indicate interpersonal difficulties, such as symptoms of SAD and BPD, anxiety sensitivity and attachment anxiety (Cooper et al., 2015; Kaufman et al., 2020; Schry & White, 2013). Moreover, in line with the assumptions of the motivational model of alcohol use, numerous studies reported the mediating effects of drinking motives between distal predictors of psychopathological symptoms and alcohol use-related outcomes. That is, these mediation models suggested that higher levels of a given psychopathological characteristic (e.g., depressive symptoms) can lead to elevated rates of a particular drinking motive (e.g., coping drinking motives) which can subsequently contribute to more adverse outcomes of alcohol use (e.g., negative consequences due to drinking) (Allan et al., 2015; Bakhshaie et al., 2021; Bravo et al., 2018; Cooper et al.,

2015; Grazioli et al., 2019; Kaufman et al., 2020; O'Hare & Sherrer, 2011; Terlecki & Buckner, 2015; Tragesser et al., 2007; Vest et al., 2018).

Regarding the link between psychopathological characteristics and alcohol use outcomes via drinking motives, it is also important to consider the roles of transdiagnostic psychological constructs. Emotion regulation, as a transdiagnostic psychopathological construct (Aldao et al., 2010), cover those "processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions" (Gross, 1998, p. 275). Numerous previous findings examined how drinking motives mediate effects on outcomes of alcohol use from constructs of emotion dysregulation, such as difficulties in emotion regulation (e.g., limited access to emotional regulation strategies, non-acceptance of emotional responses, difficulties engaging in goal-direct behavior when experiencing negative affectivity), alexithymia (e.g., difficulty identifying and describing feelings), affective lability, negative urgency (i.e., the tendency to act rashly when experiencing negative affective states) and maladaptive emotion regulation strategies (e.g., rumination which represents a repetitive concentration on signs of negative affectivity, distress). Overall, these findings were consistent that effects of emotion regulation difficulties on alcohol use-related outcomes are mediated by coping motives: higher levels of emotion dysregulation can lead to elevated levels of coping drinking motives which in turn can predict more adverse outcomes of alcohol use (e.g., alcohol-related problems) (Adams et al., 2012; Aurora & Klanecky, 2016; Bravo et al., 2018; Cooper et al., 2015; Lyvers et al., 2018; Simons et al., 2017). Moreover, the mediating effect of enhancement motives was also showed between negative urgency, difficulties in emotion regulation and problematic alcohol use (Adams et al., 2012; Aurora & Klanecky, 2016; Simons et al., 2017). That is, it might be possible that an alcohol using person who lacks using adaptive emotion regulation strategies might drink alcohol as a dominant way to regulate positive and negative emotional states (Cooper et al., 2015). In addition to emotion regulation, the links between drinking motives and transdiagnostic constructs related to self-regulation, self-control and disinhibition were also examined extensively (Cooper et al., 2015). Numerous studies demonstrated pathways from impulsivity and conscientiousness on problematic alcohol use via enhancement and coping motives. That is, higher levels of impulsivity (e.g., lack of premeditation) and lower levels of conscientiousness can lead to higher levels of enhancement and coping motives which subsequently can lead to more adverse outcomes of alcohol use (Adams

et al., 2012; Cooper et al., 2015; Jones et al., 2014; Loose et al., 2018; Magid et al., 2007). However, the theoretical background regarding the indirect pathway from self-regulation on alcohol use via drinking motives is not completely established yet. In line with this, other studies did not report significant indirect effects via drinking motives on the link between self-regulation and alcohol use (Cooper et al., 2015).

The present dissertation examined further the role of drinking motives on the relationships between psychopathological symptoms and alcohol use-related outcomes in two aspects. First, it has been noted that more research would be needed to examine the motivational background and pathways of individuals with clinically diagnosed AUD (Cooper et al., 2015). Although existing literature data suggested that different types of drinking motives are associated with distinct profiles of determinants (e.g., personality, psychopathological, contextual factors), these findings predominantly assessed symptoms and features of psychopathologies separately. That is, little is known about motivational differences in terms of alcohol use between subgroups of AUD with distinct profiles of co-occurring psychopathological symptoms. For example, comparing subgroups of AUD with different severity levels (e.g., subgroups with low vs. high overall levels of symptom severity) as well as with different qualitative constellations of psychopathological symptoms (e.g., subgroups with predominantly internalizing vs. externalizing psychopathologies) might allow to investigate more specifically motivational pathways of individuals with clinical-level of AUD and can contribute to existing theoretical (i.e., self-medication hypothesis, allostatic model) and empirical knowledge (Cooper et al., 2015; Koob, 2011; Le Moal & Koob, 2007; Turner et al., 2018) (see: Aim 2/b).

Second, previous studies highlighted that similar motivational processes determine some forms of eating behavior (e.g., binge eating) and alcohol use: these behaviors can be used as a function of emotional regulation, namely, by these behaviors it is possible to enhance and experience positive emotions, whereas it is also possible to mitigate and cope with negative affective states (Ferriter & Ray, 2011; Schulte et al., 2016; Trojanowski et al., 2019). Similar motivational background of alcohol use and eating was also highlighted by previous findings, which identified shared psychological and affective characteristics between them (e.g., negative affectivity, emotion regulation difficulties, reward seeking tendencies) (Ferriter & Ray, 2011; Schulte et al., 2016). In line with these, individuals with different forms of EDs (e.g., BN, BED) consistently showed higher levels of coping

motives and enhancement motives of alcohol use (Anderson et al., 2006; Luce et al., 2007; Mikheeva & Tragesser, 2016; Trojanowski et al., 2019). However, to the Author's best knowledge, existing studies did not explore whether drinking motives with internal source (i.e., coping and enhancement motives) mediate the relationship between symptoms of EDs and alcohol use. Based on the assumptions of the motivational model of alcohol use, testing such a mediation pathway might contribute to a better understanding of the co-occurrence between risky alcohol use and eating behavior (see: Aim 4).

III. Aims of the dissertation

The present dissertation aimed to examine co-occurrence of psychopathological symptoms and outcomes of alcohol use from different perspectives. Specifically, the present dissertation aimed to (i) identify empirically-based subgroups of alcohol users in clinical and general adult and adolescent samples and to examine their associations with various dimensions of psychopathological symptoms, and (ii) to investigate the role of drinking motives on the relationships between psychopathological symptoms and outcomes of alcohol use. Considering the reviewed literature gaps and potential contributions to the current literature, four studies were performed in the present dissertation with the following aims.

Study 1 was aimed to explore distinct subgroups of alcohol users in a representative Hungarian adult sample (Aim 1/a) and to examine associations between these alcohol drinking latent classes and various dimensions of psychopathological symptoms (Aim 1/b).

The aim of Study 2 was to identify latent classes of AUD inpatients in a treatment program with distinct profiles and change patterns of psychopathological symptoms (Aim 2/a) and to investigate differences between these subgroups in terms of drinking motives (Aim 2/b).

Study 3 was aimed to identify latent classes of alcohol and illicit drug use in a representative Hungarian adolescent sample (Aim 3/a) and to compare these substance-using subgroups in terms of GD symptom severity and criteria (Aim 3/b).

Finally, Study 4 aimed to test the mediating role of drinking motives with internal source (i.e., coping and enhancement motives) on the relationship between symptoms of EDs and alcohol use among adolescents (Aim 4).

IV. Study 1: An Empirically Based Typology of Alcohol Users in a Community Sample Using Latent Class Analysis[6,7]

IV/1. Abstract

Background: Different classification models have been proposed to explain the heterogeneity of alcohol-related problems in general populations. Such models suggest quantitatively or qualitatively different symptom endorsement characteristics between subgroups of alcohol drinkers.

Objectives: The present study aimed to identify homogenous subgroups of drinkers in a general population sample in addition to examining the relationship between the subgroups and psychopathological symptoms.

Method: Data of past-year alcohol users (N = 1520) were analyzed from the nationally representative sample of the National Survey on Addiction Problems in Hungary 2015 (NSAPH 2015). Latent Class Analysis (LCA) was conducted to identify subgroups of drinkers based on the dichotomous indicator items of the Alcohol Use Disorders Identification Test (AUDIT) questionnaire. Multinomial logistic regression and multiple comparisons were performed to explore the relationship between latent classes and socio-demographical variables and psychopathological symptoms.

Results: LCA suggested a three-class model: 'Light alcohol drinkers' (71.6%), 'Alcohol drinkers with low risk of dependence' (19.3%) and 'Alcohol drinkers with severe dependence symptoms' (9.1%). More severe subgroups showed significantly higher level of anxiety, depression, hostility, obsessive-compulsivity, interpersonal sensitivity, and

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psychiatric or AUD-related treatment involvement. Male gender, younger age, lower level of educational achievement, and earlier onset of the first alcoholic drink were associated with membership of more severe subgroups.

Conclusions: The present results indicated that severity-based subgroups of drinkers can be discriminated. Approximately 9% of the alcohol users showed severe symptoms of alcohol dependence. The present data also supported the association between more severe forms of alcohol consumption, and internalizing and externalizing characteristics. Although the two at-risk classes of alcohol drinkers did not differ in terms of alcohol consumption-related measures, they were distinguished by the level of harmful consequences due to alcohol use, psychopathological symptoms and psychiatric treatment history.

Keywords: Latent Class Analysis (LCA); Alcohol Use Disorders Identification Test (AUDIT); Alcohol Use Disorder (AUD); Alcohol consumption; Alcohol psychopathology; National survey.

IV/2. Introduction

Excessive alcohol consumption is associated with several adverse physical and psychological health outcomes, as well as social harms (World Health Organization, 2014). From a public health perspective, it is essential to identify not only those who demonstrate harmful alcohol use patterns, but also those who might be at-risk of developing adverse alcohol-related consequences subsequently (Babor & Robaina, 2016). Furthermore, excessive alcohol consumption contributes to substantial alcohol attributable burden in Hungary. Compared with the European average levels, high prevalence of alcohol use disorders (17.7%), alcohol dependence (9.4%), and high rates of liver cirrhosis-related mortality (age-standardized death rate for males and females: 57.0 and 16.8, respectively) has been presented in Hungary (World Health Organization, 2014). Due to these data and the lack of comprehensive national alcohol policy, there is a need to greater understand drinking patterns and alcohol-related problems in Hungary in a more detailed way.

Theoretical and empirically-based classification models aim to identify distinct and homogenous subgroups of drinkers which are both clinically meaningful and stable over

time. Based on such classifications, it is possible to isolate differences among subgroups of individuals with alcohol use disorder (AUD) in terms of drinking patterns, associated adverse consequences, development of AUD, and comorbid substance use disorders or psychiatric symptoms. Although some of the identified subgroups show substantially similar characteristics across different models, none of the previous classification attempts have yet been considered as generally adequate in research and clinical environments (Babor & Caetano, 2006; Casey et al., 2013).

Binary classification models have identified a severely and a mildly affected group of AUD patients based on psychopathological and AUD-related vulnerability indicators (Babor & Caetano, 2006). However, dichotomous models arguably have a restricted capability in providing a precise distinction between possible classes. Therefore, various multiclass models have also been assumed (Leggio et al., 2009). Current taxonomies consistently posit four alcohol drinking subgroups: low-severity, chronic severe, negative affect, and antisocial subtype (Del Boca & Hesselbrock, 1996; Hesselbrock & Hesselbrock, 2006; Hildebrandt et al., 2017). Additionally, these models highlight the role of comorbid externalizing and internalizing psychopathological symptoms among AUD individuals. Other typologies suggested that AUD can be examined on a continuum of severity, including subgroups that are likely to vary from each other quantitatively. This latter approach corresponds with the unidimensional concept in the latest (fifth) edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (Bucholz et al., 1996; Jackson, Bucholz, et al., 2014).

Previous classification models have predominantly relied upon clinical samples of AUD patients. However, typologies which focus on general population samples may cover a wider range of AUD severity compared to models based on clinical samples. By including non-treatment seeking individuals in classification models, they could more accurately represent the less severe forms of AUD (Leggio et al., 2009; Moss et al., 2007). Various studies which have used general population or community-based samples have identified severity-based subgroups of drinkers (Casey et al., 2013; Castaldelli-Maia et al., 2014; Jackson, Bucholz, et al., 2014; Ko et al., 2010; McBride et al., 2011; Sacco et al., 2009). Here, each of the latent classes demonstrated quantitatively different item endorsement profiles on the indicators of alcohol consumption, dependence symptoms, and negative social consequences. Although these studies have sometimes suggested models with different numbers of subgroups, each of the related latent classes showed substantially

similar characteristics across the models. Based on these models, alcohol drinkers can be separated into (i) a non-problematic class, (ii) a subgroup of regular drinkers with low probability of dependence symptoms, (iii) a subgroup of heavy drinkers with mild to moderate probability of dependence symptoms, and (iv) a highly symptomatic or severe subgroup.

However, it is also important to note that some other typologies using general population samples suggest qualitatively different item endorsement profiles between subgroups of alcohol drinkers (Rist et al., 2009; Smith & Shevlin, 2008). Furthermore, there has been evidence of subgroups characterized by a moderate to high probability of harmful consequences, but without experiencing of dependence symptoms. Similarly, Rist et al. (2009) also discriminated a latent class showing a high probability of dependence symptom endorsement without experiencing harmful consequences.

Given this background, the first aim of the present study was to (i) discriminate homogenous subgroups of drinkers on an empirical basis, based on the indicators of the Alcohol Use Disorders Identification Test (AUDIT) items. As some previous studies also used the items of the AUDIT as indicators (Kuvaas et al., 2014; Rist et al., 2009; Smith & Shevlin, 2008), it provides an opportunity to directly compare the model in the present study with these previous classification solutions. The second aim was to (ii) validate the identified latent classes based on psychopathological symptoms, such as externalizing and internalizing characteristics and socio-demographical variables.

IV/3. Material and methods

IV/3/1. Participants and procedure

The present study utilized data from a nationally representative sample of the National Survey on Addiction Problems in Hungary 2015 (NSAPH 2015). A detailed introduction to the study and descriptive statistics related to the sample characteristics have been presented elsewhere (Paksi et al., 2017). The main aim of the NSAPH 2015 was to assess epidemiological prevalence and population trends related to psychoactive substance use disorders and specific behavioral addictions. The target population of the study was the Hungarian adult population aged between 18 and 64 years. The NSAPH 2015 sample ensured proportional distribution of the participants in terms of age, regional geographic locations, and size of residence. The sample group of younger adults (aged between 18 and 34 years) was overrepresented. The study had a gross sample of 2477 participants,

and a net sample of 2274 participants. For the present analyses, participants who had used alcohol in the past 12 months were selected for further analysis (N = 1619). However, a further 99 participants were excluded because of missing data on all of the indicator variables. Consequently, the final sample comprised 1520 participants (52.2% male [N = 794]; mean age = 33.14 years; [SD = 12.32]).

IV/3/2. Measures

IV/3/2/1. Alcohol Use Disorders Identification Test (AUDIT). Items of the AUDIT were used to assess the patterns of the participants' alcohol consumption and the harmful consequences experienced (Gerevich et al., 2006; Saunders et al., 1993). The AUDIT is a widely used screening questionnaire in practice and research, which identifies different risk-based groups of participants who show excessive alcohol consumption. It contains 10 items, which cover three main aspects of drinking behavior in the past 12 months: characteristics and level of alcohol consumption (Items 1-3), symptoms of alcohol dependence (Items 4-7), and negative consequences due to alcohol consumption (Items 8-10). The instrument displayed acceptable internal consistency in this sample (Cronbach's $\alpha = 0.82$).

Due to the very high level of floor effect on the original response scales (IV/Supplementary Table 1), it was not feasible to consider the items of the AUDIT as continuous indicators during the analyses. Consequently, items were transformed into dichotomous variables for further analysis. A previous study also applied a similar approach of item transformation on AUDIT items (Smith & Shevlin, 2008). For the first question (*"How often do you have a drink containing alcohol?"*), the second response category (*Monthly or less*) was specified as the baseline category, while higher levels of responses (3 = *Two to four times a month*, 4 = *Two to four times a week*, 5 = *Four or more times a week*) were defined as the second category. For the second question (*"How many drinks containing alcohol do you have on a typical day when you are drinking?"*), the first response category (*One or two drinks*) was specified as the baseline category, while higher level of responses (2 = *Three or four drinks*, 3 = *Five or six drinks*, 4 = *Seven to nine drinks*, 5 = *Ten or more drinks*) were defined as the second category. In the case of Items 3 to 8 (e.g., Item 3: *"How often do you have six or more drink on one occasion?"*), the first response category (*Never*) was specified as the baseline category, and higher levels on the response scale (2 = *Less than monthly*, 3 = *One to three times a month*, 4 =

One to three times a week, 5 = *At least four times a week*) were coded as the second category. For Questions 9 and 10 (e.g., Item 9: *"Have you or someone else been injured because of your drinking?"*), the first response category (*Never*) was specified as the baseline category, while higher level of responses (2 = *Yes, but not in the past year*, 3 = *Yes, during the past year*) were defined as the second category.

IV/3/2/2. Brief Symptom Inventory (BSI). A modified and abbreviated version of the Brief Symptom Inventory (Derogatis & Savitz, 2000; Unoka et al., 2004) was used to assess different dimensions of psychopathological symptoms. This self-report instrument is widely used to detect and monitor various dimensions of psychological disorders in clinical practice and research. The current version of the instrument contains 27 items, which reflect the symptoms of anxiety, depression, hostility, interpersonal sensitivity, and obsessive-compulsivity. Therefore, the current version of the BSI does not cover all the conditions of the original scale. Participants had to provide responses on a five-point scale for each question. Subscales of the questionnaire presented satisfactory internal consistencies in the present sample (Cronbach's $\alpha = 0.80 – 0.87$).

IV/3/3. Data analysis

In order to identify homogenous subgroups of participants based on their characteristics of alcohol consumption, a Latent Class Analysis (LCA) was conducted (Collins & Lanza, 2009). AUDIT items were specified as dichotomous indicator variables. Model retention with the optimal number of latent classes was carried out iteratively. First, the most parsimonious model with only one latent class was fitted to the data. Thereafter, in case of the subsequent models, the number of latent classes was increased with one additional class in each of the stages. The series of model specification was viewed as complete if the model fit indices no longer indicated a more sufficient fit by the involvement of one additional subgroup. In order to retain the best fitting model, the results of multiple model fit indices were taken into account. Compared with other solutions, the best fitting model should show lower values of Akaike Information Criteria (AIC), Bayesian Information Criteria (BIC), Sample Size Adjusted Bayesian Information Criteria (SSA-BIC), and higher rate of categorization accuracy which is assessed using the index of Entropy. Moreover, significant result of the Lo-Mendel-Rubin Adjusted Likelihood Ratio Test (LMRT) displays more optimal fit for a particular model, because an additional latent

class describes the pattern of responses more closely contrasted to the previous model with fewer latent classes.

The next step of the analysis validated the identified latent classes. Therefore, multinomial logistic regression was performed with R3Step (Muthén & Muthén, 2017) to explore the effect of socio-demographical and psychological independent variables on the latent classes. The model included gender, age, level of education, employment status, age of onset related to the first alcoholic drink, and symptom levels of anxiety, depression, hostility, interpersonal sensitivity, and obsessive-compulsivity as covariates. Moreover, the level of psychopathological symptoms were also compared across the identified latent classes by using the BCH method (Asparouhov & Muthén, 2014b). Finally, the identified latent classes were cross-validated with AUDIT-based risk categories and lifetime history of psychiatric or AUD-related treatment involvement status. In the case of multinomial logistic regression and cross-validation with categorical variables, crude Odds Ratios (ORs) were calculated as an effect size measure. Data were weighted for all analyses to ensure generalizability to the population. IBM SPSS Statistics 23.0 and Mplus 8.0 statistical software were used in the analyses (Muthén & Muthén, 2017).

IV/4. Results

IV/4/1. Latent Class Analysis (LCA)

The response distribution on the original items of the AUDIT for active drinkers and the item endorsement probabilities of the dichotomous AUDIT variables in the total sample, and among males and females are presented in IV/Supplementary Tables 1 and 2. LCA was performed to identify subgroups of participants who showed similar patterns of item endorsement probabilities related to alcohol consumption and harmful consequences. Models with one to four latent classes were estimated and assessed in terms of model fit. Various model fit indices related to these models are summarized in IV/Table 1. Although the index of AIC and SSA-BIC indicated that the four-class solution fitted the data most closely, measures of BIC and Entropy implied a reduction in the level of model fit by the inclusion of the fourth latent class. Moreover, LMRT yielded a non-significant ($p > 0.05$) result in case of the model with four latent classes. Thus, the inclusion of an additional latent class over three subgroups did not provide a more parsimonious solution. Overall, the three-class solution provided the most adequate degree of model fit. The average

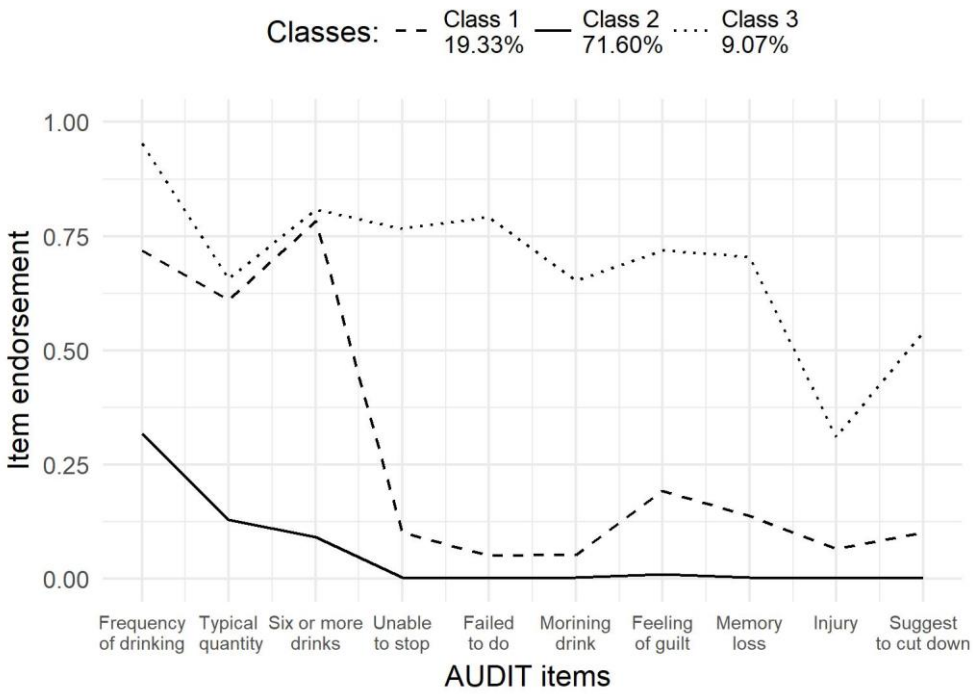
latent class probabilities for the most likely latent class membership were 0.95, 0.79 and 0.94, respectively. Further analyses were conducted with this model.

IV/Table 1. Fit indices for the latent class analysis models based on dichotomous items of the AUDIT

	AIC	BIC	SSA-BIC	Entropy	LMRT	*p*
1-class model	11160.04	11213.30	11181.54			
2-class model	8807.54	8919.39	8852.68	0.932	2345.40	< 0.001
3-class model	8588.68	8759.13	8657.47	0.812	237.91	0.002
4-class model	8545.33	8774.37	8637.77	0.795	64.55	0.760

Note. AIC = Akaike Information Criteria; BIC = Bayesian Information Criteria; SSA-BIC = Sample Size Adjusted Bayesian Information Criteria; LRT = Lo-Mendel-Rubin Adjusted Likelihood Ratio Test.

In order to interpret the three identified latent classes, item-endorsement probability characteristics were considered. Response patterns of the three latent classes are presented in IV/Table 2 and IV/Figure 1. Participants assigned to Class 1 ('Light alcohol drinkers') demonstrated the lowest rates of item endorsement probability related to indicators of alcohol consumption, dependence, and negative consequences. Class 2 ('Alcohol drinkers with low risk of dependence') was described with medium to high probability of item endorsement on alcohol consumption-related indicators, and low probability of item endorsement related to dependence and negative consequences. The subgroup of Class 3 ('Alcohol drinkers with severe dependence symptoms') showed high probability of alcohol consumption-related item endorsement, and the highest rates of symptom endorsement probability on indicators of dependence and negative consequences.



IV/Figure 1. Class-based probability of endorsing each dichotomous items of the AUDIT.

IV/4/2. Validation of the latent classes

First, the identified latent classes were contrasted in terms of psychopathological symptoms. IV/Table 2 summarizes the results of the multiple comparisons. Alcohol drinkers with low-risk of dependence and severe dependence symptoms reported the highest scores on anxiety, depression, hostility and interpersonal sensitivity. 'Light alcohol drinkers' showed the lowest levels of psychopathological symptoms in each of the multiple comparisons. Multinomial logistic regression was also conducted to validate the identified latent classes. IV/Table 3 presents the results related to the effects of socio-demographical and psychological covariates. The latent class of 'Light alcohol drinkers' was specified as a reference category. In case of 'Alcohol drinkers with low risk of dependence', male gender, younger age, economically active status, earlier onset related to the first alcoholic drink, and a higher level of depression significantly increased the odds of membership compared to Class 1. Significantly higher odds of membership were displayed for 'Alcohol drinkers with severe dependence symptoms' compared to the reference category if the participant was male, had a lower level of educational achievement, reported earlier onset related to the first alcoholic drink, and showed a higher level of hostility.

IV/Table 2. Class-based probability of endorsing each dichotomous items of the AUDIT and comparisons of latent classes.

	Class 1 'Light alcohol drinkers' *N* = 1088 (71.60%)	**Class 2 'Alcohol drinkers with low risk of dependence'** *N* = 294; (19.33%)	**Class 3 'Alcohol drinkers with severe dependence symptoms'** *N* = 138 (9.07%)	Overall Wald test (*p*)
Frequency of alcohol consumption: at least two times a month	0.32	0.72	0.95	
Typical quantity of drinks: at least three drinks on a typical day	0.13	0.61	0.66	
Six or more drinks on one occasion	0.09	0.78	0.81	
Unable to stop drinking	< 0.01	0.10	0.77	
Failed to do what was normally expected	< 0.01	0.05	0.79	
Drink in the morning	< 0.01	0.05	0.65	
Feeling of guilt or remorse after drinking	0.01	0.19	0.72	
Unable to remember what happened because of drinking	< 0.01	0.14	0.71	
Somebody injured as a result of drinking	< 0.01	0.07	0.31	
Somebody concerned about drinking, suggested to cut down	< 0.01	0.10	0.54	

	Class 1 'Light alcohol drinkers' *N* = 1088 (71.60%)	**Class 2 'Alcohol drinkers with low risk of dependence'** *N* = 294; (19.33%)	**Class 3 'Alcohol drinkers with severe dependence symptoms'** *N* = 138 (9.07%)	Overall Wald test (*p*)
Comparisons M (SE)				
Age	$42.36\ (0.48)_a$	$35.23\ (1.21)_b$	$42.34\ (1.36)_a$	26.65 ($p < 0.001$)
Anxiety	$9.13\ (0.17)_a$	$9.94\ (0.39)_{a,b}$	$10.94\ (0.53)_b$	13.01 ($p = 0.001$)
Depression	$9.23\ (0.20)_a$	$10.48\ (0.47)_b$	$11.89\ (0.75)_b$	16.28 ($p < 0.001$)
Hostility	$7.00\ (0.12)_a$	$8.39\ (0.39)_b$	$9.55\ (0.45)_b$	39.82 ($p < 0.001$)
Interpersonal sensitivity	$5.90\ (0.12)_a$	$6.59\ (0.25)_b$	$7.31\ (0.37)_b$	17.39 ($p < 0.001$)
Obsessive-compulsive	$9.25\ (0.18)_a$	$10.05\ (0.42)_a$	$11.80\ (0.58)_b$	19.39 ($p < 0.001$)

Note. Means in the same row that do not share subscripts differ at $p < 0.05$ level. BCH method was used in the comparison (Asparouhov & Muthén, 2014b).

IV/Table 3. Predictors of class memberships: a multinomial logistic regression.

	Class 2 'Alcohol drinkers with low risk of dependence' Crude OR [95% CI]	Class 3 'Alcohol drinkers with severe dependence symptoms' Crude OR [95% CI]
Gender[1]	**4.45 [2.47 – 8.04]**	**3.75 [1.73 – 8.10]**
Age	**0.94 [0.91 – 0.96]**	0.98 [0.96 – 1.00]
Level of education[2]	1.24 [0.69 – 2.20]	**3.73 [1.97 – 7.07]**
Employment status[3]	**1.91 [1.02 – 3.56]**	1.12 [0.56 – 2.24]
Young age of onset: first drink[4]	**2.14 [1.16 – 3.94]**	**3.01 [1.57 – 5.76]**
Depression	**1.10 [1.02 – 1.20]**	1.02 [0.91 – 1.15]
Hostility	1.14 [0.97 – 1.33]	**1.24 [1.07 – 1.43]**
Interpersonal sensitivity	0.97 [0.85 – 1.11]	0.89 [0.74 – 1.07]
Obsessive-compulsive	0.93 [0.82 – 1.05]	1.02 [0.89 – 1.18]

Note. Crude Odds Ratios (95% confidence intervals) of the association between validating covariates and latent class membership relative to Class 1 ('Light alcohol drinkers'). Odds ratios presented by bold figures are significant at least $p < 0.05$ level. [1]Gender: 0 = Female, 1 = Male; [2]Level of education: 0 = Participant had a graduation at vocational or high-school at least, 1 = Participant did not have vocational or high-school graduation; [3]Employment status: 0 = Unemployed, economically inactive, 1 = Working,

economically active; [4]Age of onset: first alcoholic drink: 0 = At least at the age of 15 years, or none, 1 = At the age of 14 years or earlier. Anxiety was not included in the final analysis as a predictor, due to the negative suppressor effect of depression. IV/Supplementary Table 3 contains the results of the analysis, when anxiety was also included as a predictor variable.

The identified latent classes were cross-validated with the AUDIT-based risk categories. IV/Supplementary Table 4 summarizes the distribution of the participants across these categories. The membership of 'Light alcohol drinkers' and low-risk alcohol drinking was fully overlapped (100%). The majority of 'Alcohol drinkers with low risk of dependence' (87.7%) were described as low-risk drinkers based on the AUDIT, while only small proportion (12.3%) of the respondents in this subgroup was categorized as hazardous drinkers. A high proportion of 'Alcohol drinkers with severe dependence symptoms' were categorized with hazardous drinking (65.4%), or harmful drinking and possible dependence (24.7%) based on the AUDIT.

Finally, the association between the identified latent classes and lifetime history of psychiatric and AUD-related treatment involvement were also analyzed. Frequencies of each category combinations are displayed in IV/Supplementary Tables 5 and 6. The latent class of 'Alcohol drinkers with severe dependence symptoms' had the highest proportion of individuals who reported lifetime history of psychiatric treatment (19.3%) or AUD-related treatment (12.3%) treatment. In the cases of 'Alcohol drinkers with low risk of dependence' (3.9 and 6.7% respectively) and 'Light alcohol drinkers' (0.4 and 5.0% respectively), fewer participants had received previous psychiatric or AUD-related treatment. It was also found that a small proportion of abstinent and non-active alcohol drinkers reported lifetime psychiatric treatment (N = 34; 5.2%) or AUD-related treatment (N = 5; 0.8%).

IV/5. Discussion

The present study explored subgroups of past-year alcohol users in a nationally representative population-based sample from Hungary where the prevalence of alcohol use disorder and rates of alcohol-related morbidity and mortality are among the highest in the world. Analyses demonstrated a three-class solution where each of the latent classes were heterogeneous in the level of alcohol consumption and harmful consequences due to alcohol drinking. The three latent classes identified were defined on the basis of

alcohol-drinking severity. ‘Light alcohol drinkers’ were considered as the least severe subgroup of alcohol drinkers. Although with higher rates of alcohol consumption, ‘Alcohol drinkers with low risk of dependence’ still showed a low level of alcohol-related dependence symptoms and harmful consequences. The subgroup of ‘Alcohol drinkers with severe dependence symptoms’ was described as the most severe subgroup due to high probability of alcohol dependence and harmful consequences item endorsement.

The present results indicate that alcohol-related harmful consequences sit on a continuum of severity in the general population. Instead of qualitatively different symptom profiles (Rist et al., 2009; Smith & Shevlin, 2008), subgroups of drinkers were discriminated by increasing probability of item endorsement related to alcohol dependence symptoms and negative consequences (Bucholz et al., 1996; Chung & Martin, 2001). These findings complement the unidimensional AUD approach of DSM-5 (Hildebrandt et al., 2017). Numerous previous models also suggested some forms of severity-based subgroups of alcohol drinkers based on general population and community samples (Casey et al., 2013; Castaldelli-Maia et al., 2014; Jackson, Bucholz, et al., 2014).

These typologies typically distinguish three or four latent classes of drinkers, and which show parallel and quantitatively different symptom endorsement profiles. The identified subgroups based on the present study broadly corresponded with latent classes identified in previous classification models. ‘Light alcohol drinkers’ corresponded with the ‘Non-symptomatic class’ reported by Ko et al. (2010) and Castaldelli-Maia et al. (2014), and to the ‘Non-problematic class’ reported by Casey et al. (2013), or the ‘Baseline/Very Mild consumption’ reported by Smith and Shelvin (2008). ‘Alcohol drinkers with low risk of dependence’ demonstrated similar characteristics to the ‘Minimally dependent drinkers’ reported by Jackson, Bucholz, et al. (2014) and the ‘Moderate risk’ group reported by Sacco et al. (2009). ‘Alcohol drinkers with severe dependence symptoms’ had comparable symptom profiles to the ‘High symptomatic class’ reported by Ko et al. (2010) and Castaldelli-Maia et al. (2014), and to the ‘Extreme class’ reported by Casey et al. (2013), and to the subgroup of ‘Heavy consumption with multiple negative consequences’ reported by Smith and Shelvin (2008).

In the severity-based latent class solution, the alcohol dependence related items (4-7) and negative consequences related items (8-10) were not separated, but were associated with each other. Therefore, indicator variables differentiated the identified subgroups by two

main aspects: level of alcohol consumption (Items 1-3) and harmful consequences due to drinking (Items 4-10). At the less severe level of the continuum (e.g., between Class 1 and Class 2), the indicators related to alcohol consumption differentiated more predominantly, such as frequency and quantity of alcohol drinking, and heavy episodic alcohol drinking. At the more severe level of the spectrum (e.g., between Class 2 and Class 3) similar rates of alcohol consumption were observed. Therefore, indices of harmful consequences due to drinking isolated the differences between the latent classes (Kuvaas et al., 2014). Similar patterns of differentiation have been found among participants in a national representative sample (Jackson, Bucholz, et al., 2014), older adults (Sacco et al., 2009), and college students (Kuvaas et al., 2014). However, the similar levels of alcohol consumption in the cases of Class 2 and Class 3 is in contradiction with the conceptualization of 'heavy use over time' for alcohol use problems (Rehm et al., 2013). According to Rehm and colleagues (2013), more severe levels of alcohol consumption can be accounted for by higher rates of alcohol-related harmful consequences and AUD symptoms, therefore the amount and frequency of heavy drinking should be considered as indicators of alcohol use disorder. The present study was unable to demonstrate a clear dose-response association between measures of alcohol consumption and harmful consequences. Therefore, it was not possible to distinguish latent classes of 'Alcohol drinkers with low risk of dependence' and 'Alcohol drinkers with severe dependence symptoms' solely based on dichotomous measures of alcohol consumption. It was also important to take into account the indices of harmful consequences due to drinking in order to accurately identify those individuals who were characterized with more severe patterns of drinking.

Overall, based on the present analysis, approximately 9% of the alcohol users showed severe symptoms of alcohol dependence in the population. Similarly, previous studies based on population-based nationally representative samples also reported 5-7% of the active alcohol drinkers were classified in the highly affected subgroups (Casey et al., 2013; Castaldelli-Maia et al., 2014; Smith & Shevlin, 2008). However, compared with previous epidemiological findings which assessed alcohol drinking patterns in Hungary (World Health Organization, 2014), lower prevalence rates of heavy episodic drinking and AUD among alcohol users were presented in the present study. Therefore, there is a need for future studies to obtain a more accordant view related to the different forms of problematic alcohol consumption in Hungary. The relatively high prevalence of risky

alcohol users also indicates important public health implications. Compared with light drinkers, heavy alcohol drinking classes might show heavier health care utilization, as they are more likely to be admitted to inpatient or emergency departments (Miquel et al., 2018). Heavy alcohol drinking was also linked with decreased life expectancy, as several causes of premature mortality were associated with heavy alcohol use (e.g. chronic liver disease, suicide, Alzheimer's disease) (Rehm & Probst, 2018).

Follow-up analyses also illustrated significant differences between the subgroups of alcohol drinkers in terms of alcohol-related risk categories, psychiatric treatment, and AUD-related treatment. Cross-validation of the identified latent classes with the AUDIT-based risk categories also suggested that 'Alcohol drinkers with severe dependence symptoms' were mainly classified at least as someone who shows hazardous drinking. Similarly, members of this subgroup showed the highest rates of lifetime psychiatric treatment and AUD-related treatment. Similar rates of treatment involvement related to the most severe subgroup of drinkers were reported in a US-based study using a nationally representative population sample (Ko et al., 2010). A substantial proportion of 'Alcohol drinkers with low risk of dependence' did not reach the threshold of hazardous drinking. Therefore, future prospective studies should examine whether this class shows a risk for developing more severe forms of problematic alcohol consumption (Carpenter et al., 2006).

Groups which were at the higher end of the severity-continuum also demonstrated psychopathological vulnerability. Alcohol drinkers with low-risk of alcohol dependence and severe alcohol dependence symptoms showed the highest level of anxiety, depression, hostility, interpersonal sensitivity, and obsessive-compulsive symptoms. Present findings correspond with the theoretical and clinical concept that AUD is associated with internalizing and externalizing characteristics (Hesselbrock & Hesselbrock, 2006). More specifically, a higher level of hostility and depression predicted membership of the more severe latent classes. In the case of negative affect (e.g., depression, anxiety), it is assumed that alcohol consumption might serve as a means for coping and/or mood regulation. Previous studies have also hypothesized that externalizing characteristics, such as antisocial behavior, contributes to AUD via general personality and behavioral traits of impulsivity, irresponsibility, and/or irritability (Sintov et al., 2010). Overall, the results of the present study suggest more attention is needed on externalizing symptoms when screening for AUD.

Alcohol drinkers with low-risk of dependence and severe dependence symptoms were also characterized with specific socio-demographic attributes. Males were more likely to be present in the most severe groups. Similar gender-related differences have been reported in various previous studies (Casey et al., 2013; Smith & Shevlin, 2008). However, it is important to explore whether different pathways related to excessive alcohol drinking can be assumed for females (Shireman et al., 2015). In case of 'Alcohol drinkers with severe dependence symptoms', a lower level of educational achievement enhanced the odds of being in this group. The possible risk factor related for decreased educational achievement (i.e., dropping out from school early) has consistently been demonstrated by previous studies using LCA (Carpenter et al., 2006; Castaldelli-Maia et al., 2014). Finally, 'Alcohol drinkers with low risk of dependence' were younger than their severely dependent counterparts. Therefore, it is not clear if this status is a transient one, and what proportion of the members of this group may develop severe dependence symptoms in their latter life. Further research utilizing a longitudinal design would address the transition from one group to another either from low-risk of dependence to severe dependence group, or vice versa from severe dependence group towards light use or no use at all (Moss et al., 2010). The present study was unable to capture this dynamic change among the community sample recruited.

IV/5/1. Limitations and future directions

Four major limitations should be considered in relation to the interpretation of results in the present study. First, the cross-sectional design of the research does not allow the determination of causal pathways between psychopathological symptoms and membership of latent classes. Future longitudinal studies should also examine the temporal stability and membership transitions of each of the identified latent classes reported here. Second, it might be possible that the individuals who showed more severe forms of alcohol consumption were under-represented in the present sample (World Health Organization, 2014), therefore the identified subgroups did not capture accurately the heterogeneity of alcohol-related problems. Third, as latent classes of 'Alcohol drinkers with low risk of dependence' and 'Alcohol drinkers with severe dependence symptoms' contained relatively few participants, the generalization of the finding related to these subgroups is only possible in a limited manner. Fourth, several important aspects of excessive alcohol drinking were not included in the LCA model. Thus, future studies should take into account the effect of psychoactive substance use, and history and

presence of AUD among other family members. Additional methodological bias may also have been present due to the dichotomous indicator variables used. As a consequence, it is possible that the alcohol consumption-related variables might not have properly differentiated between the latent classes. Finally, there is a possibility that the comparison between classification models were limited due to measurement- and population-related differences (Kuvaas et al., 2014).

IV/6. Conclusions

The present study identified subgroups of past-year alcohol users in a nationally representative population-based sample. The three defined latent classes provided a range of alcohol use severity (with approximately 9% showing severe symptoms of alcohol dependence in the sample). The present sample might have incorporated a wider range of problematic alcoholic drinkers due to the sample characteristics. The psychopathological vulnerability of the more severe subgroups was also found, and the significant predictive effects of hostility and depression were demonstrated. The specification of homogenous and empirically-derived subgroups of alcohol drinkers might therefore contribute to the development of more tailored prevention and screening services for those with AUD (Leggio et al., 2009).

IV/7. Supplementary materials

IV/Supplementary Table 1. Response distribution on the items of the AUDIT for active alcohol drinkers

Items	Response categories *N* (%)				
	0	1	2	3	4
1. Frequency of alcohol consumption: at least two times a month[1]	-	539 (54.5%)	288 (29.1%)	87 (8.8%)	73 (7.4%)
2. Typical quantity of drinks: at least three drinks on a typical day[2]	627 (63.4%)	161 (16.2%)	49 (4.9%)	7 (0.7%)	18 (1.8%)
3. Six or more drinks on one occasion[3]	876 (88.5%)	59 (5.9%)	20 (2.0%)	5 (0.5%)	3 (0.3%)
4. Unable to stop drinking[3]	876 (88.5%)	59 (5.9%)	20 (2.0%)	5 (0.5%)	3 (0.3%)
5. Failed to do what was normally expected[3]	884 (89.3%)	65 (6.5%)	9 (0.9%)	3 (0.3%)	2 (0.2%)
6. Drink in the morning[3]	892 (90.2%)	48 (4.9%)	11 (1.2%)	5 (0.5%)	4 (0.4%)
7. Feeling of guilt or remorse after drinking[3]	857 (86.7%)	74 (7.5%)	20 (2.0%)	8 (0.8%)	5 (0.5%)

Items	Response categories *N* (%)				
	0	1	2	3	4
8. Unable to remember what happened because of drinking[3]	874 (88.3%)	65 (6.5%)	16 (1.6%)	6 (0.6%)	2 (0.2%)
9. Somebody injured as a result of drinking[4]	932 (94.2%)	-	36 (3.7%)	-	5 (0.5%)
10. Somebody concerned about drinking, suggested to cut down[4]	900 (90.9%)	-	47 (4.7%)	-	21 (2.2%)

Note. Analysis was performed in a weighted sample (*N* = 989). Response categories: [1]0 = *Never*, 1 = *Monthly or less*, 2 = *Two to four times a month*, 3 = *Two to four times a week*, 4 = *Four or more times a week*; [2]0 = *One or two drinks*, 2 = *Three or four drinks*, 3 = *Five or six drinks*, 4 = *Seven to nine drinks*, 5 = *Ten or more drinks*; [3]0 = *Never*, 1 = *Less than monthly*, 2 = *One to three times a month*, 3 = *One to three times a week*, 4 = *At least four times a week*, [4]0 = *Never*, 2 = *Yes, but not in the past year*, 4 = *Yes, during the past year*.

IV/Supplementary Table 2. Item endorsement of the AUDIT items in the total sample, and among males and females.

Items	Endorsement in the total sample (*N* = 989)	Endorsement among males (*N* = 513)	Endorsement among females (*N* = 476)
1. Frequency of alcohol consumption: at least two times a month	448 (45.3%)	328 (64.0%)	120 (25.1%)
2. Typical quantity of drinks: at least three drinks on a typical day	234 (23.7%)	166 (32.3%)	68 (14.4%)
3. Six or more drinks on one occasion	283 (28.6%)	204 (39.8%)	79 (16.5%)
4. Unable to stop drinking	87 (8.8%)	71 (13.8%)	16 (3.3%)
5. Failed to do what was normally expected	78 (7.9%)	59 (11.5%)	19 (4.1%)
6. Drink in the morning	68 (6.9%)	55 (10.8%)	13 (2.7%)
7. Feeling of guilt or remorse after drinking	106 (10.7%)	76 (14.8%)	30 (6.3%)
8. Unable to remember what happened because of drinking	88 (8.9%)	69 (13.4%)	20 (4.2%)
9. Somebody injured as a result of drinking	42 (4.2%)	34 (6.7%)	7 (1.5%)
10. Somebody concerned about drinking, suggested to cut down	68 (6.9%)	56 (10.9%)	12 (2.6%)
Total AUDIT score M (SD)	3.46 (3.93)	4.62 (4.62)	2.19 (2.44)
Category of low-risk drinking[1] *N* (%)	744 (75.2%)	360 (70.2%)	385 (80.7%)
Category of hazardous alcohol use[2] *N* (%)	73 (7.4%)	59 (11.5%)	14 (3.0%)

Items	Endorsement in the total sample (N = 989)	Endorsement among males (N = 513)	Endorsement among females (N = 476)
Category of harmful alcohol use[3] or possible dependence[4] *N* (%)	21 (2.1%)	19 (3.8%)	1 (0.2%)

Note. Analysis was performed in a weighted sample (N = 989). [1]Category of low-risk drinking: total AUDIT score between 0-7 points; [2]Category of hazardous alcohol use: total AUDIT score between 8-15 points; [3]Category of harmful alcohol use: total AUDIT score between 16-19 points; [4]Category of possible dependence: at least 20 points on the total AUDIT scale

IV/Supplementary Table 3. Odds ratios (95% Confidence Intervals) of the association between validating covariates and latent class membership relative to Class 1 ('Light alcohol drinkers').

	Class 2 (19.33%) 'Alcohol drinkers with low risk of dependence' Crude OR [95% CI]	Class 3 (9.07%) 'Alcohol drinkers with severe dependence symptoms' Crude OR [95% CI]
Gender[1]	**4.55 [2.52 – 8.22]**	**3.26 [1.51 – 7.03]**
Age	**0.94 [0.91 – 0.96]**	0.98 [0.96 – 1.00]
Level of education[2]	1.24 [0.69 – 2.22]	**3.83 [2.00 – 7.34]**
Employment status[3]	**1.90 [1.01 – 3.56]**	1.13 [0.55 – 2.31]
Young age of onset: first drink[4]	**2.13 [1.15 – 3.94]**	**3.02 [1.58 – 5.78]**
Anxiety	0.98 [0.82 – 1.17]	**0.80 [0.67 – 0.95]**
Depression	**1.11 [1.01 – 1.22]**	1.07 [0.96 – 1.21]
Hostility	1.15 [0.97 – 1.36]	**1.33 [1.14 – 1.55]**
Interpersonal sensitivity	0.98 [0.85 – 1.13]	0.96 [0.79 – 1.16]
Obsessive-compulsive	0.93 [0.81 – 1.06]	1.09 [0.94 – 1.27]

Note. Crude Odds ratios presented by bold figures are significant at least $p < 0.05$ level. [1]Gender: 0 = Female, 1 = Male; [2]Level of education: 0 = Participant had a graduation at vocational or high-school at least, 1 = Participant did not have vocational or high-school graduation; [3]Employment status: 0 = Unemployed, economically inactive, 1 = Working, economically active; [4]Age of onset: first drink: 0 = At least at the age of 15 years, or none, 1 = At the age of 14 years or earlier.

IV/Supplementary Table 4. Association between the identified latent classes and the AUDIT-based risk categories.

	Class 1 'Light alcohol drinkers' *N* = 594 (71.0%)	Class 2 'Alcohol drinkers with low risk of dependence' *N* = 162 (19.3%)	Class 3 'Alcohol drinkers with severe dependence symptoms' *N* = 81 (9.7%)
Category of low-risk alcohol drinking[1]; *N* = 744 (88.9%)	594 (100.0%)	142 (87.7%)	8 (9.9%)
Category of hazardous alcohol use[2]; *N* = 73 (8.7%)	0 (0.0%)	20 (12.3%)	53 (65.4%)
Category of harmful alcohol use[3] or possible alcohol dependence[4]; *N* = 20 (2.4%)	0 (0.0%)	0 (0.0%)	20 (24.7%)

Note. Analysis was performed in a weighted sample (*N* = 989). Percentages in each cells represents the proportion within each latent classes. $\chi^2(4) = 604.77$; $p < 0.001$; $\varphi = 0.850$. Note. [1]Category of low-risk alcohol drinking: total AUDIT score between 0-7 points; [2]Category of hazardous alcohol use: total AUDIT score between 8-15 points; [3]Category of harmful alcohol use: total AUDIT score between 16-19 points; [4]Category of possible dependence: at least 20 points on the total AUDIT scale

IV/Supplementary Table 5. Association between the identified latent classes and lifetime history of psychiatric treatment involvement.

		Class 1 'Light alcohol drinkers' *N* = 679 (72.1%)	Class 2 'Alcohol drinkers with low risk of dependence' *N* = 180 (19.1%)	Class 3 'Alcohol drinkers with severe dependence symptoms' *N* = 83 (8.8%)
Lifetime history of psychiatric treatment	Yes *N* = 62 (6.6%)	34 (5.0%)	12 (6.7%)	16 (19.3%)
	No *N* = 880 (93.4%)	645 (95.0%)	168 (93.3%)	67 (80.7%)
Crude OR [95% CI]*		*Ref.*	1.36 [0.69 – 2.67]	4.53 [2.38 – 8.64]

Note. Analysis was performed in a weighted sample (*N* = 989). Percentages in each cells represents the proportion within each latent classes. $\chi^2(2) = 24.50$; $p < 0.001$; $\varphi = 0.161$. Crude OR = odds ratio calculated without the missing values. CI = confidence interval*: Comparison group is Class 1 (*Ref.* = reference group).

IV/Supplementary Table 6. Association between the identified latent classes and lifetime history of psychiatric and AUD-related treatment involvement.

		Class 1 'Light alcohol drinkers' *N* = 677 (72.3%)	Class 2 'Alcohol drinkers with low risk of dependence' *N* = 178 (19.0%)	Class 3 'Alcohol drinkers with severe dependence symptoms' *N* = 81 (8.7%)
Lifetime history of AUD-related treatment	Yes *N* = 20 (2.1%)	3 (0.4%)	7 (3.9%)	10 (12.3%)
	No *N* = 916 (97.9%)	674 (99.6%)	171 (96.1%)	71 (87.7%)
Crude OR [95% CI]*		*Ref.*	9.20 [2.35 – 35.94]	31.64 [8.51 – 117.65]

Note. Analysis was performed in a weighted sample (*N* = 989). Percentages in each cells represents the proportion within each latent classes. $\chi^2(2) = 52.40$; $p < 0.001$; $\varphi = 0.237$. Crude OR = odds ratio calculated without the missing values. CI = confidence interval*: Comparison group is Class 1 (*Ref.* = reference group).

V. Study 2: Patterns and temporal change of psychopathological symptoms among inpatients with alcohol use disorder undergoing a twelve-step based treatment[8,9]

V/1. Abstract

Background: Patients diagnosed with Alcohol Use Disorder (AUD) present an increased risk for experiencing severe internalizing and externalizing symptoms. Involvement in twelve-step based treatment programs, such as the Minnesota Model (MM), can contribute to improvement of psychopathological symptom profile. The present study's main objective was to examine profiles and change trajectories of psychopathological symptoms of AUD subgroups during an eight-week long period of MM treatment attendance.

Method: Inpatients with AUD (N = 303) who attended MM treatment programs participated in the present study. Latent Class Growth Analysis (LCGA) was used to evaluate the psychopathological symptom change trajectories assessed by using the Brief Symptom Inventory (BSI). Multiple comparisons and multinomial logistic regression were performed to validate the subgroups.

Results: Three subgroups were identified: low severity (48.5%), moderate severity (35.2%), and high severity (16.2%) symptomatic subgroups. The moderate severity class demonstrated the largest effect in terms of symptoms decrease. Higher severity classes showed significantly higher rates of harmful alcohol drinking and drinking motives.

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Conclusions: The present study identified three severity-based subgroups which indicate that psychopathology sits on a spectrum of severity among AUD patients. The findings highlight the associations between AUD and internalizing symptoms, negative reinforcement drinking motives, and the symptomatic improvement that can occur among those participating in MM treatment programs.

Keywords: Alcohol Use Disorder (AUD); psychopathological symptoms; twelve-step based treatment; Minnesota Model; alcohol comorbidity

V/2. Introduction

Alcohol use disorder (AUD) is a chronic problem causing significant psychological, physical, interpersonal, and social burden among alcohol users and their environment. Extensive empirical research suggests that AUD frequently co-occurs with diverse or even multiple forms of psychiatric disorders, such as major depressive disorder, panic disorder, obsessive-compulsive disorder, generalized anxiety disorder, post-traumatic stress disorder, social phobia, or antisocial personality disorder (for review, see Bradizza et al., 2006). Comorbid externalizing and internalizing psychopathology are often associated with more severe subtypes of AUD in terms of clinical characteristics and prognosis (e.g., higher drinking severity, worse health status) (Hildebrandt et al., 2017; Moss et al., 2010) and harmful treatment-related consequences (e.g., higher level of treatment drop-out, increased vulnerability of early and long-term relapse after treatment) (Farren & McElroy, 2010; Krawczyk et al., 2017). Furthermore, some psychopathological symptoms (e.g., depression or anxiety symptoms) have an integral role in the pathology of AUD, such as progression into more pathological stages of AUD, motivation and maintenance of compulsive alcohol use, craving and relapses, or even during a period of long-term abstinence. For example, the allostatic model assumes that during the progression from early stages (e.g., preoccupation with alcohol use, frequent intoxication) to the more severe, compulsive stage of AUD, there is a shift in the motivational background of alcohol use from positive reinforcement (i.e., drinking to facilitate positive emotions and hedonic states) to negative reinforcement (i.e., drinking to alleviate negative affective states related to withdrawal), and function of negative affectivity becomes central due to adverse modifications in the reciprocal emotion-regulation and reward-regulation systems (Koob, 2011; Le Moal & Koob, 2007). Another

possible form of comorbidity is alcohol-induced mental disorders, such as mood, anxiety, bipolar and psychotic disorders, where psychopathological symptoms last for 1-6 months following excessive use of alcohol (Saunders, 2017).

Changes in psychopathological symptoms over time among individuals with AUD have been examined in a large body of existing literature. For example, studies using meta-analysis and systematic review have reported that patients with comorbid AUD and psychiatric disorders show improvements in depression, anxiety, and post-traumatic stress disorder symptoms due to involvement in antidepressant pharmacological therapies, cognitive behavioral therapies, and motivational interviewing (Baker et al., 2012; Foulds et al., 2015; Hobbs et al., 2011; Riper et al., 2014; Roberts et al., 2015). Moreover, previous studies have demonstrated that participation in structured twelve-step based therapeutic approaches, such as the Minnesota Model (MM) and the Twelve Step Facilitation (TSF) treatment, not only have beneficial impact on drinking-related outcomes (e.g., reaching longer periods of abstinence) (Grønbaek & Nielsen, 2007; Kelly et al., 2020; Project Match Research Group, 1998), but also related to improvements in psychopathological-related (e.g., attenuation of depression symptoms) outcomes (Andó et al., 2016; Worley et al., 2012).

In addition to other analytical approaches (e.g., factor analysis) (Harford et al., 2015), one possible way to examine structure and patterns of psychopathological comorbidity among individuals with AUD is to use person-centered analyses, such as latent class or profile analysis (Urbanoski et al., 2015). These methods allow to obtain better understanding on the heterogeneity within AUD by distinguishing subgroups of individuals where members within each identified class show similar profiles and combinations of psychopathological comorbidity. Among individuals with AUD either from treatment seeking or general population samples, previous studies have identified three to five distinct subgroups based on indicator variables assessing co-occurring psychopathological symptom levels or disorder presence (Glass et al., 2014; Müller et al., 2020; Urbanoski et al., 2015; Villalobos-Gallegos et al., 2017; Wallen et al., 2019). These studies have been conflicting in terms of the observed differences between the identified latent classes: quantitative (e.g., parallel and severity-based psychopathological symptom profiles) (Villalobos-Gallegos et al., 2017) and qualitative differences have been suggested between the psychopathology-based subgroups (e.g., classes with mainly externalizing and internalizing comorbidity) (Glass et al., 2014).

However, to the best of the authors' knowledge, no previous study has examined how latent classes of AUD change over time or due to treatment attendance in terms of co-occurring psychopathological symptom levels. Investigation of temporal change patterns of psychopathology-based latent classes might contribute to broaden existing knowledge about AUD in two broad aspects. First, it would be possible to obtain more detailed understanding on the structure of comorbid psychopathology among individuals with AUD. Considering the principles of latent class growth analysis (Jung & Wickrama, 2008), it is assumed that individuals with AUD might show substantial variability how their psychopathological symptom levels change over time and different subtypes of AUD might present various trajectories. Therefore, comorbid psychopathology-based subgroups might have heterogenous characteristics not only in terms of severity (e.g., mild level vs. high level of psychopathology) and qualitative aspects (e.g., presence of comorbid internalizing vs. externalizing psychopathology) of psychopathology, but in temporal patterns as well (e.g., higher degree of change vs. resistance of change in the level of psychopathology). Based on this approach, clinical prognosis of subgroups of AUD can be assessed in terms of comorbid psychopathology (Urbanoski et al., 2015).

Second, by identifying comorbid psychopathology-based latent classes it is possible to examine how these subgroups of AUD might differ in terms of treatment response (Lanza & Rhoades, 2013). Existing literature data has suggested that treatment effectiveness of a given intervention can vary as a function of membership of latent classes which are characterized with different profiles of alcohol misuse and internalizing and externalizing psychopathology (Roos et al., 2017). However, to the best of the authors' knowledge, no previous study has examined treatment-seeking individuals with AUD profile characteristics as well as temporal change patterns of comorbid psychopathology-based latent classes during an AUD-related treatment participation. Classification participants based on psychopathological symptom severity and change profiles can facilitate (i) to identify subgroups of AUD to whom a given treatment form can be considered as more effective, (ii) to design more individually-customized interventions and (iii) to specify in a more in-depth way how comorbid psychopathological symptoms alter treatment outcomes (Lanza & Rhoades, 2013; Urbanoski et al., 2015; Villalobos-Gallegos et al., 2017).

The aim of the present study was to examine patterns of severity and changes of psychopathological symptoms among subgroups of AUD inpatients attending a twelve-

step based treatment program. It was hypothesized that the psychopathological-based latent classes of AUD can be discriminated in terms of symptom severity and temporal change patterns (Jung & Wickrama, 2008; Villalobos-Gallegos et al., 2017). However, it is important to note, that theoretical and practical conclusions, such as structure of psychopathology in the AUD population or assessment of specific treatment effects, can only cautiously be drawn from the present study due to its methodological limitations (e.g., lack of randomized controlled trial, follow-up data collection, examination of treatment moderators and mediators, potential self-selection bias of the participants, and inclusion of relevant confounding variables).

V/3. Methods

V/3/1. Participants and procedure

The present study was conducted between 2013 and 2018 at the Nyírő Gyula National Institute of Psychiatry and Addictions, Budapest, Hungary. The study specifically focused on the MM treatment program which was primarily designed for patients with AUD or gambling problems. The treatment includes eight weeks of community-based residential care which harmonizes professional treatment approaches and principles of the twelve-step based self-help group of Alcoholics Anonymous (AA). In line with the concepts of AA, the treatment primarily aims to aid patients to reach and maintain long-term abstinence. The treatment program emphasizes relevance of group-therapeutic context, community-based factors, and therapeutic effect of 'here-and-now' in AUD-related and psychological progress. Recovery of patients is followed and guided by a multidisciplinary staff team comprising professional therapeutic specialists (i.e., clinical psychologist, psychiatrist, addictology-specific consultant), nurses and recovering helpers (i.e., individuals who have successfully recovered from addiction-specific problems and who provide counselling in an addiction-specific treatment program) (Doukas & Cullen, 2010). During the treatment, various group and individual psychotherapeutic techniques are applied, including daily AA meetings, specific group meetings based on the theoretical and practical principles of AA, assertiveness training, relaxation and stress management training, teaching of effective coping and relapse prevention skills, art therapeutic sessions, group meetings for affected family members, and psycho-education. The structure of the applied therapeutic techniques in the treatment program is presented in V/Table 1, while short description of the main treatment forms is

shown in V/Table 2. Each patient has an individual consultant from the staff team, therefore frequent and regular individual consultations also support and facilitate progression of the participants. The daily schedule of the therapeutic sessions and related assignments are controlled by timetables (see: V/Table 1) in addition to the predefined, eight-week long structure for the program. The first two weeks of the treatment are relatively restrictive (e.g., it is not allowed to leave the therapeutic site or to have visitors) which aims to gain a more self- and therapeutic-focused attention, while in subsequent weeks, amongst others, participation in daily AA meetings and twelve-step-related functions (e.g., selection of a sponsor) are emphasized to a greater degree. An adaptation period is held in the seventh week of the treatment. During this time, the participants stay at their home, which, for example, allow for them to monitor their experiences in their ordinary environment (for further details, see Tóth, 2018). Although the main therapeutic approaches and characteristics of the treatment program remained unchanged from the beginning (e.g., structured organization and timetable, main therapeutic sessions, requirements for enrollment), it is important to take into account that some changes might have affected treatment processes over time (e.g., development of therapeutic skills and expertise over time, changes in the staff).

Overall, 303 inpatients (180 males and 123 females) with AUD participated in the present study. A total of 218 participants (71.95%) successfully completed the eight-week long program, while 85 inpatients (28.05%) dropped out from the treatment before completion (see 'Sample characteristics' subsection). Every patient who were admitted to the treatment program between March 2013 and April 2018 were included as a participant in the present study. Each of the attending patients agreed (and provided informed consent) to participate in the study. Detoxification was undertaken prior to the participants' enrolment in the program as it was required from them to have at least one-week long abstinence and show absence of physical and acute psychological withdrawal before starting the treatment program. An approximately one-hour long, semi-structured interview was administered by the treatment staff before treatment enrollment. The interview took place at the treatment site, and an addition to the individual with AUD, one of his/her relatives also participated in the interview. The presence of the affected family members provides the opportunity to obtain a more accurate picture about the complex nature of problematic alcohol use not only for the staff but also for the individual with AUD (e.g., by asking them to share how the problematic alcohol use affected the

function of the family). It also helps in observing possible psychological dynamics within the family, and motivates the relatives to participate in the treatment program (i.e., the group of affected family members). During the interview motivation for treatment and change in the problematic use of alcohol, as well as aspects of treatment contraindication, were evaluated. Lack of organic dementia, severe personality disorder, tendency to act out, and acute suicide risk were prerequisites for treatment involvement. Moreover, data were collected during the interview concerning socio-demographics (e.g., age, gender, education, and work history), sources of family of social support, while psychiatric anamnesis was also evaluated (e.g., family history of substance misuse, previous suicide attempts, psychiatric-, AUD- or SUD-related treatment involvement history). The present study also assessed if a participant had received some forms of psychiatric-related or AUD-related pre-care shortly before the treatment program. Pre-care involvement was considered if a participant was directed from an inpatient psychiatric- or AUD-related department to the MM treatment program, or reported a participation in a psychiatric-, AUD- or SUD-related treatment program within one month before the start of the MM program. Standardized questionnaires were used at two measurement points. On first entering the treatment program, alcohol consumption (e.g., harmful alcohol consumption, drinking motives), and psychopathological-related aspects were assessed. At the end of the treatment program, the levels of psychopathological symptoms were re-assessed. Research assessment was conducted by the treatment staff at both measurement points. Among those participants who dropped out from the treatment before completion, data were only available at the first measurement point. Systematic follow-up data collection either after successful treatment completion or treatment interruption was not carried out.

V/Table 1. General timetable for the treatment program

Time	**Monday**	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**	**Sunday**
7:30 - 8:00	Morning thoughts	Morning thoughts	Morning thoughts	Morning thoughts	Morning thoughts	Morning thoughts	Morning thoughts
8:00 - 8:30	-	-	-	-	-	-	-
8:30 - 9:00	Just for today	Just for today	Just for today	Just for today	Just for today		Breakfast
9:00 - 9:30	Breakfast	Breakfast	Breakfast	Breakfast	Breakfast	Breakfast	Film group with discussion
9:30 - 10:00	Assertiveness training, or AA-steps group, or Psychoeducation	Assertiveness training, or AA-steps group, or Psychoeducation	Assertiveness training, or AA-steps group, or Psychoeducation	Art therapeutic group	Week ending group	-	
10:00 - 10:30						Retrospective meeting for the week	
10:30 - 11:00	Week starting group	-	-		Film group with discussion		
11:00 - 11:30		Assertiveness training, or AA-steps group, or Psychoeducation	Full department group	-		-	-
11:30 - 12:00	-						
12:00 - 12:30	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch
12:30 - 13:00		Film group with discussion					
13:00 - 13:30	Individual consultations and/or working on therapeutic tasks		Individual consultations and/or working on therapeutic tasks	Individual consultations and/or working on therapeutic tasks	Group for affected family members	Emotion-focused board game	-
13:30 - 14:00							
14:00 - 14.30					-		
14:30 - 15:00						-	
15:00 - 15:30	Working on therapeutic tasks	Stress management training	Stress management training	-			
15:30 - 16:00							
16:00 - 16:30	-	-	-				
16:30 - 17:00							
17:00 - 17:30	AA meeting	AA meeting	AA meeting	AA meeting	AA meeting		AA meeting
17:30 - 18:00							
18:00 - 18:30							
18:30 - 19:00							
19:00 - 19:30							
19:30 - 20:00							

Time	**Monday**	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**	**Sunday**
20:00 - 20:30	Dinner	Dinner	Dinner	Dinner	Dinner	Dinner	Dinner
20:30 - 21:00	Evening thoughts	Evening thoughts	Evening thoughts	Evening thoughts	Evening thoughts	Evening thoughts	Welcome back meeting

V/Table 2. Main therapeutic forms of the treatment program

Name of the session	**Short description and examples**
Morning thoughts	Group meeting which is guided by a trained nurse. Example session: a daily quote is selected, and participants are asked to think about how it can be related to their recovery.
Evening thoughts	Group meeting which is guided by a trained nurse. Example session: participants are asked to summarize and describe their day, daily emotions, etc.
Just for today	AA-specific group meeting which is guided by a recovering helper. The session follows theoretical and practical approaches of the AA. Example session: discussion of the experiences, questions of the participants regarding daily AA meetings and their process of recovery.
Assertiveness training	Relapse prevention-specific group session which is guided by a clinical psychologist. Example session: practicing how to handle and cope with situations when there is a risk for substance use.
AA-steps group	AA-specific group session which is guided by a recovering helper. It focuses on the first two steps of the AA. It is held between the third and sixth weeks.
Psychoeducation	Group meeting which is guided by a psychiatrist with the aim of facilitating knowledge about substance use disorder-related mechanisms. Example session: discussion of the main characteristics of substance use disorders, education about relevant psychological defense mechanisms.
Week starting group	Group meeting where all inpatients and staff team members are included. Community-based therapeutic approaches are applied in

Name of the session	Short description and examples
	this group. Themes of the sessions are not pre-defined, and are typically determined by the inpatients. Example session: discussion of problems occurring during the weekend with family members.
Week ending group	Group meeting where all inpatients and staff team members are included. Community-based therapeutic approaches are applied in this group. Themes of the sessions are not pre-defined, and are typically determined by the inpatients. Example session: preparation for returning home and meeting with family members during the weekend.
Full department meeting	Group meeting where all inpatients and staff team members of the Department of Addictology are involved (i.e., not just from the Minnesota treatment program). Example session: discussing issues which affect therapeutic work of the Department of Addictology.
Art therapeutic group	Creative group meeting which is held by a clinical psychologist. Example session: participants are encouraged to present a given problematic psychological aspect of their life by drawing.
Film group with discussion	Participants watch films which are relevant in terms of substance misuse and recovery, which is either followed by a group discussion or participants are asked to summarize their thoughts and feelings, emotions about the movie in written form.
Individual consultations	Each patient has an individual consultant from the staff team, therefore frequent and regular individual consultations also support and facilitate progression of the participants.
Working on therapeutic tasks	Some therapeutic forms require participants to prepare therapeutic tasks or homework to facilitate progression. Example tasks: writing an autobiography, reading the Big Book of AA and about the twelve steps.
Stress management training	Group meeting which is held by a clinical psychologist. Relaxation and imaginative elements are included in this therapeutic form. Example session: teaching and practicing basic elements of autogenic training.

Name of the session	Short description and examples
Group for affected family members	Group meeting where affected family members of the inpatients are included. The meetings' focus is not on the patient but on providing support and opportunity for consultations for family members. Example session: discussing how the affected family members trying to cope with the individual showing problematic alcohol use.
AA meeting	Patients are required to participate in AA meetings on a daily basis. This therapeutic form is held outside of the treatment site.
Therapeutic forms on Saturday and Sunday	Patients are only required to stay on the treatment site on the first weekend of the treatment program. Therefore, the treatment forms presented in V/Table 1 on these days are not relevant on other weeks of treatment program.

V/3/2. Measures

V/3/2/1. Alcohol Use Disorders Identification Test (AUDIT). The 10-item long AUDIT was used to assess the degree of harmful alcohol consumption and consequences (Gerevich et al., 2006; Saunders et al., 1993). Participants were assessed with the instrument before the beginning of the treatment program. In line with the assumed unidimensional structure of the scale (Skogen et al., 2019), total scale point was used for analyses. The scale had sufficient internal consistency in the present sample (Cronbach's $\alpha = 0.72$).

V/3/2/2. Brief Symptom Inventory (BSI). The present study assessed psychopathological symptom severity using the 53-item long BSI at the beginning and at the end of the treatment program (Derogatis & Savitz, 2000; Unoka et al., 2004). Previous research findings supported that the BSI was an appropriate instrument to reflect on the hierarchical structure of psychiatric symptoms by assessing general and specific factors of psychopathology simultaneously (Urbán et al., 2014), therefore general symptom severity, anxiety, depression, hostility, interpersonal sensitivity, obsessive-compulsive, paranoid ideation, phobic anxiety, psychoticism, and somatization scale scores were considered for analyses. The subscales of the BSI displayed acceptable levels of internal consistency at both measurement points (pre-treatment: Cronbach's $\alpha = 0.73 – 0.89$; post-treatment: Cronbach's $\alpha = 0.68 – 0.86$).

V/3/2/3. Drinking Motivations Questionnaire–Revised (DMQ-R). In order to assess the motives underlying drinking behavior, the 20-item long DMQ-R was used at the beginning of the treatment program (Kuntsche et al., 2006a; Németh, Urbán, et al., 2011). The four subscales of the questionnaire (conformity, coping, enhancement, and social motives) provided satisfactory degree of internal consistency in the present sample (Cronbach's $\alpha = 0.79 – 0.90$).

V/3/3. Data analysis

Latent class growth analysis (LCGA) was used to identify subgroups of participants based on the evaluation of psychopathological symptom profiles and change trajectories (Jung & Wickrama, 2008). Average item scores of the BSI subscales assessed at the beginning and at the end of the treatment were specified as continuous indicator variables. According to the LCGA approach, within-class variances were set to zero. Starting with the most parsimonious, one-class solution, models with a growing number of latent

classes were assessed during an iterative estimation process. The level of model fit was evaluated based on various indices. The most sufficient model should be characterized with lower rates of Akaike Information Criteria (AIC), Bayesian Information Criteria (BIC), Sample Size Adjusted Bayesian Information Criteria (SSA-BIC), and higher level of entropy. More close fit to the data should be considered in case of a significant result of the Lo-Mendel-Rubin Adjusted Likelihood Ratio Test (LMRT) for a given model compared to the previous model with fewer latent classes. Pairwise missing data handling was used. Therefore, those who only had data at the first measurement point only contributed to the estimation of parameters related to the beginning of the treatment, while parameters related to the end of the treatment were estimated based on only those participants' responses who had data at the second measurement point (covariance coverage = 69.8 – 100%).

Next, the identified latent classes were validated by analyzing their relationship with age, gender, family history of substance misuse, previous suicide attempt, psychiatric-, AUD-, or SUD-related pre-care before the treatment program, level of harmful alcohol consumption, drinking motives, and treatment reliability change index (RCI) (Jacobson & Truax, 1992). The RCI provides a standardized assessment for each participant as to whether an individual change score is statistically significantly different from a difference that could have occurred due to random measurement error alone. It considers the difference of the post- and pre-treatment score, which is divided by standard error of the differences (Ferguson et al., 2002). It is important to note, that it does not inform whether a statistically significant change was caused by a particular intervention program. The validation analyses were carried out with multinomial logistic regression (R3Step) and the Bolck-Croon-Hagenaars (BCH) method (Asparouhov & Muthén, 2013, 2014b). Mplus 8.0 and SPSS Statistics 25.0 statistical software were used to perform the analyses.

V/4. Results

V/4/1. Sample characteristics

Sample characteristics are presented in V/Table 3. Higher proportion of the participants were male, reported a family history of SUD, and most of the respondents reported a psychiatric-related or AUD-related treatment engagement in their lifetime or shortly before the treatment program. Over two-thirds of the participants successfully completed the eight-week long MM program. Most frequently, the treatment was interrupted

because of alcohol consumption during the program, while other participants also reported about treatment-based reasons (e.g. ambivalence towards the aims and assignments of the program) non-treatment-based reasons (e.g. occupational, relationship, or administrative problems). or other or undefined reasons for treatment interruption (e.g. patient did not return to the program after weekend).

V/Table 3. Descriptive characteristics of the sample in terms of socio-demographics, psychiatric anamnesis and treatment completion (*N* = 303)

Sample characteristics	
Gender *N* (%)	
Females	123 (40.59%)
Males	180 (59.41%)
Age M (SD)	46.43 (10.32)
Family history of substance misuse *N* (%)	
Yes	197 (65.02%)
No	105 (34.65%)
Previous suicide attempt *N* (%)	
Yes	58 (19.14%)
No	244 (80.53%)
Psychiatric-, AUD- or SUD-related treatment involvement history *N* (%)	
Yes	294 (97.03%)
No	9 (2.97%)
Psychiatric- or AUD-related pre-care within 1 months before the treatment program *N* (%)	
Yes	248 (81.85%)
No	55 (18.15%)
Treatment completion statistics	
Treatment completion status *N* (%)	
Successful treatment completion	218 (71.95%)
Interruption of the treatment before completion	85 (28.05%)
Reasons of treatment interruption *N* (%)	
Alcohol consumption	27 (31.76%)
Treatment-based reasons[1]	20 (23.53%)
Non-treatment-based reasons[2]	18 (21.18%)
Other or undefined reasons[3]	20 (23.53%)

Note. [1]Treatment-based reasons: ambivalence towards the aims and assignments of the program, non-completion of the assignments of the treatment, violation of treatment rules (e.g., use of mobile phone), acting out, feelings of doubt about the necessity of treatment, unable to work or open up in group psychotherapeutic sessions, reassignment to psychiatric inpatient department because of severe depressive symptoms, unable to

continue treatment because of the circumstances in the treatment department. [2]Non-treatment-based reasons: occupational, relationship, or administrative problems. [3]Other or undefined reasons: patient did not return to the program after weekend without notification, no available reason of treatment interruption.

V/4/2. Latent class growth analysis (LCGA)

A latent class growth analysis (LCGA) was performed to identify latent classes based on distinct symptomatic profiles and to examine psychopathological symptom change trajectories. Models which contained one to four latent classes were evaluated. V/Table 4 contains the fit indices for the LCGA models with different number of latent classes. The four-class solution presented the lowest rates of AIC, BIC and SSA-BIC. However, the LMRT showed a non-significant result ($p > 0.05$) for the model with four latent classes. Therefore, the inclusion of an additional subgroup in the model over three latent classes did not contribute to a more optimal degree of model fit. For further analyses the three-class model was retained. In case of the three-class solution, the average latent class probabilities for the most likely latent class membership were 0.94, 0.96, and 0.98, respectively.

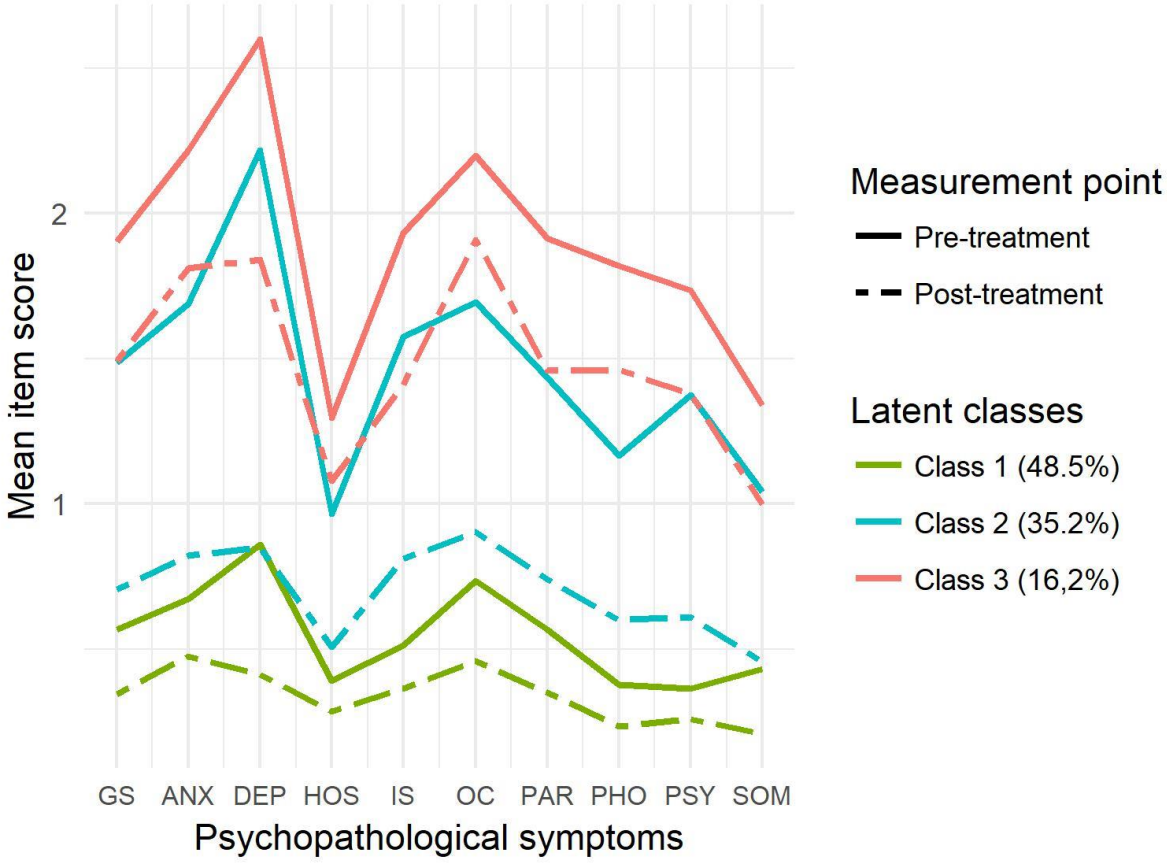
V/Table 4. Fit indices for the latent class growth analysis models based on the scales of the Brief Symptom Inventory

	AIC	BIC	SSA-BIC	Entropy	LMRT	*p*
1-class model	10618.05	10766.34	10639.48			
2-class model	8466.55	8692.68	8499.22	0.955	2175.36	< 0.001
3-class model	7811.61	8115.59	7855.53	0.946	691.17	0.016
4-class model	7386.42	7768.26	7441.60	0.935	463.32	0.403

Note. AIC = Akaike Information Criteria; BIC = Bayesian Information Criteria; SSA-BIC = Sample Size Adjusted Bayesian Information Criteria; LMRT = Lo-Mendel-Rubin Adjusted Likelihood Ratio Test.

V/Figure 1 and V/Table 5 demonstrate the symptom profiles of the three identified latent classes at the beginning and the end of the treatment program. Apart from the change of

hostility for Class 3, latent classes showed significant decreases in each dimension of psychopathological symptoms. Generally, within each latent class, participants experienced anxiety, depression, and obsessive-compulsive symptoms at the highest severity levels. Individuals assigned to Class 1 ("low severity symptomatic subgroup with mild decrease") had low-severity symptom profiles at both measurement points. For example, at the beginning of the treatment period, a 43-year-old female patient who was a member of this class reported experiencing most frequently psychiatric symptoms *"slightly"* and *"not at all"*. At the end of the treatment, this patient was predominantly free of psychiatric symptoms. Individuals assigned to Class 2 ("moderate severity symptomatic subgroup with strong decrease") had moderate levels of symptomatic severity at the beginning of the treatment, but low levels of symptomatic severity by the end of the program. For example, at the beginning of the treatment period, a 47-year-old male patient who was a member of this class reported experiencing most frequently symptoms of (i) anxiety, interpersonal sensitivity, obsessive-compulsivity, paranoid ideation, psychoticism and somatization *"slightly"* and *"moderately"*, (ii) phobic anxiety *"moderately"* and *"fairly"*, and (iii) depressive symptoms *"fairly"* and *"extremely"*. At the end of the treatment, this patent mostly reported experiencing psychiatric symptoms *"slightly"* and *"not at all"*. Individuals assigned to Class 3 ("high severity symptomatic subgroup with moderate decrease") had high levels of symptomatic severity at the beginning of the treatment program, but moderate levels of symptomatic severity by the end of the program. For example, at the beginning of the treatment period, a 59-year-old female patient who was a member of this class reported experiencing most frequently symptoms of (i) psychoticism *"moderately"* or *"not at all"*, (ii) anxiety, depression, hostility, interpersonal sensitivity, paranoid ideation and somatization *"moderately"* and *"fairly"*, and (iii) obsessive compulsivity *"fairly"* and *"extremely"*. At the end of the treatment this patient most frequently experienced symptoms of (i) hostility and psychoticism *"not at all"* and *"slightly"*, (ii) anxiety, depression and paranoid ideation *"slightly"*, (iii) interpersonal sensitivity *"slightly"* and *"moderately"*, (iv) phobic anxiety *"slightly"* and *"extremely"*, (v) symptoms of obsessive-compulsivity *"moderately"*, and (vi) symptoms of somatization *"fairly"*.



V/Figure 1. Mean item scores of the three latent classes on the subscales of the BSI before and after the treatment program. Abbreviations: GS = Global symptom severity, ANX = Anxiety; DEP = Depression; HOS = Hostility; IS = Interpersonal sensitivity; OC = Obsessive-compulsive; PAR = Paranoid ideation; PHO = Phobic anxiety; PSY = Psychoticism; SOM = Somatization.

V/4/3. Validation of the latent classes

The identified latent classes were contrasted in terms of alcohol consumption-related variables. V/Table 5 shows the results of the multiple comparisons. The “low severity” subgroup significantly demonstrated the lowest rates on alcohol consumption-related variables at the beginning of the treatment program. The “moderate severity” and “high severity” subgroups significantly demonstrated higher levels of harmful alcohol consumption and drinking motives at the beginning of the treatment program.

V/Table 5. Comparisons of the latent classes in terms of alcohol-related variables

	Class 1 "Low severity symptomatic subgroups with mild decrease" *N* = 146; 48.5%	Class 2 "Moderate severity symptomatic subgroup with strong decrease" *N* = 106; 35.2%	Class 3 "High severity symptomatic subgroup with moderate decrease" *N* = 49; 16.2%	Overall Wald test (*p*)
Parameter estimates of LCGA				
Global symptom severity I (S)	0.57 (-0.22)	1.49 (-0.78)	1.90 (-0.41)	
Anxiety I (S)	0.67 (-0.20)	1.69 (-0.87)	2.22 (-0.41)	
Depression I (S)	0.86 (-0.45)	2.22 (-1.37)	2.60 (-0.76)	
Hostility I (S)	0.39 (-0.10)	0.97 (-0.46)	1.30 (-0.22)	
Interpersonal sensitivity I (S)	0.51 (-0.15)	1.58 (-0.76)	1.93 (-0.52)	
Obsessive compulsive I (S)	0.74 (-0.28)	1.69 (-0.79)	2.20 (-0.29)	
Paranoid ideation I (S)	0.57 (-0.22)	1.43 (-0.69)	1.92 (-0.46)	
Phobic anxiety I (S)	0.38 (-0.14)	1.17 (-0.57)	1.82 (-0.36)	
Psychoticism I (S)	0.37 (-0.11)	1.37 (-0.76)	1.74 (-0.36)	
Somatization I (S)	0.43 (-0.22)	1.04 (-0.59)	1.34 (-0.34)	
Comparisons				
Harmful alcohol consumption[1] M (SE)	-0.27 $(0.09)_b$	0.22 $(0.09)_a$	0.32 $(0.15)_a$	20.93 (< 0.001)
Conformity drinking motive[1] M (SE)	-0.23 $(0.08)_b$	0.18 $(0.13)_a$	0.34 $(0.20)_a$	12.89 (0.002)
Coping drinking motive[1] M (SE)	-0.40 $(0.10)_b$	0.41 $(0.09)_a$	0.29 $(0.17)_a$	39.37 (< 0.001)
Enhancement drinking motive[1] M (SE)	-0.23 $(0.09)_b$	0.17 $(0.10)_a$	0.37 $(0.21)_a$	12.21 (0.002)
Social drinking motive[1] M (SE)	-0.18 $(0.09)_b$	0.18 $(0.11)_a$	0.14 $(0.20)_{a,b}$	6.94 (0.031)

	Class 1 "Low severity symptomatic subgroups with mild decrease" *N* = 146; 48.5%	Class 2 "Moderate severity symptomatic subgroup with strong decrease" *N* = 106; 35.2%	Class 3 "High severity symptomatic subgroup with moderate decrease" *N* = 49; 16.2%	Overall Wald test (*p*)
Reliable Change Index (RCI) – Global Symptom Severity[2] M (SE)	-1.90 (0.29)$_a$	-7.73 (0.59)$_b$	-2.83 (1.18)$_a$	77.13 (< 0.001)

Note. The presented parameter estimates of LCGA are intercepts (I) and slopes (S) in brackets. Except the change of Hostility for Class 3 ($p = 0.177$), mean slope estimates in each latent classes were significant at least $p < 0.05$ level. In the case of comparisons, means (standard errors in brackets) in the same row that do not share subscripts differ at $p < 0.05$ level. BCH method was used in the comparison (Asparouhov & Muthén, 2014b). [1]Variables measured at pre-treatment and standardized (variables' mean equals to 0 and standard deviation equals to 1) in order to ease interpretation. [2]Lower values represent more reliable symptom decrease in terms of global psychopathological severity.

Next, multinomial logistic regression analysis was performed to examine the association between latent class membership and psychopathological history-related and alcohol consumption-related covariates (V/Table 6). The "low severity" subgroup was selected as the reference category. The presence of family history of substance misuse, absence of pre-care before the treatment program, higher rates of coping drinking motives and harmful alcohol consumption all significantly increased the odds of being in the "moderate severity" subgroup membership compared to the reference category. In the case of the "high severity" subgroup, higher rates of conformity and coping drinking motives significantly contributed to the class membership compared to the reference category.

V/Table 6. Odds ratios (95% Confidence Intervals) of the association between validating covariates and latent class membership relative Class 1 ("Low severe symptomatic subgroups with mild decrease").

	Class 2 "Moderate severity symptomatic subgroup with strong decrease" *N* = 105; 34.9% OR [95% CI]	Class 3 "High severity symptomatic subgroup with moderate decrease" *N* = 49; 16.2% OR [95% CI]
Age	1.01 [0.98 – 1.05]	1.02 [0.97 – 1.08]
Gender[1]	1.36 [0.66 – 2.82]	1.99 [0.75 – 5.29]
Family history of substance misuse[2]	**2.13 [1.04 – 4.33]**	2.25 [0.91 – 5.58]
Previous suicide attempt[2]	1.39 [0.60 – 3.19]	1.75 [0.66 – 4.67]
Psychiatric-, AUD- or SUD-related pre-care shortly before the treatment program [2]	**0.36 [0.14 – 0.90]**	0.35 [0.11 – 1.13]
Harmful alcohol consumption	**1.48 [1.00 – 2.19]**	1.31 [0.72 – 2.38]
Conformity drinking motive	1.50 [0.93 – 2.40]	**1.81 [1.06 – 3.08]**
Coping drinking motive	**2.53 [1.65 – 3.88]**	**1.85 [1.03 – 3.31]**
Enhancement drinking motive	1.10 [0.70 – 1.72]	1.36 [0.64 – 2.91]
Social drinking motive	1.19 [0.74 – 1.92]	1.65 [0.79 – 3.46]

Note. Odds ratios presented by bold figures are significant at least $p < 0.05$ level. [1]Gender: 0 = Female, 1 = Male; [2]Categorical variables coded as 0 = No, 1 = Yes.

In terms of the symptom change reliability index, individuals in the "moderate severity" subgroup significantly demonstrated the highest rates of reliable symptom decreases (V/Tables 5 and 7). Compared with members of the other classes, they showed the highest level of non-random measurement error-based symptom decrease (V/Table 5), and significantly higher proportion of this class was categorized with reliable symptom decrease (as opposed to non-reliable change or reliable increase of symptoms) compared to the "light severity" class.

In terms of treatment completion, 24.7%, 27.4% and 38.3% treatment attrition rates were presented for the "low severity", "moderate severity" and "high severity" classes, respectively. There was a non-significant relationship between treatment completion status and latent class membership (V/Table 7).

V/Table 7. Association between treatment completion status, reliable change index categories and latent class membership

	Class 1 "Low severity symptomatic subgroups with mild decrease" *N* = 146; 48.5%	Class 2 "Moderate severity symptomatic subgroup with strong decrease" *N* = 106; 35.2%	Class 3 "High severity symptomatic subgroup with moderate decrease" *N* = 49; 16.2%
Treatment completion status			
Successfully completed treatment; *N* = 218 (71.9%)	110 (75.3%)	77 (72.6%)	30 (61.2%)
Dropped from treatment; *N* = 85 (28.1%)	36 (24.7%)	29 (27.4%)	19 (38.3%)
OR [95% CI]* - Successful completion	*Ref.*	0.87 [0.49 – 1.54]	0.52 [0.26 – 1.03]
Categories of Reliable Change Index (Global symptom severity)			
Reliable decrease of symptoms *N* = 134 (63.8%)	50 (47.2%)	66 (88.0%)	18 (62.1%)
Non-reliable change *N* = 59 (28.1%)	49 (46.2%)	5 (6.7%)	5 (17.2%)
Reliable increase of symptoms *N* = 17 (8.1%)	7 (6.6%)	4 (5.3%)	6 (20.7%)
OR [95% CI]* - Reliable decrease	*Ref.*	**8.21 [3.71 – 18.17]**	1.83 [0.79 – 4.25]

Note. Percentages in each cells represents the proportion within each latent classes. Treatment completion status: $\chi^2(2) = 3.66$; $p = 0.160$. Categories of Reliable Change Index: $\chi^2(4) = 44.05$; $p < 0.001$. OR: odds ratio. CI: confidence interval. *Comparison group is Class 1 (*Ref.* = reference group). A dichotomous outcome variable was constructed for comparisons: 0 = Non-reliable change or reliable increase of symptoms, 1 = Reliable decrease of symptoms. OR presented with bold figures are significant at least $p < 0.05$ level.

V/5. Discussion

The present study aimed to identify subgroups of participants with AUD attending a twelve-step based treatment program based on psychopathological symptom profiles and change trajectories. To the best of the authors' knowledge, no previous study has examined how latent classes of AUD change during treatment attendance in terms of co-occurring psychopathological symptom levels. Three latent classes were identified: (i) a low severity symptomatic subgroup at baseline with mild decrease, (ii) a moderate

severity symptomatic subgroup at baseline with strong decrease, and (iii) a high severity symptomatic subgroup at baseline with moderate decrease.

In line with some of the previous findings, quantitative differences were observed between the subgroups (Urbanoski et al., 2015; Villalobos-Gallegos et al., 2017). Namely, classes were separated by symptom profiles with intensifying severity at both measurement points. However, it is important to note, the present study had limited assessment of externalizing characteristics (e.g., absence of antisocial personality disorder, drug misuse, etc.) which might have influenced characteristics of the latent classes. This data pattern also corresponds with the concept of hierarchical structure of psychopathology: a higher order dimension of internalizing psychopathology might explain the interrelations of psychopathological symptoms within each class and represent a severity-based risk for experienced psychopathological difficulties (Kotov et al., 2017; Villalobos-Gallegos et al., 2017).

The identified latent class model is also comparable with previous studies using latent class analysis in treatment seeking or general population samples of individuals with AUD. The “low severity” subgroup had comparable symptom profile to the “low comorbidity” AUD subtype by Müller et al. (2020), the “mild” class by Villalobos-Gallegos et al. (2017) or the “comorbidity unaffected” group by Glass et al. (2014). The “moderate severity” subgroup corresponded with the “moderate” class by Villalobos-Gallegos et al. (2017). Finally, the “high severity” subgroup presented similar characteristics to the “internalizing comorbidity” group by Glass et al. (2014), “multimorbidity” class by Urbanoski et al. (2015) or the “moderate/severe” class by Villalobos-Gallegos et al. (2017).

The subgroups not only differed by severity of symptoms, but also showed different levels of symptomatic change during the eight-week long period. Symptom decrease with the largest and most reliable (non-measurement error-related) effect was demonstrated in the case of the “moderate severity” subgroup. The “low severity” and “high severity” subgroups demonstrated significant but less intensive attenuation in each of the psychopathological domains. However, it is important to highlight, that design of the present study (e.g., lack of randomized controlled trial) was unable to determine whether these patterns of symptomatic decrease were attributable to the effect of the Minnesota Model treatment. Therefore, it limits the possibility of linking the present findings to

previous twelve-step based treatment related research data which has demonstrated that attendance in these interventions can lead to attenuation of psychopathological symptoms (e.g., depression symptoms) (Worley et al., 2012). However, by using a latent class-based approach it was possible to examine more specifically if different subgroups of AUD demonstrated different trajectories in terms of psychopathological symptom reductions (Lanza & Rhoades, 2013).

The present study also explored the association between latent class membership and alcohol consumption-related variables. Participants in the moderate and high severity symptomatic subgroups presented significantly higher rates of baseline harmful alcohol consumption. These findings are consistent with previous empirical research which have demonstrated that some subgroups of AUD with increased severity of alcohol misuse are also characterized with more serious internalizing and externalizing symptoms (Hildebrandt et al., 2017; Moss et al., 2010). Additionally, multivariate analyses identified the substantial role of baseline coping and conformity motives in the cases of the more severely affected classes. Both coping and conformity motives have been described as negative reinforcement-based motives of drinking which are implicated in self-medication tendencies of the participants. Regarding the coping motives, it was assumed that that alcohol consumption serves as a form of emotion regulation among patients with a higher severity symptomatic level, which helps individuals mitigate and cope with unpleasant feelings and emotions (Berking et al., 2011). Previous studies have also demonstrated that subtypes of AUD with elevated internalizing symptomatology show increased rates of drinking in order to relief or self-medicate psychological distress (Hildebrandt et al., 2017; Müller et al., 2020). In the case of conformity motives, it was hypothesized that the "high severity" symptomatic class might show tendencies also to use alcohol as a means for reducing symptoms related to social anxiety (Villarosa et al., 2014).

V/5/1. Limitations

The present findings should be interpreted cautiously due to several limitations related to the study. First, the lack of control comparison group impeded to accurately interpret the efficacy of the MM in terms of psychopathological symptom reduction. Second, due to the absence of follow-up data collection, the present design did not assess the long-term alcohol use-related and psychopathological-related outcomes among the participants who

successfully completed the program and among those who dropped out from it. Third, the present study did not examine the role of potential third variables which might have mediated or moderated the treatment effect (e.g., AA involvement, comorbid psychiatric diagnosis). Fourth, it is important to consider that the composition of the present sample was based on availability of the patients, therefore the generalizability of the results to a broader population with AUD is arguably limited. For example, findings from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) and a nationally representative sample from Hungary have both shown that the lifetime treatment rates for AUD among those with AUD and in the latent class of 'Alcohol drinkers with severe dependence symptoms' are much lower than those in the present sample (Hasin & Grant, 2015; Horváth et al., 2019). As the applied treatment form was highly-structured and built upon the principles of the twelve-step approach, it might contribute to a self-selection effect of more motivated patients with a history of psychiatric treatment involvement. Fifth, as the research assessment was conducted by the treatment staff, it might had some effect on the participants' response tendencies, in addition to the possible bias in responses due to the participation of affected family members in the admission interview. Sixth, design of the present study did not allow to analyze the causal relationship between AUD and psychopathological symptoms. Finally, the effects of important covariates were not controlled during the analyses which might have influenced profile characteristics and changed trajectories of psychopathological symptoms, such as effects of detoxification in the first weeks of the treatment and potential period effects related to changes in therapeutic characteristics over the five-year period of the study.

V/6. Conclusions

The present study examined psychopathological symptom profiles and change trajectories among patients undergoing a twelve-step based MM treatment. The present study identified three severity-based subgroups of inpatients with AUD undergoing MM treatment. During the eight-week long period of the study, each of the three AUD severity classes demonstrated significant reductions in terms of psychopathological symptoms. Further studies, with more precise methodological design, are warranted to provide evidence whether structured, more intensive, and community-based residential treatment forms that facilitate twelve-step involvement can contribute to beneficial outcomes among AUD patients with more severe psychopathological symptomatic profiles

(Karriker-Jaffe et al., 2018). Previous studies have suggested that integrated treatment forms, which simultaneously address AUD-related and psychological-related impairments might have beneficial effects among patients with comorbid AUD and psychiatric disorders (McClean et al., 2014). Interventions which combined treatment approaches focusing on AUD and co-occurring psychiatric disorders were used to facilitate simultaneous improvements in symptomatology of AUD as well as comorbid disorders. For example, in the cases of comorbid AUD and internalizing psychiatric disorders, one might consider teaching effective coping and emotion-regulation strategies to control negative emotions. This includes cognitive restructuring techniques to explore and correct situational and cognitive risk processes (e.g., beliefs) underlying AUD and comorbid internalizing disorders (e.g., anxiety, depression disorders), and understanding and altering expectancies and motivational processes of alcohol use which can be associated with symptoms of negative affectivity (Morris et al., 2005; Roberts et al., 2015).

VI. Study 3: Polysubstance use is positively associated with gaming disorder symptom severity: A latent class analytical study[10,11]

VI/1. Abstract

Introduction: The link between gaming disorder (GD) and substance use among adolescents is not clear. Some studies reported positive associations whereas others suggested that alcohol and illicit drug use are not related to GD severity.

Objective: The present study aimed to identify empirically based latent classes of alcohol and illicit drug use among adolescents and explore their associations with GD symptom severity and whether endorsement of specific criteria of GD is linked to the membership of latent classes of alcohol and illicit drug use.

Methods: Data of the national sample of Hungarian adolescents from the Health Behaviour in School-aged Children (HBSC) survey (N = 2768; females: 52.08%; mean age: 16.73 years) were analysed. Measures for frequency of alcohol and illicit drug use, gaming, GD symptom severity and life satisfaction were included in the analyses.

Results: Latent class analysis discriminated four subgroups of alcohol and illicit drug use: polysubstance users, high-risk alcohol users, moderate alcohol users and infrequent substance users. Polysubstance users presented significantly higher levels of GD

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[11] Acknowledgements. (i) *Statement of Ethics*. The authors declare that all procedures followed the ethical standards of the Declaration of Helsinki. Informed consent was obtained from all participants for inclusion in the study. The study was approved by the Scientific and Research Ethics Committee of the Medical Research Council (5555-5/2018/EKU). (ii) *Conflict of Interest Statement.* Zsolt Demetrovics is a member of the Editorial Board of the journal. Other than this, the authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. (iii) *Funding Sources*. The study was supported by the Hungarian National Research, Development and Innovation Office (Grant numbers KKP126835, NKFIH-1157-8/2019-DT and TKP2020-IKA-05). Orsolya Király was supported by the János Bolyai Research Scholarship of the Hungarian Academy of Sciences and by the ÚNKP-20-5 New National Excellence Program of the Ministry for Innovation and Technology from the source of the National Research, Development and Innovation Fund. Zsolt Horváth was supported by the ÚNKP-20-3 New National Excellence Program of the Ministry for Innovation and Technology from the source of the National Research, Development and Innovation Fund. (iv) *Author Contributions*. Zsolt Horváth, Orsolya Király, Zsolt Demetrovics and Róbert Urbán wrote the manuscript; Ágnes Németh and Dóra Várnai designed the study and performed data collection; and Zsolt Horváth conducted statistical analyses under Róbert Urbán's supervision. All authors have critically revised the manuscript and approved its final version.

symptom severity and higher odds for endorsement of criteria of ‘giving up other activities’ and ‘negative consequences’.

Conclusions: Positive associations were shown between higher GD severity and the polysubstance using class. The roles of criteria of ‘giving up other activities’ and ‘negative consequences’ were highlighted in more severe substance use patterns. However, GD severity and criteria did not differ as a function of the level of alcohol use. These findings may imply common roots of GD and illicit drug use in adolescents.

Keywords: Gaming Disorder; Internet Gaming Disorder; comorbidity; alcohol use; substance use; adolescence

VI/2. Introduction

Experimental, frequent or even problematic forms of psychoactive substance use (e.g. alcohol, cannabis) and potentially addictive behaviours (e.g. gaming, gambling) can develop and occur during adolescence (van Rooij et al., 2014). According to statistics from the large cross-national Health Behaviour in School-aged Children (HBSC) survey and considering the high variability in lifetime prevalence rates between countries, more than half (females: 59%; males: 60%) of 15-year-old adolescents have drunk alcohol in their lifetime and approximately one-fifth (females: 18%; males: 22%) of them have reported lifetime drunkenness (Inchley et al., 2020b). The most frequently used illicit drug type is cannabis in this age group, with a lifetime prevalence rate of 13% (Inchley et al., 2020b), whereas for other illicit drug types (e.g. ecstasy, amphetamine) the prevalence rates are around 1–2% (ESPAD Group, 2016). Problematic gaming behaviour can be conceptualized as a continuum of severity of symptoms, impairments and negative consequences related to gaming (American Psychiatric Association, 2013), which is defined in the major diagnostic systems as internet gaming disorder (IGD; defined in Section III of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-5) (American Psychiatric Association, 2013) and gaming disorder (GD; defined in the eleventh edition of the International Classification of Diseases, ICD-11) (World Health Organization, 2018b). Throughout the present paper the term ‘gaming disorder’ (GD) is used in order to facilitate consistency in terminology. However, it is important to note that the cited studies might have applied different terminology to describe problematic gaming. Based on representative data from seven European

countries, a prevalence rate of 1.6% was presented for GD among 14–17-year-old adolescents (Müller et al., 2015).

Problematic features of GD (e.g. impaired control of use, continuation despite negative consequences) overlap with symptoms related to alcohol and substance use disorders (Na et al., 2017; Smith et al., 2015). Moreover, congruous underlying psychological risk factors have been identified between these risk behaviours, such as difficulties in emotion regulation, negative affectivity, similar motivational mechanisms (e.g. reward sensitivity, self-medication tendencies – using gaming or substances to avoid or escape problems) and higher levels of impulsivity (Burleigh et al., 2019; Dong & Potenza, 2014; Na et al., 2017). GD and substance use disorders also show several shared neurobiological alterations (e.g. increased regional homogeneity in the posterior cingulate cortex), which can explain cognitive and behavioural similarities between these disorders, amongst others, related to problems in response-inhibition, cognitive control and decision-making (Dong et al., 2019; Kim et al., 2015; Kuss et al., 2018; Zsidó et al., 2019).

However, conflicting findings have been reported in the literature when the association between GD and substance use was analysed among adolescents. Some of the studies have reported that higher levels of GD were related to earlier onset of alcohol and cannabis use, higher levels of alcohol consumption, cannabis and other illicit drug use and elevated rates of problems related to alcohol and illicit drug use among adolescents (Coëffec et al., 2015; Estévez et al., 2017; Kotyuk et al., 2020; Männikkö et al., 2020; Marmet, Studer, Wicki, et al., 2019; van Rooij et al., 2014). On the other hand, various studies have found non-significant and inconclusive relationships (i.e. associations with opposite directions in a longitudinal design) among adolescents between levels of GD and alcohol consumption, cannabis use and related problems (Coëffec et al., 2015; Krossbakken et al., 2018; Mérelle et al., 2017).

Due to these inconclusive findings, further investigation is warranted in this area. Specifically, to our best knowledge, existing literature has not yet examined how the severity of GD is associated with different patterns and profiles of alcohol and illicit drug use among adolescents, with the exception of one study focusing on alcohol use among young adults (Erevik et al., 2019). Research has mostly focused on a narrow range of substances (i.e. tobacco, alcohol, cannabis) (Turel & Bechara, 2019); furthermore, it is not clear whether endorsement of specific symptoms of GD constitutes an elevated risk

for higher rates of alcohol and illicit drug use. By establishing latent classes based on alcohol and illicit drug use patterns and examining their relationships with GD severity and specific GD criteria, a more detailed understanding of substance use patterns (e.g. patterns of concurrent use of different substances, severity-based differences in substance use) for individuals with elevated levels of GD can be acquired. Studies using latent class analysis to identify distinct subgroups of substance use among adolescents repeatedly distinguished classes with no or decreased consumption of psychoactive substances, classes using predominantly single substances (e.g. alcohol consumption only), subgroups of alcohol use with increasing severity (e.g. light, moderate and heavy alcohol use) and polysubstance using latent classes characterized by concurrent use of multiple substances (e.g. cannabis and other illicit drugs) (Davoren et al., 2016; Tomczyk et al., 2016). Adolescents within latent classes with polysubstance use and more problematic alcohol consumption patterns have presented elevated rates of internalizing and externalizing characteristics and psychological distress compared to other latent classes with less extensive substance consumption (Cranford et al., 2013; White et al., 2013).

VI/2/1. Aims

The present study aimed to identify empirically based latent classes of alcohol and illicit drug use among adolescents and explore their associations with GD symptom severity and whether endorsement of specific criteria of GD is linked to membership of latent classes of alcohol and illicit drug use. To our best knowledge, previous studies have not applied such approaches (i.e. latent class-related and symptomatic-level analyses) to examine the co-occurrence between GD and substance use.

VI/3. Materials and methods

VI/3/1. Participants and procedure

Data from a representative Hungarian sample of the HBSC survey in 2018 were used in the present study (Németh, 2019). In line with the international HBSC protocol, the sample was randomly drawn from the list of educational institutions and classes. The sampling unit was the class. Stratification was used during sampling according to the type of education, school grade, maintainer of the school, geographical region and settlement type. Data collection was performed among students attending fifth-, seventh-, ninth and eleventh grades. Informed consent was obtained at school (e.g. headmaster) and student levels, whereas passive consent was required from parents. Overall, the 2018 HBSC

Hungarian sample consisted of data from 6003 students. However, because data regarding illicit drug use was only collected among students in the ninth and eleventh grades, adolescents in lower grades were excluded from the present analyses. Consequently, the final sample comprised responses from 2768 students: 52.08% (*N* = 1439) females, 58.78% (*N* = 1627) ninth grade students and mean age 16.73 years (SD = 1.21).

VI/3/2. Measures

VI/3/2/1. Alcohol consumption. The level of alcohol consumption was assessed via four questions in the present study. Separate questions measured the frequency of alcohol use and drunkenness in the participants' lifetime and past month (i.e. past 30 days). Originally, both alcohol use-related items were rated on a seven-point scale (1 = *Never*; 7 = *30 days or more*) and adolescents provided responses on a five-point scale for both drunkenness-related items (1 = *No, never*; 5 = *Yes, more than 10 times*). However, in order to facilitate interpretability of the latent classes and avoid potential methodological issues (e.g. exaggeration of the importance of alcohol use over illicit drug use in classification) (Tomczyk et al., 2016), items were recoded into two separate variables with three frequency categories in each (alcohol use: 0 = *Lifetime abstinence of alcohol use*, 1 = *Lifetime alcohol use but not in the past month*, 2 = *Past month alcohol use*; drunkenness: 0 = *Lifetime absence of drunkenness*, 1 = *Lifetime drunkenness but not in the past month*, 2 = *Past month drunkenness*). Previous studies also used similarly coded items of alcohol use in order to identify latent classes of alcohol and illicit drug use (Gilreath et al., 2014; Göbel et al., 2016).

VI/3/2/2. Illicit drug use. Seven forms of illicit drug use were included in the present study: lifetime cannabis use; cannabis use in the past month (i.e. in the past 30 days); lifetime use of ecstasy or MDMA; lifetime use of amphetamines; lifetime non-medical use of medications; lifetime concurrent use of alcohol and medications; and lifetime use of designer drugs (i.e. synthetic cannabinoids and new psychoactive substances). Frequency of consumption for each substance was evaluated on a seven-point scale (1 = *Never*; 7 = *30 days or more*); however, due to the substantial floor effects, these variables were dichotomized (0 = *No use at all*; 1 = *Consumption at least once*). Moreover, in the case of cannabis use, a separate variable with three frequency categories was constructed (0 = *Lifetime abstinence of cannabis use*; 1 = *Lifetime cannabis use but not in the past month*; 2 = *Past month cannabis use*). Previous studies that aimed to identify substance

use-related latent classes also used binary variables to measure lifetime use of various illicit drug types and similar single-item variables to assess the frequency of cannabis use (Gilreath et al., 2014; Göbel et al., 2016; Snyder & Smith, 2015; White et al., 2013).

VI/3/2/3. Frequency of gaming. Two separate questions measured the frequency of gaming on average schooldays and on weekend days. The degree of gaming for both items was rated on a nine-point scale (1 = *Not at all*; 9 = *Approx. 7 hours or more*) and instructions of the questions required the adolescents to consider various platforms of online and offline gaming (e.g. PC, console, smartphone, tablet, etc.). Moreover, a separate variable was constructed to measure gaming status, which divided respondents into two categories (0 = *Never-gamers*; 1 = *Gamers*). Due to the high correlation between the two frequency indices of schooldays and weekend days (r = 0.73), a principal component analysis was also used to create a composite score of gaming frequency for multivariate analyses among gamers (explained variance = 86.66%; α = 0.84).

VI/3/2/4. Gaming disorder. An abbreviated, five-item version of the Ten-Item Internet Gaming Disorder Test (IGDT-10) (Király et al., 2017, 2019) was applied to measure the criteria of GD as proposed in the ICD-11: loss of control; giving up other activities; continuation of gaming despite negative consequences; and negative consequences, which was measured via two items – jeopardizing or losing a significant relationship and negative consequences on school performance. Each impairment was assessed on a three-point scale (0 = *Never*; 1 = *Sometimes*; 2 = *Often*), which allowed a score to be constructed for GD symptom severity. Furthermore, during analyses that focused on exploring the relationships with the endorsed GD criteria, each item was dichotomized in a way that was consistent with the proposal for the ten-item version of the scale (0 = *Never or Sometimes;* 1 = *Often*). A satisfactory level of internal consistency was presented (ω = 0.87).

VI/3/2/5. Life satisfaction. As a general indicator of well-being, the one-item Cantril ladder was used in the present study (Levin & Currie, 2014). Adolescents had to rate their level of satisfaction with life by using an eleven-point scale (0 = *Worst possible life*; 10 = *Best possible life*).

VI/3/3. Data analysis

Latent class analysis (LCA) was performed to distinguish subgroups with distinct patterns of alcohol consumption and drunkenness (lifetime and in the past month) and use of

various illicit drug types (cannabis use during lifetime and in the past month; lifetime uses of ecstasy and MDMA, amphetamines, designer drugs and medications with a non-medical purpose; and lifetime concurrent use of alcohol and medications). Previous studies used a similar set of indicator variables to identify typologies of substance use among adolescents (Tomczyk et al., 2016). As a first step of the LCA, more and more complex models with a growing number of latent classes were estimated. The optimal solution was that with the lowest levels of the Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC) and the sample size-adjusted Bayesian Information Criterion (SSA-BIC). However, the decision on the number of latent classes was based primarily on the outcome of the Lo-Mendel-Rubin adjusted likelihood ratio test (LMRT): a significant result ($p < 0.05$) of the LMRT for a particular model was desirable when compared to a more parsimonious, alternative model with one less latent class. Classification accuracy was represented by the entropy index and average latent class probabilities for the most likely latent class memberships (higher values indicate a more optimal solution). After selecting the best fitting latent class model, the aim was to examine the associations between GD and latent class memberships of alcohol and illicit drug use. This approach is in line with previous studies that examined associations between substance use-related latent classes and internalizing and externalizing psychological characteristics (Cranford et al., 2013; White et al., 2013). Therefore, latent classes were compared in terms of gaming status (by using chi-square statistics), gaming frequency, GD symptom severity (by using the BCH method) (Asparouhov & Muthén, 2014b) and dichotomous GD criteria (by using chi-square statistics). Comparisons regarding gaming frequency and GD-related variables were performed among gamers to ensure differentiation of non-gamers and non-problematic gamers. The relationships between GD symptom severity, relevant GD criteria and alcohol and illicit drug use-related latent classes were also assessed via multinomial logistic regression among gamers (R3Step method) (Asparouhov & Muthén, 2014a) while controlling for the effects of gender, school grade, life satisfaction and frequency of gaming. Analyses were conducted using Mplus 8.0 (Muthén & Muthén, 2017) and IBM SPSS Statistics 25.0 software.

VI/4. Results

VI/4/1. Preliminary analyses

Bivariate correlations between various measures of alcohol and illicit drug use and gaming-related variables are presented in VI/Table 1. Higher frequency of gaming on schooldays had significant, positive and weak correlations with drunkenness and cannabis use in the past month, lifetime uses of amphetamines and medications with a non-medical purpose and lifetime concurrent use of alcohol and medications. GD symptom severity presented significant, positive and weak associations with all forms of illicit drug use but not alcohol use. The 'giving up other activities' criterion showed significant, positive and weak associations with lifetime uses of amphetamines and medications with a non-medical purpose and lifetime concurrent use of alcohol and medications. Significant, positive and weak relationships were demonstrated between the endorsement of the 'negative consequences' criterion and drunkenness in the past month, cannabis use over lifetime and in the past month, lifetime uses of ecstasy, MDMA, amphetamines, medications with a non-medical purpose and designer drugs, and lifetime concurrent use of alcohol and medications. Prevalence rates, descriptive statistics and gender and school grade-based comparisons in terms of alcohol and illicit drug use- and gaming-related variables are presented in VI/Supplementary Tables 1 and 2.

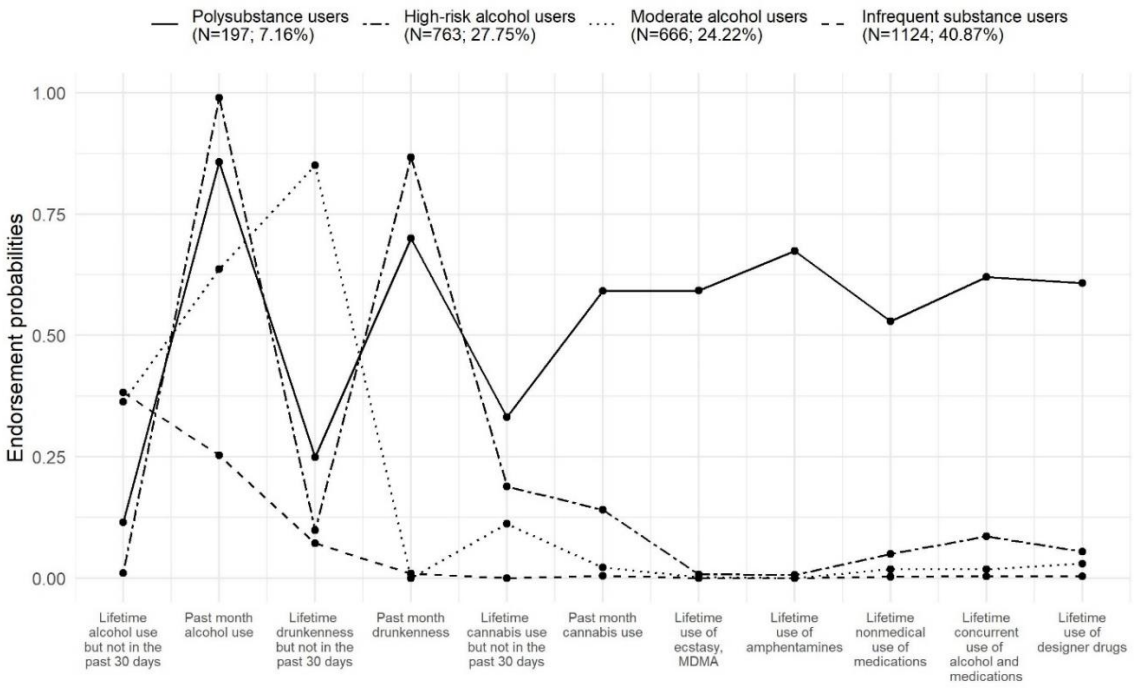
VI/Table 1. Associations between gaming frequency, gaming disorder symptom severity, gaming disorder criteria and alcohol and illicit drug use

	Gaming status (never-gamers vs. gamers)	Frequency of gaming: schooldays	Frequency of gaming: weekends	Gaming disorder symptom severity	Gaming disorder criteria			
					Loss of control	Giving up other activities	Continuation	Negative consequences
Lifetime alcohol use	-0.01	0.00	-0.03	-0.04	-0.06*	-0.06**	-0.03	-0.03
Past month alcohol use	0.01	0.04	0.01	0.02	-0.01	-0.03	0.00	-0.01
Lifetime drunkenness	-0.04	0.06**	0.01	0.02	0.00	-0.01	-0.05*	0.05*
Past month drunkenness	-0.03	**0.09*** **	0.02	0.05*	0.02	0.01	0.01	**0.08*** **
Lifetime cannabis use	-0.03	0.07**	0.03	**0.10*** **	0.03	0.04	0.02	**0.11*** **
Past month Cannabis use	-0.02	**0.11*** **	0.05*	**0.09*** **	0.06**	0.07**	0.00	**0.13*** **
Lifetime use of ecstasy, MDMA	0.02	0.07**	0.03	**0.08*** **	0.02	0.05*	0.02	0.06**
Lifetime use of amphetamines	0.02	**0.09*** **	0.02	**0.10*** **	0.03	**0.08*** **	0.03	**0.08*** **
Lifetime non-medical use of medications	0.01	**0.10*** **	0.05*	**0.12*** **	0.04	**0.11*** **	0.03	**0.12*** **
Lifetime concurrent use of alcohol and medications	-0.02	**0.09*** **	0.04	**0.08*** **	0.07**	**0.09*** **	0.04	**0.11*** **
Lifetime use of designer drugs	0.00	0.08**	0.07**	**0.11*** **	0.03	0.07**	0.04	**0.13*** **

Note. Correlations with gaming frequency- and gaming disorder-related variables are presented among those who were gamers (N = 1897–1995), whereas correlation with the presence of gaming is presented in the total sample (N = 2625–2735). Level of significance: *p < 0.05; **p < 0.01; ***p < 0.001. In order to control for familywise type I error, Pearson correlation estimates are considered significant at p < 0.001 (presented as bold values). Gaming status is coded by 0 = *Never-gamers* and 1 = *Gamers*. Variables regarding gaming disorder criteria are coded by 0 = *Never or sometimes* and 1 = *Often*. Variables regarding alcohol and illicit drug use are coded by 0 = *No* and 1 = *Yes*.

VI/4/2. Latent class analysis

LCA with one to five latent classes was performed to classify participants based on measures of alcohol consumption, drunkenness and use of various illicit drug types. Model fit indices for the latent class models are shown in VI/Supplementary Table 3. Decreasing rates of the AIC, BIC and SSA-BIC were found with the inclusion of additional latent classes in each step. However, the non-significant result of the LMRT in the case of the five-class solution indicated that it was not reasonable to select a more complex model over the model with four latent classes. As the four-class model was superior compared to the three-class solution in terms of LMRT, it was retained for further analyses. Average probabilities for the most likely latent classes were 0.92, 0.93, 0.83 and 0.89, respectively.



VI/Figure 1. Profile characteristics of the latent classes of alcohol and illicit drug use.

Class-based profile characteristics are summarized in VI/Figure 1. Members assigned to Class 1 (polysubstance users: *N* = 197; 7.16%) had very high probabilities for drinking alcohol and drunkenness in the past month and a moderately high probability for using cannabis in the past month, in addition to high probabilities for using all other types of

illicit drugs in their lifetime. Class 2 (high-risk alcohol users: *N* = 763; 27.75%) was characterized by very high probabilities for consuming alcohol and drunkenness in the past month, a low probability for cannabis use and very low rates of lifetime uses of other illicit drug types. Adolescents within Class 3 (moderate alcohol users: *N* = 666; 24.22%) showed very high probabilities for drinking alcohol in the past month and lifetime drunkenness (but not in the past month) whereas any form of lifetime illicit drug use was extremely unlikely. Finally, Class 4 (infrequent substance users: *N* = 1124; 40.87%) demonstrated a pattern of low–very low probabilities for lifetime alcohol use and drunkenness as well as alcohol consumption in the past month, with an absence of illicit drug use also being characteristic of this class.

VI/4/3. Association between latent classes of alcohol and illicit drug use and GD

Pairwise comparisons of the latent classes in terms of life satisfaction, gaming status and frequency and GD symptom severity and criteria are presented in VI/Table 2. Latent classes did not differ significantly in terms of gaming status, frequency of gaming on weekends and the GD criterion of loss of control. Polysubstance users showed significantly lower levels of life satisfaction compared to the other three classes (d = 0.30–0.45) whereas infrequent substance users had significantly higher life satisfaction compared to high-risk alcohol users (d = 0.12). Polysubstance users showed significantly higher levels of gaming frequency compared to moderate alcohol users and infrequent substance users on schooldays (d = 0.23 and 0.36, respectively); also, high-risk alcohol users practiced gaming significantly more frequently during schooldays compared to infrequent substance users (d = 0.19). The class of polysubstance users presented significantly higher rates of GD symptom severity (d = 0.36–0.43) and elevated odds to 'experience negative consequences on relationships or school performance' and to 'give up other activities because of gaming' compared to the other three classes. Moreover, polysubstance users also had significantly higher endorsement rates of the GD criterion of 'continuation' compared to moderate alcohol users.

VI/Table 2. Comparison of the latent classes of alcohol and illicit drug use

	Polysubstance users *N* = 197 (7.16%)	High-risk alcohol users *N* = 763 (27.75%)	Moderate alcohol users *N* = 666 (24.22%)	Infrequent substance users *N* = 1124 (40.87%)	χ^2 (*p*)
Life satisfaction M (SE)	6.28 (0.21)[a]	6.99 (0.08)[b]	7.03 (0.09)[b,c]	7.24 (0.06)[c]	23.60*** (< 0.001)
Presence of gaming *N* (%)	139 (72.40%)	533 (70.22%)	488 (73.49%)	833 (74.57%)	4.48 (0.214)
Gaming frequency: schooldays M (SE)	4.09 (0.22)[a]	3.73 (0.10)[a,b]	3.52 (0.11)[b,c]	3.32 (0.07)[c]	18.70*** (< 0.001)
Gaming frequency: weekend M (SE)	4.80 (0.23)[a]	4.82 (0.11)[a]	4.78 (0.13)[a]	4.66 (0.09)[a]	1.40 (0.706)
Gaming disorder symptom severity M (SE)	2.78 (0.23)[a]	1.93 (0.10)[b]	1.79 (0.10)[b]	1.86 (0.08)[b]	16.51** (0.001)
Criterion: Loss of control *N* (%)	13 (9.22%)	32 (6.07%)	23 (4.78%)	45 (5.43%)	4.24 (0.236)
OR [95% CI]	*Ref.*	0.64 [0.32 – 1.25]	0.49 [0.24 – 1.00]	0.57 [0.30 – 1.08]	
Criterion: Giving up other activities *N* (%)	19 (13.48%)	29 (5.49%)	24 (4.98%)	59 (7.13%)	14.25** (0.003)
OR [95% CI]	*Ref.*	0.37** [0.20 – 0.69]	0.34*** [0.18 – 0.63]	0.49** [0.28 – 0.85]	
Criterion: Continuation *N* (%)	24 (17.02%)	59 (11.17%)	47 (9.77%)	115 (13.91%)	8.29* (0.040)
OR [95% CI]	*Ref.*	0.61 [0.37 – 1.02]	0.53* [0.31 – 0.90]	0.79 [0.49 – 1.27]	
Criterion: Negative consequences *N* (%)	25 (14.18%)	47 (8.92%)	34 (7.08%)	56 (6.76%)	20.48*** (< 0.001)
OR [95% CI]	*Ref.*	0.45** [0.27 – 0.77]	0.35*** [0.20 – 0.62]	0.34*** [0.20 – 0.56]	

Note. Comparisons regarding frequency of gaming and gaming disorder-related variables are performed among those who were gamers. In the case of continuous variables, the BCH method was used for pairwise comparisons and the Wald test was calculated as the test statistic. Means in rows sharing the same letter are not significantly different at $p < 0.05$. Chi-square statistics (χ^2) were calculated for dichotomous variables regarding gaming status (0 = *Never-gamer*; 1 = *Gamer*) and gaming disorder criteria (0 = *Never or sometimes*; 1 = *Often*). In these cases, odds ratios (OR) with 95% confidence intervals (95% CI) were calculated to compare the latent classes, and the subgroup with the highest within-class criterion endorsement proportion (%) was selected as a reference category (*Ref.*). Level of significance in these pairwise comparisons: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

The findings of the multinomial regression analyses are displayed in VI/Table 3. In the multivariate analyses the polysubstance users presented significantly higher rates of GD symptom severity compared to the other three classes while controlling for the effects of the included covariates (see VI/Table 3, Model 1). This class of users also was characterized by significantly lower levels of life satisfaction compared to the other three classes, as well as having significantly higher odds of being in a higher school grade compared to the infrequent substance users. Moreover, significant predictive effects were demonstrated if the criteria of 'giving up other activities' and 'negative consequences' were included separately in the multinomial logistic regression analyses (see VI/Table 3, Models 2 and 4). Membership of the class of polysubstance users was significantly predicted by higher endorsement of the GD criterion of 'giving up other activities' compared to high-risk and moderate alcohol users, whereas significant, positive predictive effects of the GD criterion of 'negative consequences' on polysubstance users were shown compared to the other three latent classes. In these two models, polysubstance users had significantly lower levels of life satisfaction compared to the other three classes, and membership of this class was significantly and positively associated with male gender and higher school grade compared to infrequent substance users. Finally, the effects of the 'continuation' criterion did not remain significant when the effects of other covariates were included in the model (see VI/Table 3, Model 3).

VI/Table 3. Multinomial logistic regression: Predictors of alcohol and illicit drug use-related latent class memberships

Model 1 – Gaming disorder symptom severity			
	High-risk alcohol users OR [95% CI]	Moderate alcohol users OR [95% CI]	Infrequent substance users OR [95% CI]
Gender[1]	1.00 [0.61 – 1.65]	0.77 [0.47 – 1.27]	0.66 [0.41 – 1.07]
School grade[2]	1.02 [0.66 – 1.57]	1.05 [0.68 – 1.62]	**0.27 [0.18 – 0.42]**
Life satisfaction	**1.17 [1.03 – 1.33]**	**1.16 [1.02 – 1.31]**	**1.23 [1.09 – 1.39]**
Frequency of gaming	1.12 [0.85 – 1.46]	1.07 [0.82 – 1.40]	0.92 [0.72 – 1.19]
Gaming disorder symptom severity	**0.82 [0.73 – 0.93]**	**0.82 [0.73 – 0.92]**	**0.87 [0.78 – 0.98]**
Model 2 – Criterion: Giving up other activities			
	High-risk alcohol users OR [95% CI]	Moderate alcohol users OR [95% CI]	Infrequent substance users OR [95% CI]
Gender[1]	0.87 [0.54 – 1.40]	0.66 [0.41 – 1.06]	**0.60 [0.38 – 0.94]**
School grade[2]	1.01 [0.66 – 1.56]	1.04 [0.67 – 1.60]	**0.27 [0.18 – 0.42]**
Life satisfaction	**1.18 [1.04 – 1.33]**	**1.17 [1.04 – 1.32]**	**1.23 [1.10 – 1.38]**
Frequency of gaming	1.04 [0.81 – 1.32]	0.99 [0.78 – 1.26]	0.86 [0.69 – 1.08]
Criterion: Giving up other activities	**0.35 [0.17 – 0.75]**	**0.34 [0.16 – 0.74]**	0.64 [0.33 – 1.25]
Model 3 – Criterion: Continuation			
	High-risk alcohol users OR [95% CI]	Moderate alcohol users OR [95% CI]	Infrequent substance users OR [95% CI]
Gender[1]	0.85 [0.53 – 1.37]	0.65 [0.40 – 1.04]	**0.57 [0.36 – 0.90]**
School grade[2]	1.01 [0.66 – 1.56]	1.04 [0.67 – 1.60]	**0.27 [0.18 – 0.41]**
Life satisfaction	**1.18 [1.05 – 1.34]**	**1.18 [1.04 – 1.33]**	**1.25 [1.11 – 1.40]**
Frequency of gaming	1.02 [0.79 – 1.30]	0.98 [0.77 – 1.24]	0.81 [0.65 – 1.03]
Criterion: Continuation	0.61 [0.33 – 1.13]	0.55 [0.29 – 1.04]	1.12 [0.64 – 1.97]
Model 4 – Criterion: Negative consequences			
	High-risk alcohol users OR [95% CI]	Moderate alcohol users OR [95% CI]	Infrequent substance users OR [95% CI]
Gender[1]	0.67 [0.41 – 1.08]	0.87 [0.54 – 1.41]	**0.61 [0.39 – 0.97]**
School grade[2]	1.03 [0.67 – 1.60]	1.01 [0.65 – 1.56]	**0.27 [0.18 – 0.42]**

Life satisfaction	**1.17 [1.03 – 1.32]**	**1.18 [1.04 – 1.33]**	**1.23 [1.09 – 1.38]**
Frequency of gaming	0.98 [0.77 – 1.24]	1.01 [0.79 – 1.29]	0.88 [0.70 – 1.10]
Criterion: Negative consequences	**0.39 [0.19 – 0.77]**	**0.48 [0.26 – 0.91]**	**0.45 [0.25 – 0.81]**

Note. Reference category = polysubstance users. Analyses were performed among gamers. Significant ($p < 0.05$) regression coefficients (odds ratios with 95% confidence intervals) are presented as bold values. [1]Coded as: 0 = Female; 1 = Male. [2]Coded as: 0 = ninth graders; 1 = eleventh graders. [3]Coded as: 0 = *Never or sometimes*; 1 = *Often*.

VI/5. Discussion

The present study aimed to examine the associations between empirically derived typologies of alcohol and illicit drug use and GD symptom severity and GD criteria among adolescents. To our best knowledge, this is the first time that the co-occurrence between GD and alcohol and illicit drug use has been investigated via latent class-related and symptomatic-level analyses.

Four, quantitatively and qualitatively different subgroups were discriminated with distinct patterns of alcohol and illicit drug use: polysubstance users, high-risk alcohol users, moderate alcohol users and infrequent substance users. High-risk alcohol users, moderate alcohol users and infrequent substance users were predominantly characterized by single substance use patterns (i.e. only alcohol use) and formed a continuum of alcohol use severity, with a rise of alcohol use and drunkenness frequency in the more severe classes. However, polysubstance users differed considerably from the other classes by presenting a pattern of concurrent use of multiple substances (e.g. not only frequent alcohol use but also high rates of illicit drug use). It is important to note that in the literature there is no clear consensus on the definition of polysubstance use. The diagnostic category of polysubstance dependence in DSM-IV (which is no longer considered in DSM-5) refers to a pattern of using three or more substances (not caffeine or nicotine) where substance use-related problems emerge due to the combination of consumed substances rather than the substances per se (Schuckit et al., 2001). More recently, it was suggested that polysubstance use should be considered when an adolescent uses three or more substances, which most frequently refers to the combined use of alcohol, tobacco and cannabis (Tomczyk et al., 2016). Our latent class model is in line with the typologies suggested previously. For example, multiple studies have

reported differentiating subgroups according to patterns of use: low or infrequent alcohol use (i.e. infrequent substance users); mostly consuming just alcohol with high frequency and with increasing levels of excessive forms of alcohol use, such as drunkenness (i.e. moderate and high-risk alcohol users); and concurrent use of multiple substances (i.e. polysubstance users) (Davoren et al., 2016; Tomczyk et al., 2016).

Comparison of the latent classes revealed that membership of the class of polysubstance users was positively and mostly weakly associated with higher levels of GD symptom severity compared to the other classes, and this difference remained significant even after controlling for age, gender, frequency of gaming and life satisfaction. This pattern is consistent with previous findings, which reported positive, moderate–weak associations between GD severity and cannabis use and other, unspecific measures of illicit drug use (Männikkö et al., 2020; Muñoz-Miralles et al., 2016; van Rooij et al., 2014; Walther et al., 2012). Similarly, based on a hypothesized U-shaped association between gaming frequency and illicit drug use, a very high level of gaming was positively associated with the use of various types of illicit drugs among adolescents, such as marijuana, amphetamines and MDMA (Turel & Bechara, 2019). The positive and weak associations between lifetime uses of some types of illicit drugs (e.g. stimulants such as amphetamines) and GD symptom severity might suggest that adolescents consume some substances in order to achieve cognitive enhancement and improve their performance in games (Burleigh et al., 2019; Škařupová et al., 2018). It is possible that the co-occurrence between the polysubstance use pattern and GD symptom severity can be explained by processes of within-person convergence of risk-taking and problem behaviours among adolescents (de Looze et al., 2015; Jessor, 1991; Ong et al., 2016). In line with this, in previous studies, classes with a similar polysubstance use profile were characterized with elevated rates of externalizing characteristics but also showed a risk for internalizing problems (Tomczyk et al., 2016). Moreover, this relationship highlights the possibility of shared psychological mechanisms underlying GD and illicit drug use: elevated reward and sensation-seeking tendencies might contribute to extensive substance-seeking behaviour and the emergence of problematic features of GD (Dong & Potenza, 2014; Smith et al., 2015). Finally, it is important to consider that shared neurobiological mechanisms can also explain the association between GD and polysubstance use (Kuss et al., 2018).

Bivariate analyses and comparison of the latent classes with increasing levels of alcohol use (i.e. differences between infrequent substance users, moderate alcohol users and high-risk alcohol users) indicated that the level of GD symptom severity did not differ as a function of the level of alcohol use. These findings are in line with those studies that reported non-significant associations between different forms of alcohol consumption and GD (Brunborg et al., 2014; Coëffec et al., 2015; Männikkö et al., 2020; Mérelle et al., 2017; Walther et al., 2012). From a neurobiological perspective, the non-significant relationship between alcohol use and GD symptom severity might highlight the possibility that the two risk behaviours share common neurobiological mechanisms instead of the clinical severity of each risk behaviour per se, thus GD and increased alcohol use might represent different expressions of the same neurobiological alterations (Kuss et al., 2018).

The present study also investigated the differential effects of the GD criteria on the typologies of alcohol and illicit drug use. The 'giving up other activities' and 'experiencing negative consequences related to relationships or school performance because of gaming' criteria were more likely to be endorsed by polysubstance users. These effects remained significant even after controlling for age, gender, frequency of gaming and life satisfaction. Previous studies using adolescent and adult samples have consistently suggested that the 'giving up other activities' criterion has high discrimination and information capacity at high levels of GD severity, whereas the criterion of 'negative consequences' is associated with lower levels of GD severity (Gomez et al., 2019; Király et al., 2017; Rehbein et al., 2015). As both criteria can represent adverse social effects or impairment of the individual's life due to gaming (e.g. decrease in other leisure activities, relationship satisfaction, school performance), their relationships with polysubstance use might represent an attempt to counterbalance or mitigate negative psychological states related to negative social consequences: namely, excessive gaming might lead to less satisfactory peer relationships and behaviour- and emotional-bonding to school, which can motivate heightened substance-seeking behaviour among affected adolescents, partly mediated by the possible difficulties in terms of emotion-regulation and coping (Dickens et al., 2012; Estévez et al., 2017; Männikkö et al., 2020; Milani et al., 2018). However, it cannot be ruled out that the relationship between the polysubstance using pattern and the above-mentioned GD criteria might have the opposite effect. Based on previous studies examining the

interaction between substance use and gambling behaviour (Baron & Dickerson, 1999; Ellery et al., 2005), it might be possible that consumption of psychoactive substances in certain cases leads to excessive gaming (e.g. consuming psychoactive substances prior to gaming can weaken the capacity to control gaming behaviour), which might subsequently contribute to negative social consequences due to gaming, such as a decrease in other leisure activities, relationship satisfaction and school performance. However, the present study could not test these causal mechanisms, therefore such assumptions should be interpreted with caution.

VI/5/1. Limitations

Various limitations of the present study should be considered when drawing conclusions. First, the cross-sectional design of the study impeded the determination of causal relationships between GD and alcohol and illicit drug use. Second, several measurement limitations hindered revealing the complete associations between GD and alcohol and illicit drug use. It would have been desirable to cover more symptoms of GD and it is also important to consider that the applied measurement of using some of the items of the IGDT-10 to measure the criteria of GD as proposed in the ICD-11 was not validated. Although the five-item version of the IGDT-10 covered the core criteria of GD defined by the ICD-11, some criteria might have been assessed incompletely (e.g. the item regarding the criterion of loss of control only reflected on the desire to reduce gaming and did not cover other aspects of impaired control over gaming, such as intensity and duration of gaming) and other diagnostic features of GD not evaluated (e.g. whether the gaming pattern was continuous or episodic and recurrent). Therefore, future studies should explore how validly the five IGDT-10 items cover the GD criteria defined by the ICD-11. Moreover, the present study did not cover important aspects of substance use, such as other potentially relevant psychoactive substances (e.g. caffeine), simultaneous use of gaming and psychoactive substances (Ream et al., 2011; Škařupová et al., 2018), age of initiation of alcohol and illicit drug use and problems related to the use of alcohol and illicit drugs. For example, regarding the latter, it can be assumed that GD symptom severity might have shown a stronger relationship with problems related to alcohol and illicit drug use (e.g. impaired control over consumption) than its association with the frequency of consumption of different psychoactive substances (Ream et al., 2011). In addition to these limitations, the present study did not assess lifetime psychiatric comorbidities (e.g. attention deficit hyperactivity disorder, ADHD) or measure relevant

potential third variables (e.g. impulsivity), which can simultaneously influence GD and alcohol and illicit drug use. For example, previous literature has shown that adolescent ADHD can be related to GD as well as to alcohol and illicit drug use (Groenman et al., 2017; Marmet et al., 2018), thus the lack of data in the present study regarding ADHD symptoms and comorbidity status might bias the findings on the associations between GD and alcohol and illicit drug use (e.g. the association between GD and substance use might be explained by ADHD symptom severity). Finally, low prevalence rates for GD criteria and several illicit drugs, as well as item transformation of the variables regarding alcohol and illicit drug use, might have affected the characteristics of the retained latent classes and their relationships with GD.

VI/6. Conclusions and implications

The present study examined the typologies of alcohol and illicit drug consumption among adolescents and their associations with GD severity, in addition to exploring the differential effects of GD criteria on alcohol and illicit drug use patterns. While GD symptom severity was not associated with increasing levels of alcohol use, polysubstance user adolescents were characterized with higher severity of GD. The analyses also suggested that the GD criteria of 'giving up other activities' and 'negative consequences' due to gaming were related to the latter substance use profile. The present findings suggest the need to assess and screen alcohol and illicit drug use patterns among adolescents who show a risk for GD, and the qualitative exploration of GD symptoms might also provide relevant indications of co-occurring risk for GD and alcohol and illicit drug use. Comprehensive therapeutic and intervention approaches for potentially addictive behaviours and substance use can be beneficial in this population by focusing on shared underlying risk factors, such as the motivational background of addictive behaviours, the facilitation of self-control, emotion regulation and social support (Kim & Hodgins, 2018).

VI/7. Supplementary materials

VI/Supplementary Table 1. Prevalence rates and gender- and school grade-based comparisons of various psychoactive substances

		Total sample *N* (%)	**Females *N* (%)**	**Males *N* (%)**	**χ^2 (*p*)**	**Effect size (φ)**	**9th grade students *N* (%)**	**11th grade students *N* (%)**	**χ^2 (*p*)**	**Effect size (φ)**
Alcohol use	Lifetime abstinence	376 (14.27%)	200 (14.47%)	174 (13.95%)	11.12 (0.004)	0.07	289 (18.84%)	87 (7.91%)	118.10 (< 0.001)	0.21
	Lifetime alcohol use but not in the past month	656 (24.91%)	379 (27.42%)	276 (22.13%)			441 (28.75%)	215 (19.55%)		
	Past month alcohol use	1602 (60.82%)	803 (58.10%)	797 (63.91%)			804 (52.41%)	798 (72.55%)		
Drunkenness	Lifetime absence of drunkenness	1067 (40.74%)	600 (43.76%)	464 (37.33%)	17.11 (< 0.001)	0.08	786 (51.27%)	281 (25.87%)	170.14 (< 0.001)	0.26
	Lifetime drunkenness but not in the past month	743 (28.37%)	393 (28.67%)	349 (28.08%)			363 (23.68%)	380 (34.99%)		
	Past month drunkenness	809 (30.89%)	378 (27.57%)	430 (34.59%)			384 (25.05%)	425 (39.13%)		
Cannabis use	Lifetime abstinence	2136 (80.30%)	1168 (84.15%)	964 (76.09%)	27.50 (< 0.001)	0.10	1327 (84.90%)	809 (73.75%)	52.68 (< 0.001)	0.14
	Lifetime cannabis use but not in the past month	284 (10.68%)	122 (8.79%)	161 (12.71%)			120 (7.68%)	164 (14.95%)		
	Past month cannabis use	240 (9.02%)	98 (7.06%)	142 (11.21%)			116 (7.42%)	124 (11.30%)		

		Total sample *N* (%)	Females *N* (%)	Males *N* (%)	χ^2 (*p*)	Effect size (φ)	9th grade students *N* (%)	11th grade students *N* (%)	χ^2 (*p*)	Effect size (φ)
Ecstasy, MDMA	Lifetime abstinence	2626 (95.46%)	1388 (96.79%)	1233 (93.98%)	12.48 (< 0.001)	0.07	1566 (96.97%)	1060 (93.31%)	20.55 (< 0.001)	0.09
	Lifetime use	125 (4.54%)	46 (3.21%)	79 (6.02%)			49 (3.03%)	76 (6.69%)		
Amphetamines	Lifetime abstinence	2607 (94.94%)	1378 (96.30%)	1224 (93.44%)	11.63 (0.001)	0.07	1551 (96.22%)	1056 (93.12%)	13.26 (< 0.001)	0.07
	Lifetime use	139 (5.06%)	53 (3.70%)	86 (6.56%)			61 (3.78%)	78 (6.88%)		
Nonmedical use of medications	Lifetime abstinence	2585 (94.10%)	1345 (93.92%)	1235 (94.27%)	0.15 (0.698)	0.01	1526 (94.61%)	1059 (93.39%)	1.79 (0.181)	0.03
	Lifetime use	162 (5.90%)	87 (6.08%)	75 (5.73%)			87 (5.39%)	75 (6.61%)		
Concurrent use of alcohol and medications	Lifetime abstinence	2538 (92.39%)	1320 (92.11%)	1213 (92.67%)	0.30 (0.587)	0.01	1512 (93.74%)	1026 (90.48%)	10.08 (0.001)	0.06
	Lifetime use	209 (7.61%)	113 (7.89%)	96 (7.33%)			101 (6.26%)	108 (9.52%)		
Designer drugs	Lifetime abstinence	2561 (93.03%)	1338 (93.37%)	1218 (92.62%)	0.59 (0.443)	0.02	1517 (93.93%)	1044 (91.74%)	4.94 (0.026)	0.04
	Lifetime use	192 (6.97%)	95 (6.63%)	97 (7.38%)			98 (6.07%)	94 (8.26%)		

Note. χ^2 = Chi-square statistics. φ = Phi effect size estimate (in absolute value). Percentages shown in the gender- and school grade-related columns represent proportions within each gender- and school grade-related category.

VI/Supplementary Table 2. Prevalence rates and gender- and school grade-based comparisons of gaming related measures

		Total sample	**Females**	**Males**	**Test statistics (*p*)**	**Effect size**	**9th grade students**	**11th grade students**	**Test statistics (*p*)**	**Effect size**
Gaming status *N* (%)	Never-gamers	744 (27.07%)	582 (40.87%)	159 (12.05%)	χ^2 = 288.38 (< 0.001)	φ=0.32	431 (26.74%)	313 (27.55%)	χ^2=0.23 (0.636)	φ=0.01
	Gamers	2004 (72.93%)	842 (59.13%)	1160 (87.95%)			1181 (73.26%)	823 (72.45%)		
		Total sample of gamers	**Females**	**Males**	**Test statistics (*p*)**	**Effect size**	**9th grade students**	**11th grade students**	**Test statistics (*p*)**	**Effect size**
Gaming frequency: schooldays M (SD)		3.55 (2.01)	3.27 (1.94)	3.75 (2.04)	*t* = 5.32 (< 0.001)	*d* = 0.24	3.57 (2.02)	3.52 (2.00)	*t* = 0.50 (0.616)	*d* = 0.02
Gaming frequency: weekend M (SD)		4.75 (2.32)	4.17 (2.19)	5.17 (2.31)	*t* = 9.83 (< 0.001)	*d* = 0.44	4.78 (2.35)	4.71 (2.27)	*t* = 0.72 (0.473)	*d* = 0.03
Gaming disorder symptom severity M (SD)		1.93 (2.02)	1.30 (1.72)	2.39 (2.10)	*t* = 12.68 (< 0.001)	*d* = 0.56	1.93 (1.99)	1.95 (2.07)	*t* = 0.22 (0.823)	*d* = 0.01
Criterion: Loss of control *N* (%)	Never or sometimes	1875 (94.27%)	802 (96.39%)	1071 (92.73%)	χ^2 = 12.03 (0.001)	φ = 0.08	1107 (94.45%)	768 (94.00%)	χ^2 = 0.18 (0.670)	φ = 0.01
	Often	114 (5.73%)	30 (3.61%)	84 (7.27%)			65 (5.55%)	49 (6.00%)		
Criterion: Giving up other activities *N* (%)	Never or sometimes	1858 (93.37%)	799 (96.03%)	1058 (91.52%)	χ^2 = 16.00 (< 0.001)	φ = 0.09	1092 (93.09%)	766 (93.76%)	χ^2 = 0.34 (0.559)	φ = 0.01
	Often	132 (6.63%)	33 (3.97%)	98 (8.48%)			81 (6.91%)	51 (6.24%)		
Criterion: Continuation *N* (%)	Never or sometimes	1741 (87.58%)	774 (93.14%)	966 (83.64%)	χ^2 = 40.23 (< 0.001)	φ = 0.14	1020 (86.96%)	721 (88.47%)	χ^2 = 1.01 (0.316)	φ = 0.02
	Often	247 (12.42%)	57 (6.86%)	189 (16.36%)			153 (13.04%)	94 (11.53%)		

		Total sample	Females	Males	Test statistics (*p*)	Effect size	9th grade students	11th grade students	Test statistics (*p*)	Effect size
Criterion: Negative consequences *N* (%)	Never or sometimes	1821 (91.60%)	795 (95.55%)	1025 (88.82%)	χ^2 = 28.60 (< 0.001)	φ = 0.12	1071 (91.46%)	750 (91.80%)	χ^2 = 0.07 (0.789)	φ = 0.01
	Often	167 (8.40%)	37 (4.45%)	129 (11.18%)			100 (8.54%)	67 (8.20%)		
Number of endorsed gaming disorder criteria *N* (%)	0 criteria	1552 (77.83%)	717 (86.07%)	834 (72.02%)	χ^2 = 59.99 (< 0.001)	φ = 0.17	899 (76.45%)	653 (79.93%)	χ^2 = 10.42 (0.034)	φ = 0.07
	1 criterion	285 (14.30%)	84 (10.08%)	201 (17.36%)			188 (15.99%)	97 (11.87%)		
	2 criteria	107 (5.37%)	24 (2.88%)	83 (7.17%)			61 (5.19%)	46 (5.63%)		
	3 criteria	35 (1.76%)	7 (0.84%)	27 (2.33%)			23 (1.96%)	12 (1.47%)		
	4 criteria	14 (0.70%)	1 (0.12%)	13 (1.12%)			5 (0.43%)	9 (1.10%)		

Note. χ^2 = Chi-square statistics. φ = Phi effect size estimate (in absolute value). *t* = Independent-samples t-test statistics (in absolute value). *d* = Cohen's d effect size estimate (in absolute value). Percentages shown in the gender- and school grade-related columns represent proportions within each gender- and school grade-related category. Comparisons related to gaming frequencies and gaming disorder-related variables are performed among gamers.

VI/Supplementary Table 3. Model fit indices of the models with different number of latent classes

	AIC	BIC	SSA-BIC	Entropy	LMRT	*p*
1-class model	19989.28	20054.40	20019.45	-	-	-
2-class model	17410.22	17546.37	17473.29	0.84	2575.96	< 0.001
3-class model	16357.68	16564.86	16453.65	0.81	1065.33	< 0.001
4-class model	16182.89	16461.10	16311.76	0.77	196.73	< 0.001
5-class model	16031.91	16381.15	16193.69	0.79	173.16	0.100

Note. AIC = Akaike Information Criteria. BIC = Bayesian Information Criteria. SSA-BIC = Sample size adjusted – Bayesian Information Criteria. LMRT = Lo Mendel Rubin adjusted likelihood ratio test.

VII. Study 4: Alcohol consumption and risk for feeding and eating disorders in adolescence: The mediating role of drinking motives[12,13]

VII/1. Abstract

Background: A complex and bidirectional association has been assumed between feeding and eating disorders (FEDs) and alcohol consumption. Previous research has demonstrated that alcohol use among individuals with different forms of FEDs is more frequently motivated by two subtypes of internal drinking motives: coping and enhancement motives. Namely, these individuals might use alcohol primarily to regulate internal states, such as to mitigate negative emotions or enhance positive emotions.

Objectives: The present study investigated the mediating role of internal drinking motives on the association between risk for FEDs and alcohol consumption over the effects of relevant covariates, such as depressive symptoms or body mass index (BMI).

Methods: Hungarian data of the European School Survey Project on Alcohol and Other Drugs (ESPAD) from 2015 were used. The final sample included responses from 5457 adolescents (50% males; mean age: 16.62 years). Validated self-report psychometric instruments assessed the level of alcohol use, depressive symptoms and risk for FEDs, and drinking motives.

Results: Risk for FEDs presented a significant positive relationship with internal drinking motives and alcohol use. In the mediation analysis, a significant indirect effect was identified between risk for FEDs and alcohol use via internal drinking motives among females.

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Conclusions: Results demonstrated that risk for FEDs was positively associated with internal drinking motives and alcohol use. An indirect effect of risk for FEDs on alcohol consumption via internal drinking motives was discriminated over the impact of depressive symptoms. However, the latter relationship was only found among females which may highlight the gender differences in the relationship between risk for FEDs and alcohol use.

Keywords: feeding and eating disorders; drinking motives; gender differences; adolescents; alcohol consumption; alcohol comorbidity

VII/1. Introduction

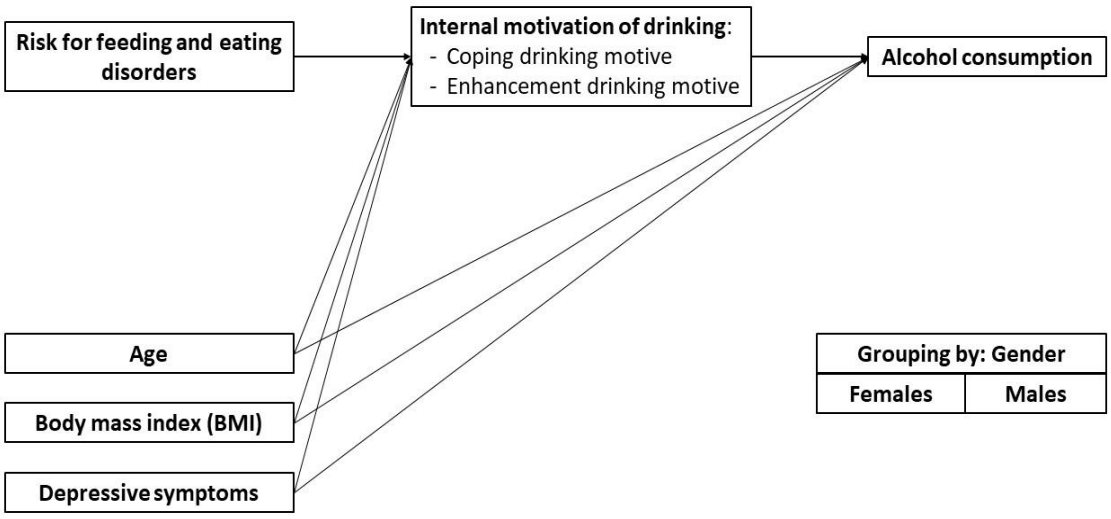
According to the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the category of feeding and eating disorders (FEDs) is a heterogenous group of disorders defined by psychological and physiological difficulties related to eating behavior or appetite. The diagnostic category of FEDs includes various distinct disorders, such as anorexia nervosa, avoidant/restrictive food intake disorder, binge eating disorder, bulimia nervosa, pica, and rumination disorder (American Psychiatric Association, 2013). Pre-adolescence and adolescence are considered as crucial developmental phases due to the progression of various risk behaviors, such as development of clinical and sub-clinical forms of FEDs (Pearson et al., 2014). Approximately 3.7% of adolescents are affected with any form of clinical level FED (Flament et al., 2015). However, an even higher proportion of adolescents show sub-threshold or sub-clinical types of FEDs (e.g., 6.1% for bulimia nervosa, 4.6% for binge eating disorder) (Stice et al., 2009). Females generally show higher prevalence rates for various forms of FEDs during adolescence (Croll et al., 2002; Kjelsås et al., 2004), while it has also been reported that incidence rates of any forms of FEDs might increase until around the age of 16-17 years during adolescence (Javaras et al., 2015). Among adolescents, various symptom of FEDs (e.g., drive for thinness, bulimic symptoms) are associated with higher rates of body mass index (BMI) (Fan et al., 2010). Sub-threshold forms of different FEDs during adolescence constitute a risk for experiencing increased symptom severity or clinical forms of different FEDs later in adulthood (Neumark-Sztainer et al., 2011). Furthermore, subclinical forms of FEDs are also associated with higher rates of co-occurring psychopathological symptoms, such as symptoms of

depression or anxiety (Herpertz-Dahlmann et al., 2015; Touchette et al., 2011). In the present study, the term of "risk for feeding and eating disorders (FEDs)" is applied to reflect general and non-specific FED-related symptom severity which might indicate a higher risk for having some forms of FED in absence of formal clinical diagnosis (Richter et al., 2017).

Co-occurrence of different forms of FEDs and substance misuse has been consistently reported in previous studies. Symptoms of different types FEDs are associated with alcohol use among treatment-seeking and non-treatment-seeking adolescents (Arias et al., 2009; Baker et al., 2018). More specifically, a higher severity of anorexia nervosa or bulimia nervosa symptoms among adolescents has been associated with more severe patterns of alcohol consumption (e.g. more frequent intoxication), more adverse social and psychological consequences (e.g. higher level of alcohol-related physical symptoms) (Arias et al., 2009; Castro-Fornieles et al., 2010). Within the cluster of FEDs, bulimic characteristics, such as binge eating or purging, have shown a more robust association with alcohol use compared to restrictive FED features such as anorexia nervosa (Baker et al., 2017; Gregorowski et al., 2013). Longitudinal findings have suggested that symptoms of alcohol misuse develop subsequently with the onset of FEDs rather than the other way around (Baker et al., 2010; Franko et al., 2005).

One explanation concerning the co-occurrence of different forms of FEDs and elevated levels of alcohol use is that they share underlying emotional risk mechanisms, such as elevated reward sensitivity and negative affect dysregulation (Schulte et al., 2016; Stewart et al., 2006). In the cases of reward-seeking behaviors, individuals seek reinforcing activities, such as using alcohol or binge eating in order to enhance positive emotions, and they perceive these behaviors as highly pleasant (Birch et al., 2007; Dawe & Loxton, 2004; Schulte et al., 2016). Related to the 'self-medication' concept, it has been assumed that different forms of FEDs (e.g. binge eating disorder) and alcohol consumption occur as a coping response with the aim of mitigating negative affect (Birch et al., 2007; Cook et al., 2014; Stewart et al., 2006). Alternatively, the impulsivity facet of negative urgency might also play an important role, which is the tendency to act rashly when experiencing negative affective states. Individuals with high level of negative urgency are likely to engage in the aforementioned risk behaviors impulsively when experiencing negative emotions (Fischer et al., 2004).

The aforementioned positive and negative reinforcement mechanisms are also represented in interrelated motives for FEDs (e.g. binge eating disorder) and alcohol use (Luce et al., 2007). Due to the aforementioned similar risk characteristics of emotion regulation, individuals who show risk for FEDs might be more likely consume alcohol because of positive and negative reinforcement mechanisms in terms of affective states. Within the framework of the motivational model of alcohol use (Cooper, 1994), it is expected that alcohol consumption is more likely to be motivated by internal motives for those who show a risk for FEDs. Namely, these individuals might use alcohol more frequently in order to regulate their internal or affective states, such as to mitigate negative affect (coping motives) or enhance positive emotions (enhancement motives). To date, only a few studies have investigated the relationship between different forms of FEDs and drinking motives (Anderson et al., 2006; Luce et al., 2007; Mikheeva & Tragesser, 2016). Based on these findings, individuals with different forms of FEDs, such as bulimia nervosa or binge eating disorder, have shown higher levels of coping motives for drinking.



VII/Figure 1. Hypothesized conceptual model related to the association between risk for FEDs and alcohol consumption

The present study aimed to obtain a more comprehensive understanding of the interrelations between risk for FEDs, drinking motives, and alcohol use. A conceptual model was hypothesized and tested (VII/Figure 1) where the association between risk for FEDs and alcohol consumption would be mediated by internal drinking motives. It was assumed that alcohol use among adolescents who show higher risk for FEDs would be driven by coping (e.g., drinking to forget about problems) and enhancement motives (e.g., drinking because it is exciting) to a greater extent, which subsequently contribute to more severe forms of alcohol consumption (Anderson et al., 2006; Birch et al., 2007; Luce et al., 2007). Therefore, based on the aforementioned theoretical considerations (i.e., shared, positive and negative affective reinforcement mechanisms which might explain the co-occurrence of different forms of FEDs and elevated levels of alcohol use, and their implications for the motivational background of alcohol use), the present study primarily assessed the mediating role of internal motives (i.e., using alcohol to regulate internal or affective states) between risk for FEDs and alcohol use. Due to this, the indirect effects of risk for FEDs on alcohol consumption via external motives (i.e., social and conformity motives) were only investigated as supplementary analyses. It was expected that the effect of risk for FEDs on alcohol consumption would be separately demonstrable among males and females over the co-occurring effect of depressive symptoms (Herpertz-Dahlmann et al., 2015; Touchette et al., 2011) as well as after considering the possible covariance between age, BMI, and risk for FEDs (Fan et al., 2010; Javaras et al., 2015). To the best of the authors' knowledge, no previous study has examined the potential mediating role of drinking motives between risk for FEDs and alcohol consumption.

VII/3. Methods

VII/3/1. Participants and procedure

The present study's data derived from the Hungarian data of the European School Survey Project on Alcohol and Other Drugs (ESPAD) from 2015. The aim of the ESPAD study is to collect data on adolescents' tobacco, alcohol, and other substance use to facilitate temporal and cross-national comparisons (Elekes, 2016). The target population of this study consisted of ninth- and tenth-grade students in general and vocational secondary schools (i.e., 16 year old students born in 1999). Stratified cluster sampling was applied to assure representativeness of the sample in terms of geographic region, grade, and school type. In total, 7% of the selected schools declined to participate in the study. The

study of the ESPAD 2015 Hungary comprised data from 6664 students. Data from 443 students were excluded from the analyses due to an invalid questionnaire or inconsistent response patterns. Only data were considered for the final analyses from those adolescents who reported alcohol consumption in their lifetime. Consequently, data from further 764 participants were excluded from the analyses because of lifetime abstinence of alcohol consumption. Therefore, the final sample included responses from 5457 participants (proportion of males: 50.0% [*N* = 2731]; mean age = 16.62 [SD = 0.94]; mean BMI = 21.66 [SD = 3.78]).

VII/3/2. Measures

VII/3/2/1. Alcohol consumption. Six alcohol consumption-related items were selected for the analyses to reflect frequency of alcohol use (ESPAD Group, 2016). Frequency of alcohol use and drunkenness were assessed during the past 12 months and 30 days using a seven-point frequency scale (1 = *0 times*, 7 = *40 or more times*). The level of binge drinking was also taken into account: individuals had to assess how frequently they consumed at least five drinks in one occasion during the past 30 days on a six-point scale (1 = *0 times*, 6 = *10 or more times*). Finally, participants estimated on a ten-point scale the level of self-reported drunkenness on the last occasion when they consumed alcohol (1 = *Alcohol did not have an effect*; 10 = *I was very drunk, I did not remember what happened to me*). The level of alcohol consumption was represented by a composite continuous latent variable which was defined by the aforementioned observed alcohol use indicators ($\omega = 0.89$). Illustration of the construction of the continuous one-factor latent variable assessing alcohol use and factor loadings related to the observed indicators are presented in VII/Supplementary Figure 1. Previous studies have also applied similar approach and assessed the level of alcohol consumption by a continuous unidimensional latent factor based on various observed indicators of alcohol use (Källmén et al., 2019; LaBrie et al., 2011; Sher et al., 1996).

VII/3/2/2. Center of Epidemiological Studies Depression-Scale (CES-D). Depressive symptomatology was assessed using the short form of the CES-D-Scale (Demetrovics, 2007; Kokkevi & Fotiou, 2009). It comprises six items reflecting various symptoms of depression during the past seven days, such as concentration issues and mood disturbances. Adolescents had to provide responses for each question on a four-point scale (1 = *Nearly never*; 4 = *Nearly always*). The scale presented a good level of internal

consistency in the present sample (α = 0.84). Depressive symptoms were specified as one-factor latent variable in the present analyses (ω = 0.89).

VII/3/2/3. Drinking Motives Questionnaire – Short Form (DMQ-SF). Participants' reasons for using alcohol was assessed using the 12-item shortened version of the DMQ (Kuntsche & Kuntsche, 2009; Németh, Kuntsche, et al., 2011). The instrument originally assessed four types of drinking motives: (i) coping, (ii) conformity, (iii) enhancement, and (iv) social motives. Students answered each item on a five-point scale (1 = *Never*; 5 = *Always*). Based on predominantly theoretical considerations, only internal (i.e., coping and enhancement) motives were involved in the current analyses related to the hypothesized conceptual model. A good level of internal consistency was demonstrated related to the two selected subscales of the DMQ in the present sample (enhancement: α = 0.82; coping: α = 0.89).

However, due to the extremely high level of correlation between coping and enhancement motives (r = 0.86), it was not possible to include both factors of internal drinking motives separately in the analysis. Therefore, in the mediation analysis, a latent factor of 'internal drinking motives' was specified which incorporated items of the coping and enhancement subscales (ω = 0.94). Consequently, this factor represented motives of general affect regulation for alcohol consumption irrespective of their valence. In line with this, previous studies have also underlined that enhancement and coping drinking motives are not distinct but rather more combined constructs, and it is hard to separate them at within-person level (Goldstein & Flett, 2009). Similarly, other studies have suggested the existence of a broad and non-specific construct of drinking motives (Lac & Donaldson, 2017; Urbán et al., 2008).

VII/3/2/4. SCOFF Questionnaire. In order to assess the risk for FEDs among respondents, the SCOFF questionnaire was used (Dukay-Szabó et al., 2016; Morgan et al., 1999). The scale was originally designed to screen for FEDs. It contains five items which reflect on the core symptoms of anorexia nervosa and bulimia nervosa. The name of the questionnaire is an acronym reflecting on the content of the symptoms included in the scale (e.g., letter 'C'– for 'control'– denotes worrying about losing control over eating). Participants had to decide in the case of each item if it was true for themselves or not (0 = *No*; 1 = *Yes*). Traditional measure of reliability presented inadequate degree of internal consistency in the present sample (α = 0.55). Previous studies also reported inadequate α

levels for the SCOFF because it contains small number of items and its items represent symptoms of disparate disorders (Burton et al., 2016; Garcia et al., 2010). In order to overcome this problem, risk for FEDs was defined as a one-factor latent variable in the present analysis. Satisfactory level of model fit was presented for the one-factor measurement model ($\chi^2(5) = 74.71$; $p < 0.001$; CFI = 0.970; TLI = 0.941; RMSEA [90% CI] = 0.051 [0.041 – 0.061]) in addition to the acceptable degree of model-based internal consistency ($\omega = 0.78$). Previous studies using confirmatory factor analysis and item response theory analysis have also supported the unidimensional latent structure of the questionnaire (Bean, 2019; Richter et al., 2017).

VII/3/3. Data analysis

Structural equation modeling (SEM) was performed to examine the indirect effect of risk for FEDs on alcohol use via internal drinking motives. The analysis was performed separately for males and females in order to control the possible gender-related differences in terms of FEDs (Croll et al., 2002). The effects of age, BMI, and depressive symptoms were also taken into account during the analyses. Because comorbidity might be present between different forms of FEDs and depressive symptoms among adolescents (Santos et al., 2007), it was necessary to distinguish the effects of risk for FEDs on drinking motives and alcohol use outcomes from those of depressive symptoms. In the mediation analysis, risk for FEDs and depressive symptoms, internal drinking motives, and level of alcohol consumption were specified as a continuous one-factor latent variables. Total, direct, and indirect effects of risk for FEDs on alcohol use via internal drinking motives were assessed.

Supplementary analyses were also conducted to separately test the mediating effect of each drinking motive between risk for FEDs and alcohol consumption. Although due to theoretical considerations the present study did not aim to examine the mediating role of external drinking (i.e., conformity and social) motives between risk for FEDs and alcohol use, interested readers can investigate these findings in VII/Supplementary Figure 2A-D. Moreover, additional supplementary analyses also demonstrated that if the effect of highly correlating drinking motives (e.g., relationship among social, enhancement, and coping motives: $r = 0.70$-0.88) were simultaneously included in the mediation model, conformity and enhancement motives presented negative relationships with alcohol consumption which are considered as a statistical artefact (i.e., negative suppressor

effects). This was indicated because of the significant and positive associations which were demonstrated between drinking motives and alcohol consumption in mediation models separately containing each of the drinking motives. These latter results are in line with previous literature findings and theoretical considerations indicating that higher levels of drinking motives can predict higher rates of alcohol use (Crutzen et al., 2013). Previous studies have also reported similar negative suppressor effects due to the high level of correlation between factors of drinking motives, especially in the case of conformity motives (Németh, Urbán, et al., 2011).

Except for the variable assessing the level of drunkenness on the last occasion, all indicator variables of the continuous latent variables were specified as categorical observed variables. The model estimation was based on the Weighted Least Squares Mean and Variance (WLSMV) technique. Degree of model fit was determined based on various model fit indices. Optimal level of model fit was indicated by values of at least 0.90 – 0.95 in the case of the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI). A value below 0.05 of the of Root Mean Squared Error of Approximation (RMSEA) index marks an adequate model fit. All analyses used weighted data to ensure representativeness of the sample. Moreover, cluster effect due to class-based sampling and possible non-independence of the observations within each cluster was also modeled. Analyses were performed using MPlus 8.0 (Muthén & Muthén, 2017) and IBM SPSS Statistics 23.0 software. For preliminary analyses conducted by using the latter software, missing data were handled by listwise deletion, while for SEM analyses conducted by MPlus 8.0, pairwise deletion was applied to handle missing data.

VII/4. Results

VII/4/1. Preliminary analyses

VII/Table 1 presents the prevalence of alcohol use-related indicators and risk for FEDs in the total sample, and among males and females. Except for alcohol consumption status in the past 12 months, males presented significantly higher odds for engaging in each alcohol consumption-related outcomes compared to females. Females demonstrated approximately three times higher odds for having a risk for FEDs compared with males.

Additional analyses revealed that those individuals who reported of having symptoms of making themselves sick because feeling uncomfortably full, recently losing more than one stone in weight, and believing themselves to be fat when others say they are too thin

consistently, showed significantly higher odds of engaging in various alcohol consumption-related outcomes, such as alcohol consumption, drunkenness and binge drinking in the past 30 days, and drunkenness in the past 12 months (VII/Supplementary Table 1).

VII/Table 1. Prevalence of alcohol use-related indicators and risk for FEDs in the total sample, and among males and females

	Total sample *N* (%)	Males *N* (%)	Females *N* (%)	χ^2 (*p*)	OR [95% CI]
Alcohol consumption in the past 12 months	5081 (93.75%)	2539 (93.76%)	2542 (93.73%)	< 0.001 (0.966)	1.00 [0.81 – 1.25]
Alcohol consumption in the past 30 days	3531 (65.08%)	1826 (67.36%)	1703 (62.75%)	12.66 (< 0.001)	**1.22 [1.10 – 1.37]**
Drunkenness in the past 12 months	2816 (52.46%)	1480 (55.35%)	1335 (49.55%)	18.06 (< 0.001)	**1.26 [1.13 – 1.40]**
Drunkenness in the past 30 days	1185 (22.05%)	635 (23.71%)	550 (20.40%)	8.57 (0.003)	**1.21 [1.07 – 1.38]**
Binge drinking in the past 30 days	2613 (47.89%)	1426 (52.23%)	1187 (43.54%)	41.28 (< 0.001)	**1.42 [1.27 – 1.58]**
Risk for FEDs[1]	1384 (26.39%)	411 (15.79%)	973 (36.83%)	298.78 (< 0.001)	**0.32 [0.28 – 0.37]**

Note. χ^2: Chi square statistics representing comparisons between males and females. OR: Odds Ratio. 95% CI: 95% Confidence Interval for Odds Ratios. Odds ratios in bold are significant at least $p < 0.05$ level. In each comparison, females were specified as the reference category. [1] Risk for FEDs was determined by using the threshold of the SCOFF questionnaire: at least two positive responses on the instrument was assessed as a case for having a risk for FEDs. Number of missing cases in the total sample in the order of the variables presented in the first column: $N = 37$; $N = 31$; $N = 89$; $N = 83$; $N = 1$; $N = 212$. Number of cases excluded from gender-based comparisons by using listwise deletion because of the missing data in each variable in the order of variables presented in the first column: $N = 37$; $N = 32$; $N = 91$; $N = 83$; $N = 1$; $N = 212$.

VII/4/2. Testing the indirect effect of risk for FEDs on alcohol consumption via drinking motives

SEM was conducted separately for males and females to test the indirect effect of risk for FEDs on alcohol consumption via internal drinking motives. Bivariate correlations between the variables are displayed in VII/Table 2. For interested readers, VII/Supplementary Figure 2 contains results of these analyses which were performed to test the mediating effect of each drinking motives separately between risk for FEDs and alcohol consumption.

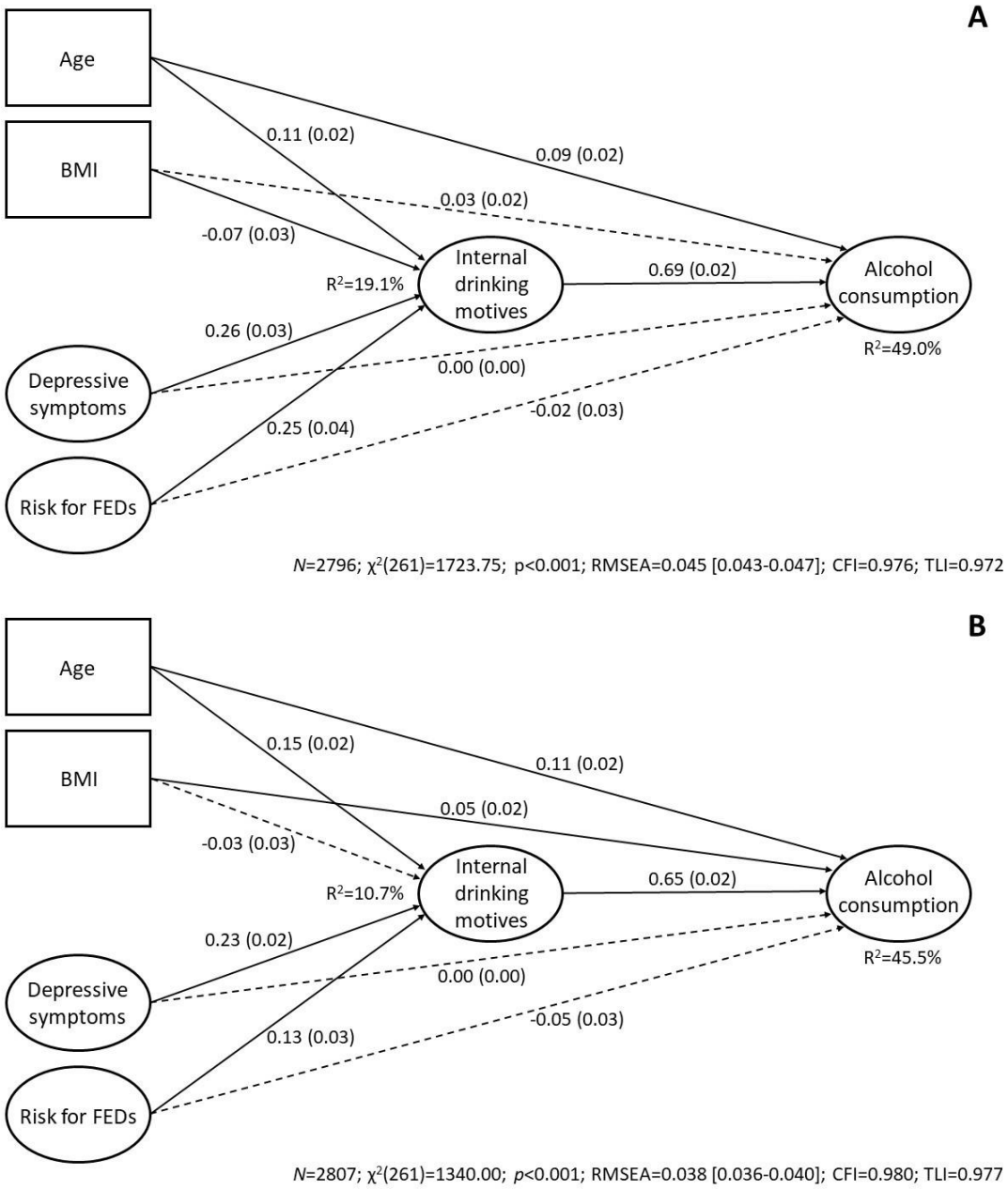
VII/Table 2. Correlation coefficients between latent and observed study variables among males and females

	1.	2.	3.	4.	5.	6.
1. Age	-	**0.09**	0.05	**0.09**	**0.17**	**0.22**
2. BMI	0.01	-	0.04	**0.37**	0.03	**0.07**
3. Depressive symptoms	-0.04	0.03	-	**0.21**	**0.27**	**0.17**
4. Risk for FEDs	-0.01	**0.31**	**0.45**	-	**0.17**	**0.09**
5. Internal drinking motives	**0.09**	0.02	**0.37**	**0.35**	-	**0.66**
6. Alcohol consumption	**0.16**	0.03	**0.24**	**0.23**	**0.69**	-

Note. Significant ($p < 0.05$) correlation coefficients are in bold. Correlation coefficients above the diagonal are assessed among males ($N = 2807$), while correlation coefficients below diagonal are assessed among females ($N = 2796$). Missing data statistics are equivalent to the data presented in VII/Figure 2.

Regression path coefficients between the predictor and outcome variables are shown in VII/Figure 2A for females and in VII/Figure 2B for males. Optimal level of model fit was presented for males and females. For both gender groups, in the final model the regression path coefficient between depressive symptoms and alcohol use was fixed at 0 in order to avoid negative suppressor effects (unconstrained β for males = -0.06, $p = 0.006$; unconstrained β for females = -0.11, $p = 0.002$). Risk for FEDs and depressive symptoms presented a significant and positive predictive effect on internal drinking motives among males and females. The direct effect between risk for FEDs and alcohol use was non-significant in both groups. The latent variable of internal drinking motives demonstrated a significant, positive and strong relationship with alcohol consumption irrespective of

gender. Distal predictors partly explained the higher amount of the variance related to the latent factor of internal drinking motives among females. It was assumed that this difference was due to the slightly stronger relationship between ED symptoms and internal drinking motives among females (*B* [95% CI] for males = 0.13 [0.05 – 0.21], *B* [95% CI] for females = 0.28 [0.18 – 0.38]). The gender-based models explained 45.5 – 49.0% of the variance of alcohol consumption.



VII/Figure 2. Standardized regression coefficients representing the association between risk for FEDs, internal drinking motives and alcohol consumption among females (A) and

males (B). Note. VII/Figure 2A presents findings among females, while VII/Figure 2B presents findings among males. Solid lines represent significant ($p < 0.05$) standardized (β) regression coefficients. Dashed lines represent non-significant ($p > 0.05$) standardized (β) regression coefficients. Related to each regression coefficients, standard error (S.E.) values are presented in brackets. Regression coefficient between depressive symptoms and alcohol consumption was fixed at 0. Number of missing data patterns among females: $N = 97$. Covariance coverage among females: mean covariance coverage = 96.2% range of covariance coverage = 92.7% – 100.0%. Number of missing data patterns among males: $N = 107$. Covariance coverage among males: mean covariance coverage = 94.9%. range of covariance coverage = 90.8% – 99.9%.

Effect size indices relating to the total, direct, and indirect effects from risk for FEDs upon alcohol consumption are shown in VII/Table 3. The total effect of risk for FEDs on alcohol use was significant among females, but it was non-significant for males. Therefore, for males, the indirect effect from risk for FEDs to alcohol consumption was not estimated. Among both genders, the direct effect of risk for FEDs did not remain significant after taking into account the effect of drinking motives. The indirect effect was significant via internal drinking motives among females. Therefore, higher risk for FEDs predicted higher level of internal drinking motives which subsequently contributed to elevated rates of alcohol consumption. This indirect effect was demonstrated over the influence of depressive symptoms.

VII/Table 3. Standardized and unstandardized effect size indices related to the total, direct and indirect effects from risk for FEDs to alcohol consumption among males and females

	Males		Females	
	B (S.E.)	*β* (S.E.)	*B* (S.E.)	*β* (S.E.)
Total effect	0.05 (0.05)	0.03 (0.04)	**0.22 (0.05)**	**0.16 (0.04)**
Direct effect	-0.07 (0.05)	-0.05 (0.03)	-0.02 (0.05)	-0.02 (0.03)
Indirect effect through internal drinking motives	-[1]	-[1]	**0.24 (0.04)**	**0.17 (0.03)**

Note. Unstandardized (*B*) and standardized (β) effect size measures presented with bold figures are significant at least $p < 0.05$ level. [1]Indirect effect from risk for FEDs to alcohol consumption was not estimated among males due to non-significant total effect.

VII/5. Discussion

The present study investigated the complex relationship between risk for FEDs, internal drinking motives, and alcohol use. The main aim of the study was to investigate the mediating effect of internal drinking motives in the association between risk for FEDs and alcohol use. To the best of the authors' knowledge, this is the first time that the complex relationship between risk for FEDs, drinking motives, and alcohol use has been examined among adolescents. According to the results of the present study, the association between risk for FEDs and alcohol consumption was mediated by internal drinking motives among females. Supporting the hypothesized conceptual model of the study, the indirect effect including internal drinking motives demonstrated that more severe risk for FEDs predicted higher level of drinking motives which enhanced positive or mitigated negative emotions, which subsequently contributed to elevated rates of alcohol consumption. This indirect effect was present while controlling for the effect of depressive symptoms.

This result is in line with previous research findings emphasizing the occurrence of high level of coping drinking motives among participants with different forms of FEDs, such as bulimia nervosa or binge eating disorder (Anderson et al., 2006; Luce et al., 2007; Mikheeva & Tragesser, 2016). The present outcomes also fit the theoretical considerations which have attempted to identify similar features of problematic alcohol use and different forms of FEDs. These studies assumed that problematic forms of eating behavior (e.g. binge eating) and alcohol use might be motivated by alleviating negative emotions (Cook et al., 2014; Stewart et al., 2006). Moreover, the mediational model supported the assumption that adolescents with higher risk for FEDs might engage in more heavy forms of alcohol consumption in order to experience more pleasant internal states (Dawe & Loxton, 2004; Schulte et al., 2016). However, it is important to note that it was not possible to assess the contribution of coping and enhancement motives separately due to their strong correlation. Therefore, reasons which mitigated negative (e.g., using alcohol to cheer up when an individual is in a bad mood) or enhanced positive emotions (e.g., using alcohol because it is exciting) overlapped greatly among adolescents. Internal drinking motives represent general affect regulation reasons for alcohol consumption (Goldstein & Flett, 2009). The strong positive relationship between internal drinking motives and alcohol use is in accordance with previous studies which suggested that higher levels of enhancement and coping motives are associated with more

harmful alcohol use outcomes (Kuntsche et al., 2005) The present research focused primarily on drinking motives which emphasize the emotion regulation aspect of alcohol use, therefore, external drinking motives, such as social and conformity motives, were not included in the mediation model testing the hypothesized conceptual model. However, previous research data presented evidence that, among treatment-seeking individuals with co-occurring alcohol-related problems and binge eating, alcohol use might occur in a social context as well, therefore it is not induced by motives of emotion control only (Birch et al., 2007). In line with this, supplementary analyses showed that when the mediating effects of drinking motives were examined separately, significant indirect effects of risk for FEDs on alcohol consumption via conformity motives among males and females and via social motives among females were found.

It is important to highlight that a different pattern of findings was observed for males and females in the mediation analyses. The total effect of risk for FEDs on alcohol consumption was only significant among females, as well as the indirect effect via internal drinking motives, which was also only significant among females. These findings suggest that the self-medicating role of alcohol use among individuals with a higher risk for FEDs was only demonstrated among females. These different patterns might be explained by gender-specific variations in FEDs. Previous studies have reported that females show a higher symptom severity of some forms of FEDs (e.g. weight or shape concerns, restraint), while males present a higher age of onset for FEDs, and different patterns of symptomatology (e.g., higher tendencies for excessive exercise as a compensatory mechanism, or muscular ideal of the body). In addition, females with different forms of FEDs might show elevated rates of comorbid mood disorders, and experience higher levels of distress related to their symptomatology (Kinasz et al., 2016; Murray et al., 2017). Also, different pattern of findings between males and females might be explained by drinking motive-specific differences among adolescents. During early adolescence, females are more likely to drink due to coping motives, while it is also possible, that adolescent males with a risk for FEDs show a higher tendency for motives with positive valence, such as social motives, which might play a more important role in predicting alcohol use among them (Kuntsche et al., 2006b).

VII/5/1. Limitations and future directions

The present results should be interpreted cautiously due to various limitations. First, because of the cross-sectional nature of the study, it was not possible to explore causal relationships between the variables examined. Because the mediational model was unable to control for bidirectional relationships, future studies should use longitudinal or ecological momentary assessment (EMA) design in order to obtain a more accurate picture of the interrelationship between the variables (Pisetsky et al., 2016). Second, the present findings might not provide a comprehensive representation of the associations between risk for FEDs and alcohol use, because important predictor variables, such as negative urgency (Fischer et al., 2012), were not included in the mediational model. Third, from a psychometric assessment perspective, the SCOFF questionnaire provided a non-differentiated measurement of a risk for FEDs which may have biased the present results. The broad diagnostic category of FEDs includes both heterogeneous and distinct disorders which show disparate symptomatology, such as restricting type of anorexia or binge eating disorder. By using the SCOFF questionnaire, the present study was only able to superficially assess some core symptoms of anorexia nervosa and bulimia nervosa. Furthermore, while multiple possible diagnostic categories might be simultaneously related to each item of the questionnaire and some disorders (e.g., binge eating disorder) within the cluster of FEDs, they are not covered entirely by the SCOFF. Although the study aimed to assess a general and non-differentiated construct of a risk for FEDs, it is important to consider that FEDs are not a single syndrome and in-depth exploration of symptomatology of divergent disorders within the diagnostic category of FEDs. Furthermore, previous findings indicated that individuals in different diagnostic categories within the cluster of FEDs show different motives for substance use (Baker et al., 2010). Therefore, future studies need to examine (e.g., separately for individuals with anorexia nervosa and bulimia nervosa) the mediating role of drinking motives between different types of FEDs and alcohol use by specific diagnoses. Fourth, the present study only limitedly assessed the unique contribution of each drinking motive. Future studies should explore the relationship between risk for FEDs and alcohol use by simultaneously assessing indirect effects related to each subscale of drinking motives. Finally, the present study assessed patterns of alcohol consumption in an overall manner. Therefore it did not address investigating the varying effects of risk for FEDs on different indicators of alcohol use (e.g., alcohol consumption in the past 12 months or binge drinking), or

membership of alcohol consumption-based subgroups (e.g., latent classes characterized with frequent alcohol use with small quantities and high quantities).

VII/6. Conclusions

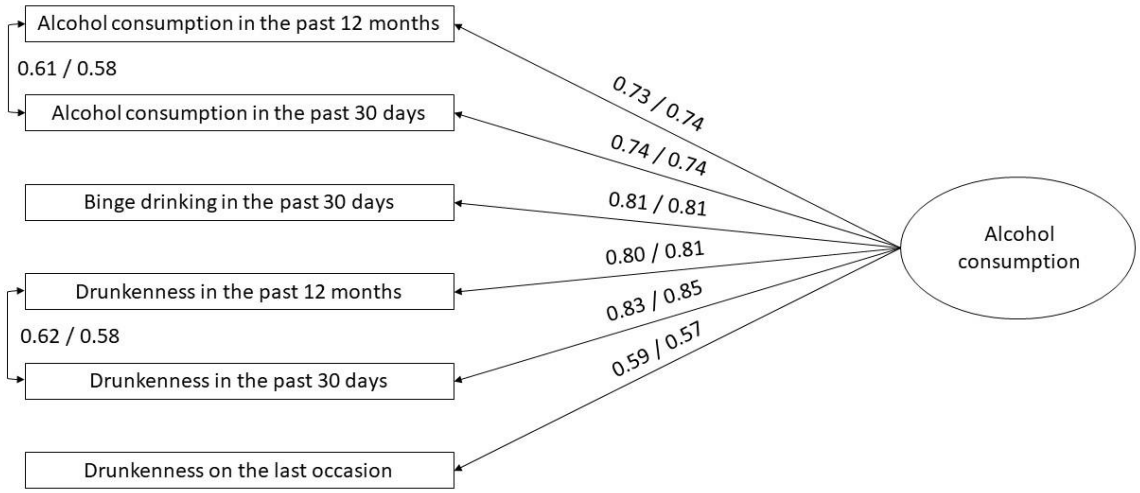
The present study investigated the indirect effect of risk for FEDs on alcohol consumption via drinking motives. Overall, the findings suggested that risk for FEDs were positively associated with internal drinking motives and alcohol use among males and females. It was possible to discriminate for females an indirect effect of risk for FEDs on alcohol consumption via internal drinking motives over the impact of depressive symptoms. Consequently, these findings fit previous data proposing a positive relationship between different symptoms of FEDs (e.g. purging, bingeing) and problematic alcohol use among adolescents (Arias et al., 2009; Kirkpatrick et al., 2019), and correspond with the assumption that individuals with a higher levels for restrained and disinhibited FED-related behaviors show a greater tendency for preoccupation with alcohol-related cognitions and prompts (Higgs & Eskenazi, 2007). Because individuals with co-occurring FEDs and heavy alcohol use might be at risk to experience more severe consequences due to alcohol consumption, treatment and prevention programs among treatment-seeking and non-treatment seeking adolescents should take into account and explore the role of drinking motives. Drinking motives are considered as important proximal predictors of alcohol use, therefore intervening at the level of drinking motives might subsequently contribute to less severe levels of alcohol use. Interventions working with adolescents showing a risk for FEDs might (i) provide personalized feedback for these individuals in terms of their drinking motives, (ii) help to identify high-risk situations of alcohol use when experiencing intense positive and negative emotions, (iii) introduce alternative strategies to regulate their positive and negative affect states instead of alcohol use, and (iv) train alcohol use-related protective or refusal skills for them when experiencing intense positive and negative emotions (Carey et al., 2007).

VII/7. Supplementary materials

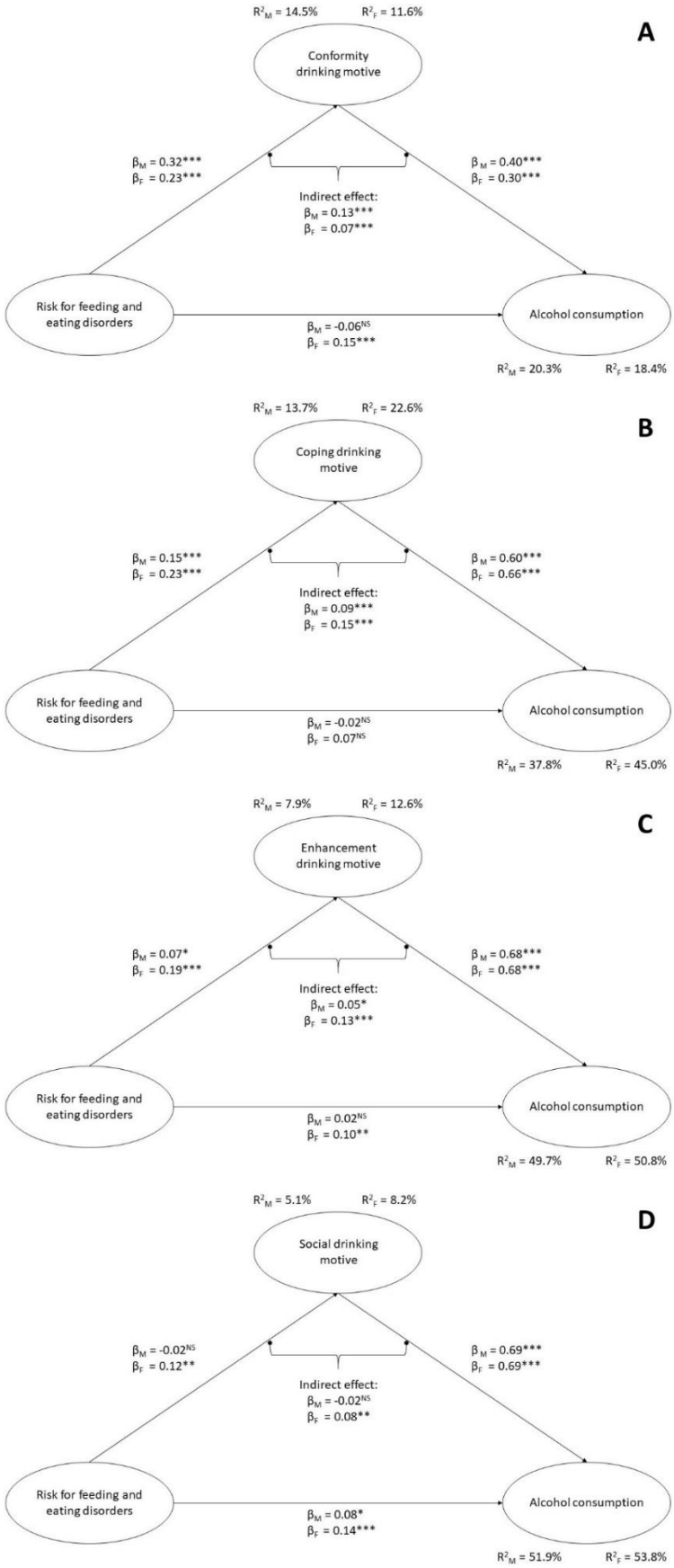
VII/Supplementary Table 1. Bivariate associations between symptoms of feeding and eating disorders (FEDs) and prevalence of alcohol use-related indicators

	Make her/himself sick because feeling uncomfortably full			Worry about losing control over eating			Recently lost more than one stone			Belief of being fat when others say to be too thin			Dominance of food over life		
	N (%)	χ^2 (*p*)	OR [95% CI]	*N* (%)	χ^2 (*p*)	OR [95% CI]	*N* (%)	χ^2 (*p*)	OR [95% CI]	*N* (%)	χ^2 (*p*)	OR [95% CI]	*N* (%)	χ^2 (*p*)	OR [95% CI]
Alcohol consumption in the past 12 months	238 (92.2%)	1.09 (0.297)	0.78 [0.49 – 1.25]	1486 (94.2%)	0.72 (0.396)	1.11 [0.87 – 1.43]	1006 (94.2%)	0.45 (0.504)	1.10 [0.83 – 1.47]	1177 (94.7%)	2.62 (0.106)	1.26 [0.95 – 1.66]	672 (93.6%)	0.03 (0.863)	0.97 [0.70 – 1.34]
Alcohol consumption in the past 30 days	183 (71.2%)	4.75 (0.029)	**1.36 [1.03 – 1.79]**	1037 (65.6%)	0.58 (0.446)	1.05 [0.93 – 1.19]	725 (67.8%)	5.08 (0.024)	**1.18 [1.02 – 1.36]**	840 (67.6%)	5.04 (0.025)	**1.17 [1.02 – 1.34]**	487 (67.9%)	3.39 (0.066)	1.17 [0.99 – 1.39]
Drunkenness in the past 12 months	160 (64.3%)	15.30 (<0.001)	**1.69 [1.30 – 2.20]**	827 (52.9%)	0.62 (0.431)	1.05 [0.93 – 1.18]	627 (59.5%)	28.96 (<0.001)	**1.46 [1.27 – 1.67]**	683 (55.8%)	8.39 (0.004)	**1.21 [1.06 – 1.38]**	389 (55.1%)	2.89 (0.089)	1.14 [0.98 – 1.35]
Drunkenness in the past 30 days	82 (33.2%)	19.93 (<0.001)	**1.85 [1.41 – 2.43]**	369 (23.6%)	4.43 (0.035)	**1.16 [1.01 – 1.34]**	290 (27.6%)	26.50 (<0.001)	**1.50 [1.28 – 1.75]**	319 (26.0%)	16.55 (<0.001)	**1.36 [1.17 – 1.58]**	170 (24.2%)	3.05 (0.081)	1.18 [0.98 – 1.42]
Binge drinking in the past 30 days	170 (65.6%)	35.05 (<0.001)	**2.17 [1.67 – 2.82]**	759 (47.8%)	0.01 (0.927)	1.01 [0.89 – 1.13]	613 (57.0%)	47.23 (<0.001)	**1.60 [1.40 – 1.84]**	636 (50.9%)	6.65 (0.010)	**1.18 [1.04 – 1.34]**	380 (52.7%)	8.55 (0.003)	**1.26 [1.08 – 1.48]**

Note. In each cell values of *N* represent the number of participants who reported about the use of the given form of alcohol consumption at least one occasion in the given time frame within those individuals who showed the given symptom of FEDs. In each cell values of % represent the proportion of participants who reported about the use of the given form of alcohol consumption at least one occasion in the given time frame within those individuals who showed the given symptom of FEDs. χ^2: Chi square statistics representing comparisons between those individuals who show and who do not show the given symptom of FEDs. OR: Odds Ratio. 95% CI: 95% Confidence Interval for Odds Ratios. Odds ratios in bold are significant at least $p < 0.05$ level. In each comparison, those were specified as reference category who did not show the given symptom of FEDs.



VII/Supplementary Figure 1. Construction of the latent variable measuring the level of alcohol consumption. Note. Square-shaped objects represent various observed indicator variables of alcohol use, while the ellipse-shape object represents the continuous latent variable measuring the level of alcohol consumption. Except for the variable assessing the level of drunkenness on the last occasion, all indicator variables of the latent variables were specified as categorical variables. Coefficients related to single-ended arrows are standardized factor loadings (λ), and coefficients related to double-ended arrows are error correlation coefficients (r) between observed indicator variables. Values left to the dash sign are coefficients measured among males (N = 2807), while values right to the dash sign are coefficients observed among females (N = 2796). All coefficients are significant at least $p < 0.001$ level.



VII/Supplementary Figure 2. Separate mediation models representing associations between risk for FEDs, different drinking motives (A: conformity-, B: coping-, C:

enhancement-, D: social drinking motive) and alcohol consumption. Note. Path coefficients are standardized regression coefficients (β). Indices presented with a letter "M" in subscript (e.g. β_M, R^2_M) are findings among males (N = 2807), while indices presented with a letter "F" in subscript (e.g. β_F, R^2_F) are findings among females (N = 2976). During the analyses, the effects of age, body mass index (BMI), depressive symptoms were controlled. "NS" in superscript represents non-significant ($p > 0.05$) regression coefficients, while p-values for significant ($p < 0.05$) regression coefficients are displayed by the following order: *$p \leq 0.05$; **$p \leq 0.01$; ***$p \leq 0.001$.

VIII. General discussion

VIII/1. Brief review of the findings of Studies 1-4

Although an extensive body of literature exists on the associations between alcohol consumption, problematic alcohol use, latent classes of alcohol users, drinking motives and various forms of psychopathological symptoms and disorders (Castillo-Carniglia et al., 2019; Cooper et al., 2015; Leggio et al., 2009), studies which were conducted as a part of this dissertation attempted to examine these relationships from new perspectives as well as in new contexts. Specifically, the present dissertation aimed to (i) identify empirically-based subgroups of alcohol users in clinical and general adult and adolescent samples and to examine their associations with various dimensions of psychopathological symptoms, and (ii) to investigate the role of drinking motives on the relationships between psychopathological symptoms and outcomes of alcohol use.

In Study 1, three subgroups of alcohol users were differentiated in a general adult population sample: (i) 'Light alcohol drinkers', (ii) 'Alcohol drinkers with low risk of dependence' and (iii) 'Alcohol drinkers with severe dependence symptoms' (Aim 1/a). The latter two latent classes of alcohol users presented higher levels of psychopathological symptoms. In multivariate analysis, there were significant and positive effects of depressive symptoms on the membership of 'Alcohol drinkers with low risk of dependence' and hostility symptoms on the membership of ''Alcohol drinkers with severe dependence symptoms' classes compared to 'Light alcohol drinkers' (Aim 1/b).

In Study 2, three latent classes of AUD inpatients attending a MM treatment program were identified with distinct profiles and change trajectories of psychopathological symptoms: (i) 'Low severity symptomatic subgroup with mild decrease', (ii) 'Moderate severity symptomatic subgroup with strong decrease', and (iii) 'High severity symptomatic subgroup with moderate decrease' (Aim 2/a). The subgroups with moderate and high symptomatic severity at the baseline demonstrated significantly higher levels of harmful alcohol use and drinking motives. Compared to the low symptomatic severity subgroup, multivariate analyses revealed significant and positive predictive effects of harmful alcohol use and conformity motives on the membership of 'Moderate severity symptomatic subgroup with strong decrease' class, whereas coping and conformity

motives were significantly and positively associated with the class membership of 'High severity symptomatic subgroup with moderate decrease' (Aim 2/b).

In Study 3, four latent classes of alcohol and illicit drug use were distinguished in a general adolescent population sample: (i) 'Infrequent substance users', (ii) 'Moderate alcohol users', (iii) 'High-risk alcohol users' and (iv) 'Polysubstance users' (Aim 3/a). Polysubstance users were characterized by elevated rates of GD symptom severity as well as higher endorsement rates of GD criteria of 'giving up other activities' and 'negative consequences' compared to the other substance using latent classes (Aim 3/b).

In Study 4 the mediating effect of internal drinking motives (i.e., a factor combining enhancement and coping motives) was shown between risk for EDs and alcohol use among females in a general adolescent population sample. That is, risk for EDs had a significant and positive effect on internal drinking motives, whereas internal drinking motives were positively associated with higher levels of alcohol use (Aim 4).

VIII/2. Discussion of the findings

Following the organization of the General introduction section, in this section it was aimed to review and discuss how the results of Studies 1-4 fit into the existing literature in three broad areas: (i) classification models of alcohol use and AUD, (ii) associations between externalizing and internalizing psychopathological symptoms and alcohol use-related outcomes, and (iii) the role of drinking motives on the relationships between psychopathological symptoms and alcohol use-related outcomes.

VIII/2/1. Classification models of alcohol use and AUD

In Studies 1 and 3 we used representative and population-based samples of adults and adolescents to identify subgroups of alcohol users and alcohol and illicit drug users. Both latent class models suggested that subgroups show increasing levels of risk for hazardous alcohol and substance use, and they can be placed along continuums of severity (Davoren et al., 2016; Halladay et al., 2020; Jackson, Bucholz, et al., 2014; Kuvaas et al., 2014; Sacco et al., 2009; Tomczyk et al., 2016). Specifically, latent classes in Study 1 were discriminated along a dimension of alcohol involvement severity ranging from light and infrequent drinking (i.e., 'Light alcohol drinkers') to heavy and problematic alcohol drinking (i.e., 'Alcohol drinkers with severe dependence symptoms'). Subgroups of alcohol and illicit drug use in Study 3 formed a dimension of substance use where the

subgroup of ‘Infrequent substance users’ were at lower severity levels (i.e., low level and experimental use of alcohol in addition to the absence of drunkenness and illicit drug use), whereas ‘Polysubstance users’ represented the higher end of this substance use continuum as they were characterized by the presence of current drunkenness and cannabis use and by the experiment of wide range of other illicit drug types.

At increasing levels of these continuums of alcohol and substance use involvement, distinct indicators differentiated between latent classes with rising severity levels (Halladay et al., 2020; Jackson, Bucholz, et al., 2014; Kuvaas et al., 2014; Sacco et al., 2009). In Study 1 differences between subgroups with low and moderate severity levels (i.e., ‘Light alcohol drinkers’ vs. ‘Alcohol drinkers with low risk of dependence’) were captured by alcohol consumption-related measures (i.e., frequency and quantity of use, heavy episodic drinking [HED]), whereas classes at moderate and higher severity points of the dimension (i.e., ‘Alcohol drinkers with low risk of dependence’ vs. ‘Alcohol drinkers with severe dependence symptoms’) were diverged by increasing levels of AUD symptoms and negative consequences. In Study 3 at low and moderate levels of the substance use continuum the presence of alcohol use in the past month and lifetime drunkenness differentiated between latent classes (i.e., ‘Infrequent substance users’ vs. ‘Moderate alcohol users’), between latent classes at moderate and high severity levels of the substance involvement continuum (i.e., ‘Moderate alcohol users’ vs. ‘High-risk alcohol users’) predominantly the presence of past month drunkenness discriminated, whereas at high and very high levels of the substance use spectrum the presence of past month cannabis use and lifetime use of other illicit drugs had high discrimination capacity between the latent classes (i.e., ‘High-risk alcohol users’ vs. ‘Polysubstance users’). These patterns in both studies are in accordance with previous studies using item response theory (IRT) which suggested that alcohol and substance use involvement can be conceptualized as a unidimensional latent continuum where discrimination capacity of different indices varies at different severity levels of the latent continuum (e.g., binge drinking is more informative at lower levels compared to AUD symptoms, whereas in the case of the substance use involvement continuum alcohol use is located at lower levels and illicit drug use at higher levels) (Kirisci et al., 2002; Saha et al., 2020).

The identified latent classes in Studies 1 and 3 correspond to previous findings regarding subgroups of adult alcohol use and adolescent substance use. For example, previous classification models among adults repeatedly discriminated classes of low and infrequent

alcohol users, regular and/or heavy episodic users with only mild levels of AUD symptoms, and high-risk alcohol users who experience AUD symptoms and negative consequences (Jackson, Bucholz, et al., 2014; Sacco et al., 2009; Smith & Shevlin, 2008). The classification model of alcohol users in Study 1 can also be harmonized with the DSM-5's severity-based distinction of AUD subtypes (i.e., increasing levels of AUD symptoms and negative consequences across the three latent classes) and with the hierarchical structure of problematic alcohol use proposed by the ICD-11 (i.e., differentiating heavy alcohol drinking groups with and without the presence of AUD symptoms) (American Psychiatric Association, 2013; World Health Organization, 2018b). Moreover, classification models of adolescent alcohol and illicit drug use consistently identified subgroups of light or experimental alcohol drinkers, regular alcohol users with moderate levels of consumption, heavy alcohol users with frequent and excessive consumption and polysubstance users with excessive alcohol use and use of wide range of illicit drug types (Dauber et al., 2009; Davoren et al., 2016; Gohari et al., 2020; Halladay et al., 2020; Jackson, Denny, et al., 2014; Tomczyk et al., 2016). Overall, the findings of Studies 1 and 3 can contribute to enhance cross-cultural generalizability of adult and adolescent alcohol and substance use classes. To the best of the Author's knowledge, previous studies only limitedly examined classification models of alcohol and substance use in representative and general population samples in the CEE region (and specifically in Hungary) which is characterized by high levels of adult and adolescent alcohol use and alcohol use-related harms (Bräker et al., 2015; Göbel et al., 2016; Halladay et al., 2020; Inchley et al., 2020b; Shield et al., 2020; Tomczyk et al., 2016).

The latent classes of AUD in Study 2, which were isolated based on distinct profiles and change trajectories of psychopathological symptoms, highlight the importance of considering co-occurring psychopathological disorders and symptoms in typologies of AUD. Similarly, previous binary and multiclass taxonomies of AUD also emphasized that co-occurring externalizing and internalizing psychopathology (e.g., subgroups with marked negative affectivity and/or antisocial characteristics) can explain at least party the heterogeneity among individuals with AUD (Del Boca & Hesselbrock, 1996; Hesselbrock & Hesselbrock, 2006; Hildebrandt et al., 2017; Leggio et al., 2009; Moss et al., 2007; Windle & Scheidt, 2004). The classification model in Study 2 suggested severity-based and quantitative differences between subgroups of AUD, thus latent classed had mostly parallel and quantitatively distinct symptomatic profiles and they did

not show qualitatively distinct constellations of psychopathological symptoms (i.e., subgroups differed in the overall severity level of psychopathological symptoms and there were no classes with predominantly internalizing or externalizing psychopathology). The retained typology of AUD individuals is also comparable with previous studies which attempted to identify subgroups of AUD by considering exclusively co-occurring psychiatric disorder presence or psychopathological symptom levels. Specifically, Villalobos-Gallegos et al.'s (2017) taxonomy also proposed severity-based discrimination between classes of AUD ranging between mild and severe psychopathological symptom levels. Moreover, the identified subgroups in Study 2 were congruous with previous studies which used co-occurring psychiatric disorder presence as classification indicators as they also repeatedly identified classes with overall low and moderate-high levels of comorbid psychopathological severity (Glass et al., 2014; Müller et al., 2020; Sintov et al., 2010; Urbanoski et al., 2015). However, it is important to note that the identified latent classes of Study 2 are only limitedly comparable with the latter studies as they used binary classification indicators to measure the presence of a comorbid psychiatric disorder (i.e., instead of continuous variables measuring symptom severity), some of these classification models were based on non-treatment seeking samples of individuals with AUD in addition to the more widespread measurement of externalizing psychiatric disorders in these studies (e.g., different forms of SUDs, ASPD) (Glass et al., 2014; Müller et al., 2020; Sintov et al., 2010; Urbanoski et al., 2015). Taken together, the findings of Study 2 suggest that not only the qualitative features and types of comorbid psychiatric disorders (i.e., AUD with co-occurring internalizing or externalizing pathology) but differences along a continuum of overall psychopathological severity should be considered when accounting for the heterogeneity within AUD population (see further discussion in the next section).

As Study 2 focused not only on the differences between subgroups of AUD in terms of psychopathological symptom severity and specific symptomatic constellations but also considered changes in symptomatic levels, the retained classification model might contribute to broadening existing knowledge on taxonomies of AUD. Specifically, one of the identified subgroups (i.e., 'Moderate severity symptomatic subgroup with strong decrease' class) presented higher levels of psychopathological symptomatic improvement during the eight weeks-long period of the MM treatment program. Although methodological limitations did not allow to specify treatment effects on the symptomatic

changes (e.g., not controlling for withdrawal symptoms and detoxification effects, absence of randomized controlled trial [RCT] design), this classification model highlighted the importance of investigating longitudinal, psychopathology-related changes and stability of classes of AUD with different characteristics of psychopathological symptoms as well as examining differences in treatment outcomes and responses between subgroups of AUD with different comorbid psychopathological severity levels (Roos et al., 2017). For example, in a previous study, a latent class that was characterized by elevated rates of comorbid ASPD and internalizing psychiatric disorders (e.g., MDD) showed more adverse longitudinal outcomes in terms of stability of AUD, treatment utilization, levels of mental health and alcohol use-related outcomes (Moss et al., 2010).

These findings on psychopathological changes among individuals with AUD also underline the need for understanding on how twelve step-based treatment forms, such as the MM program, can contribute to reductions in psychopathological symptom severity. As these therapeutic approaches primarily focus on addressing patients' problems related to alcohol use, the possible beneficial effects on co-occurring psychopathological outcomes might be mediated or moderated by other factors. For example, it cannot be ruled out that detoxification effects and attenuation of withdrawal symptoms at the initial period of the treatment attendance can explain at least partly the decrease in psychopathological distress over the eight week-long treatment period. Alternatively, it might be possible that the application of supplementary therapeutic techniques (e.g., stress management training, assertiveness training) contributed to the beneficial effects in psychopathological symptom levels. Finally, previous studies suggested that the positive link between increased levels of AA attendance and reductions in depressive symptoms can be explained by possible beneficial effects related to spiritual growth (e.g., increasing rates of self-esteem, social support, use of adaptive coping strategies) (Wilcox et al., 2015).

VIII/2/2. Associations between psychopathological symptoms and alcohol use-related outcomes

VIII/2/2/1. Externalizing psychopathological characteristics

The findings of Studies 1-3 demonstrated co-occurrence of different forms of externalizing psychopathological characteristics and alcohol use, problematic alcohol

consumption. In addition to this, these studies also confirmed the relevance of externalizing psychopathological symptoms in the classification models of alcohol use and AUD (Halladay et al., 2020; Hildebrandt et al., 2017; Leggio et al., 2009; Moss et al., 2007).

Studies 1 and 2 showed significant and positive associations between hostility and alcohol use-related outcomes: subgroups of alcohol use and AUD with higher levels of harmful alcohol use (i.e., consumption and negative consequences) were characterized by elevated rates of hostility compared to the least severe latent classes. The measured construct of hostility in both studies encompassed symptoms of aggressive tendencies (e.g., urges to harm someone), difficulties to regulate distress (e.g., temper outbursts) and irritability (e.g., being easily annoyed) (Derogatis & Savitz, 2000; Unoka et al., 2004; Urbán et al., 2014). Previous studies also reported significant and positive associations between anger, hostility and higher severity levels of alcohol use (Bácskai et al., 2011; Gerevich et al., 2007). Although the applied methodological designs and statistical analyses in Studies 1 and 2 did not allow to determine causal and structural relationships between the variables, previous theoretical and empirical findings can suggest explanations for the co-occurrence between hostility and problematic alcohol use. It might be possible that hostility, alcohol use and problems related to alcohol consumption are all indicators of a broad, higher-order and transdiagnostic dimension of externalizing disorders which explains the co-occurrence between them and represents a shared liability to externalizing behaviors (e.g., neurobiological and psychological features of negative affectivity, stress regulation, behavioral dysregulation, impulsivity) (Eaton et al., 2015; Krueger et al., 2007; Krueger & South, 2009). In other words, this hierarchical concept of externalizing behaviors suggests that variations in terms of hostility, alcohol use and symptoms of AUD is simultaneously explained by the corresponding specific factors (e.g., hostility, AUD) and by a higher-order and transdiagnostic factor of externalizing behaviors (Krueger et al., 2007; Krueger & South, 2009). Alternatively, dimensional psychiatric classification models, such as the Hierarchical Taxonomy of Psychopathology (HiTOP), differentiated two broad, higher-order dimensions of externalizing disorders which can capture the co-occurrence between them: disinhibited and antagonistic externalizing disorders (Castillo-Carniglia et al., 2019; Kotov et al., 2017). According to the HiTOP, AUD is a part of the disinhibited externalizing spectra at the level of disorders (under the subfactor of substance abuse), whereas alcohol use and alcohol problems are included in the model as

components of the disinhibited externalizing spectra at lower, symptom component level of the hierarchy. Symptoms of hostility are included at lower, symptom component level in this model. However, multiple higher-order dimensions contain components and traits which are related to the construct of hostility: irritability, emotional lability and hostility are components and traits of the distress subfactor (under the higher-order internalizing factor), whereas aggressive behaviors and urgency are components and traits of the antisocial behavior subfactor (under the higher-order factors of disinhibited and antagonistic externalizing disorders) (Kotov et al., 2017). However, it is important to note that other causal explanations can also be assumed for the significant and positive association between hostility and problematic alcohol use, such as one can use alcohol as a means for coping with the distressful affective states of irritability and aggressive urges, whereas symptoms of hostility can also emerge as a consequence of problematic alcohol use (i.e., as withdrawal symptoms).

In Study 3, the latent class of 'Polysubstance users' provided support for the co-occurrence of high levels of alcohol use (i.e., frequent alcohol use with excessive patterns) and illicit drug use (i.e., current use of cannabis and at least experimental use of a wide range of other illicit drugs). According to Tomczyk et al.'s (2016) recommendation for definition, the pattern of polysubstance use refers to the concurrent use of at least three psychoactive substances, such as alcohol, tobacco and cannabis. Previous studies demonstrated that concurrent use of alcohol and illicit drugs is associated with increased risk for negative outcomes, such as substance use-related problems, negative social consequences, and adverse outcomes of mental health (e.g., high levels of externalizing and internalizing psychopathologies, such as antisocial behaviors, ADHD, depressive symptoms) (Halladay et al., 2020; Tomczyk et al., 2016; Yurasek et al., 2017). Although only cautious explanations are possible for the background of the co-occurrence of alcohol and illicit drug use as Study 3 did not test any mechanisms on this issue, it is might be worth considering the assumptions of the common liability to addiction (CLA) model (Vanyukov et al., 2012). The CLA is conceptualized as a continuous, latent dimension which represents increasing risk levels for the presence of AUD and SUDs. Moreover, this model suggests that there are shared, non-substance-specific etiologic risk factors and correlates of alcohol and drug use and problems that contribute to the CLA as well as can be accounted for the concurrent use of these substances. For example, there is an overlap between different substance use forms in neurobiological characteristics

(e.g., areas responsible for behavioral regulation, reward mechanisms and reward deficiency syndrome, stress response), environmental risk factors (e.g., child abuse and trauma, being a member of a peer group with antisocial tendencies) and psychological mechanisms (e.g., impulsivity, behavioral dysregulation, sensation seeking) (Thatcher & Clark, 2008; Vanyukov et al., 2012). In line with this approach, it was shown that different psychoactive substances can be placed along a unidimensional latent continuum of substance use involvement which can explain the associations among the included substances (Kirisci et al., 2002).

However, the 'Polysubstance users' class were not only characterized by the concurrent use of alcohol and illicit drugs, but these adolescents also showed elevated rates of GD symptom severity and criteria endorsement. Therefore, these findings show similarities with previous studies which reported significant and positive associations between levels of GD and outcomes of alcohol and drug use (Burleigh et al., 2019; Estévez et al., 2017; Kotyuk et al., 2020; Marmet, Studer, Wicki, et al., 2019). However, to the best of the Author's knowledge, Study 3 was the first which examined whether the concurrent use of alcohol and illicit drugs (i.e., polysubstance use pattern) is associated with levels of GD. Overall, it might be possible that a subgroup of at-risk individuals can be distinguished who not only show risky substance use patterns (i.e., frequent and excessive alcohol use in addition to current cannabis use and at least experimental use of other illicit substances), but also characterized by problematic use of potentially addictive behaviors (i.e., higher symptomatic severity and criteria endorsement of GD). It is important to note that Study 3 did not allow exploring the background mechanisms of this co-occurrence; therefore, any explanations regarding the co-occurrence between GD and polysubstance use is only cautiously and limitedly possible. Previous empirical findings and theoretical models of addictions highlighted that there are shared and common genetic, neurobiological (e.g., areas responsible for reward functions and reward deficiency syndrome, executive functions) and psychological precursors (e.g., impulsivity, negative affectivity, maladaptive emotion regulation, coping motivations behind these potentially addictive behaviors) and similar symptomatic characteristics (e.g., overlapping and similar problems related to these addictive behaviors, obsessive-compulsive features in symptomatology) between substance use-related problems and potentially addictive behaviors (Burleigh et al., 2019; Estévez et al., 2017; Kotyuk et al., 2020; Marmet, Studer, Lemoine, et al., 2019; Paulus et al., 2018; Walther et al., 2012). Alternatively, causal

pathways can also be assumed between alcohol and illicit drug use and GD, such as substance use before and during gaming might promote decreased control over gaming behavior and might lead to negative consequences, whereas those who experience adverse consequences due to gaming behavior can also start to use alcohol and illicit drugs in order to dampen stress and negative emotional states due to these gaming problems (Cowlishaw et al., 2014; Škařupová et al., 2018).

VIII/2/2/1. Internalizing psychopathological characteristics

Studies 1, 2 and 4 provided support for the positive associations between various internalizing psychopathological symptoms and alcohol use-related outcomes. However, to obtain more accurate understanding on these significant relationships it might be worth to take into account the measured construct of alcohol use (e.g., alcohol consumption, alcohol use-related problems) and the type of the internalizing psychiatric disorders (e.g., symptoms of mood disorders and ADs, EDs).

To assess the unique associations between levels of alcohol consumption (i.e., considering primarily levels of frequency and quantity of use, HED and not alcohol problems) and symptoms of MDD and ADs in Study 1, differences between classes of 'Light alcohol drinkers' and 'Alcohol drinkers with low risk of dependence' were investigated. Bivariate and multivariate analyses indicated that higher levels of alcohol use were associated with elevated symptom severity of MDD, whereas in terms of symptoms of ADs (i.e., GAD, OCD) non-significant differences were shown between 'Light alcohol drinkers' and 'Alcohol drinkers with low risk of dependence'. That is, these findings highlighted the role of MDD symptoms among non-symptomatic alcohol users with at least moderate levels of alcohol use frequency and quantity and presence of HED. However, previous classification models which used adult samples from the general population were incongruous with these findings: negative and non-significant associations were shown between measures of MDD and classes with moderate-high levels of alcohol consumption and low-mild levels of alcohol use-related consequences (compared to classes with low/mild consumption levels) (Sacco et al., 2009; Smith & Shevlin, 2008). Previous typologies of alcohol use among non-clinical adult individuals rather suggested that rates of MDD and GAD varies as a function of alcohol use-related symptoms and problems and not by the levels of alcohol consumption per se. That is, subgroups of alcohol users with higher rates of alcohol use-related symptoms and

problems showed increased levels of MDD and GAD (Kuo et al., 2008; Sacco et al., 2009; Smith & Shevlin, 2008). In line with this, other literature findings with variable-centered analytical approaches also showed that levels of MDD are more closely correlated with symptoms of AUD than with measures of alcohol consumption (Bulloch et al., 2012). Moreover, modelling non-linear or quadratic (i.e., U- and J-shaped) associations between levels of MDD and alcohol consumption can also contribute to obtaining a more accurate understanding. Namely, the presence of alcohol abstinence and heavy alcohol drinking is positively linked to elevated rates of MDD compared to light and moderate alcohol users (Gea et al., 2012; O'Donnell et al., 2006). Finally, although it was not the primary aim of Study 4, significant, weak and positive bivariate association was shown between symptoms of MDD and alcohol use among adolescents. Similarly, meta-analytic findings showed significant, weak associations between higher frequency and quantity of alcohol consumption and elevated rates of MDD among adolescents (Cairns et al., 2014).

Studies 1 and 2 allowed to explore associations between alcohol use-related problems and symptoms of MDD and different types of ADs in the adult general population and clinical samples. Bivariate analyses in Study 1 presented that 'Alcohol drinkers with severe dependence symptoms' showed increased symptomatic levels of MDD, GAD and OCD compared to 'Light alcohol drinkers'; however, these associations did not remain significant in the multivariate model (most likely due to the effect of hostility). Previous classification models which used adult samples from the general population reported significant and positive associations between MDD, GAD and class memberships of alcohol users with high rates of alcohol use and alcohol consumption-related problems in bivariate and multivariate models (compared to non-problematic and low alcohol users) (Casey et al., 2013; Kuo et al., 2008; Sacco et al., 2009; Smith & Shevlin, 2008). The suppressing effect of hostility might implicate that there are a more robust associations between different indicators of the broad, higher-order externalizing spectrum (i.e. hostility, alcohol use and alcohol problems) than the links between symptoms of AUD and internalizing psychopathologies (Eaton et al., 2015; Kotov et al., 2017; Krueger & South, 2009). However, it is also important to note that the construct of hostility also contains distress-related elements which can show overlap with elements of MDD and GAD (i.e., irritability, emotional lability) and might have to be considered when assessing the relationships between AUD and mood disorders and ADs (Kotov et al., 2017). Moreover, it is also important to consider that not all internalizing psychopathological

symptom domains of the BSI were measured in Study 1 (e.g., subscales of somatization, phobic anxiety, paranoid ideation, and psychoticism were not included in the applied abbreviated version of the BSI). That is, it cannot be ruled out that some of these, unmeasured internalizing psychopathological symptoms would have shown a significant relationship with the class membership of 'Alcohol drinkers with severe dependence symptoms'. On the other hand, practical as well as theoretical considerations justified the use of this abbreviated version of the BSI. First, by using the 27-item version instead of the full, 53-item of the BSI it was possible to reduce the participants' burden and fatigue regarding completion of the questionnaire. Second, previous studies reported that the specific symptom subscales of the BSI provide limited unique explanatory effect over the general psychopathological distress factor in explaining common variances of the scores in the questionnaire (Urbán et al., 2014). Thus, it might be possible that the inclusion of the aforementioned subscales would have increased only limitedly the information on psychopathological symptoms.

Bivariate and multivariate analyses of Study 2 demonstrated that latent classes of AUD with at least moderate psychopathological symptomatic severity at the baseline showed significantly higher levels of harmful alcohol consumption (which simultaneously considered levels of alcohol consumption and alcohol problems) compared to the 'Low severity symptomatic subgroup with mild decrease' subgroup. Specifically, symptomatic profiles of the more severe classes suggested that members of the 'Moderate severity symptomatic subgroup with strong decrease' and 'High severity symptomatic subgroup with moderate decrease' classes experienced symptoms of MDD, GAD and OCD at the highest levels among the psychopathological symptoms. Similarly, previous studies which used co-occurring psychiatric disorder presence as classification indicators also reported that compared to subgroups with low levels of comorbid psychopathological severity more severe classes (e.g., classes with predominantly externalizing or internalizing comorbid psychiatric disorders, and classes with high overall comorbid psychopathological levels) were characterized by higher levels of alcohol consumption and symptomatic severity of AUD (Glass et al., 2014; Müller et al., 2020; Sintov et al., 2010).

Taken together, the latter findings of Studies 1 and 2 are at least partly in line with previous literature data, which suggested significant and positive associations between AUD and MDD and different types of ADs, such as GAD and OCD (Anker & Kushner,

2019; Boden & Fergusson, 2011; Castillo-Carniglia et al., 2019; Conner et al., 2009; Cuzen et al., 2014; Grant et al., 2015; Lai et al., 2015; Smith & Randall, 2012). Moreover, other studies also highlighted the importance of considering co-occurring internalizing psychopathological symptoms (e.g., MDD, GAD) as an important element of classification models of alcohol use and AUD (Del Boca & Hesselbrock, 1996; Hesselbrock & Hesselbrock, 2006; Hildebrandt et al., 2017; Leggio et al., 2009; Moss et al., 2007; Windle & Scheidt, 2004). Due to the applied methodological designs, only cautious assumptions should be made on the possible mechanisms which can account for the co-occurrence of elevated levels of alcohol use, alcohol problems and symptoms of MDD and ADs. In the case of Study 2, the positive association between harmful alcohol use and subgroups of AUD with increasing overall (predominantly internalizing) psychopathological severity might indicate the possible presence of a higher-order, general psychopathological factor. For example, the p-factor (i.e., a general psychopathological factor in a hierarchical, bifactor model where symptomatic variations of a particular psychiatric disorder are simultaneously explained by the general and specific psychopathological factors, such as externalizing, internalizing or thought disorders; Caspi et al., 2014) as well as the higher-order super spectra level in the HiTOP model (Kotov et al., 2017) can account for the covariation of internalizing and externalizing psychiatric disorders. These dimensions can represent a non-specific, common liability factor to suffer from various forms of psychiatric disorders and account for psychopathological severity and impairment and shared etiological factors (Caspi et al., 2014; Kotov et al., 2017). That is, higher levels on a higher-order, shared liability psychopathological factor might explain the co-occurrence of externalizing (i.e., higher rates of alcohol use, alcohol problems) and internalizing characteristics (i.e., more severe symptoms of MDD and ADs) among individuals at the moderate and high severity subgroups. This approach can also be harmonized with those findings which reported shared genetic, neurobiological (e.g., functions related to stress response), environmental (e.g., early negative life and abusive experiences) and psychological influences (e.g., emotion regulation difficulties, anxiety sensitivity) between MDD, various types of ADs and AUD (Aldao et al., 2010; Anker & Kushner, 2019; Castillo-Carniglia et al., 2019; Garey et al., 2020; Hussong et al., 2011; Selby et al., 2008; Smith & Randall, 2012). However, causal models can also be assumed to explain the associations between MDD, various types of ADs and alcohol use and negative consequences due to alcohol use in Studies 1 and 2. For example, based on the self-medication hypothesis, symptoms of

MDD and ADs can predict subsequent alcohol use and problems via the motivation to mitigate symptoms of MDD and ADs and cope with elevated distress related to higher psychopathological severity with alcohol drinking. Alternatively, increased and problematic alcohol use can lead to the subsequent presence of symptoms of MDD and various types of ADs via neurobiological changes (e.g., higher sensitivity to negative affectivity due to withdrawal symptoms) and adverse social consequences (e.g., difficulties in interpersonal life, family, financial situation due to problematic alcohol use) (Anker & Kushner, 2019; Boden & Fergusson, 2011; Conner et al., 2009; Groenman et al., 2017; Hussong et al., 2011; Le Moal & Koob, 2007; Pedrelli et al., 2016; Smith & Randall, 2012; Turner et al., 2018).

Finally, in Study 4, higher levels of symptom severity of EDs (i.e., core symptoms of AN and BN) were associated with higher rates of alcohol consumption (i.e., simultaneously considering levels of alcohol use frequency and excessive use) among female adolescents even over the effects of age, BMI and depressive symptoms. That is, this finding is in accordance with previous empirical data which presented significant and positive links between measures of alcohol use (e.g., drunkenness, past month alcohol use) and different forms and characteristics of EDs (e.g., drive for thinness, body dissatisfaction, symptoms of BN) (Arias et al., 2009; Baker et al., 2017, 2018). However, Study 4 can only limitedly contribute to the existing knowledge on the co-occurrence of different forms of EDs and alcohol use-related outcomes (Bahji et al., 2019; Bogusz et al., 2021; Gadalla & Piran, 2007) as symptoms of AUD or problems related to alcohol use as well as symptomatic severity of specific forms of EDs (e.g., AN, BN, BED) were not assessed. Specifically, the SCOFF questionnaire provided superficial measurement for a few core symptoms of AN and BN, whereas symptoms of BED were not assessed. Moreover, most items of the SCOFF are simultaneously related to multiple diagnostic categories (e.g., symptoms of "Make her/himself sick because feeling uncomfortably full", "Worry about losing control over eating" and "Dominance of food over life" can be presented in both AN and BN), therefore it was only limitedly possible to examine specific relationships between alcohol use and symptoms of AN and BN (see: VII/Supplementary Table 1). The cross-sectional design did not allow to test any hypotheses on the mechanisms of the co-occurrence between symptoms of EDs and alcohol use; therefore, the below assumptions on this issue should be interpreted cautiously. Shared neurobiological correlates (e.g., areas related to reward processes and behavioral control) and psychological and affective characteristics

(e.g., both risk behaviors associated with elevated levels of internalizing symptoms, neuroticism, maladaptive emotion regulation, impulsivity as well as similar motivational mechanisms are shared between them) can explain the aforementioned positive correlation (Ferriter & Ray, 2011; Schulte et al., 2016). Alternatively, the concept of food and alcohol disturbance (FAD) suggested that symptoms and characteristics of EDs (e.g., restrictive tendencies, bulimic characteristics) and alcohol use can be associated functionally (Choquette et al., 2018; Rahal et al., 2012). Namely, one might perform compensatory, calorie restrictive behaviors before (e.g., eating less to get drunk), during (e.g., not eating or eating only low-calorie foods to get drunk or to compensate the calories in alcohol) and after (e.g., skipping meals to compensate the calories due to previous alcohol intake) alcohol use in order to enhance the effects of alcohol or to compensate previous or anticipated calorie intake (Rahal et al., 2012).

VIII/2/3. The role of drinking motives on the relationships between psychopathological symptoms and alcohol use-related outcomes

Studies 2 and 4 contributed to broadening the existing knowledge on the function of drinking motives on the relationships between psychopathological symptoms and alcohol use-related outcomes. Specifically, these findings highlighted the role of drinking motives with negative reinforcement mechanisms (i.e., coping and conformity motives) as well as drinking motives with internal sources (i.e., enhancement and coping motives).

Multivariate analysis in Study 2 showed that compared to AUD individuals with low overall psychopathological severity, those with moderate and high baseline severity levels showed elevated rates of coping and conformity motives. According to the motivational model of alcohol use, both types of motives are based on negative reinforcement mechanisms; that is, in these cases, alcohol use is motivated to avoid and reduce negative expected effects and consequences. In coping motives, alcohol drinking aims to alleviate and mitigate negative emotional states, whereas alcohol use due to conformity motives aims to avoid social disapproval or rejection (Cooper et al., 2015; Cox & Klinger, 1988; Kuntsche et al., 2005). Some authors hypothesized that drinking motives with negative reinforcement mechanisms can provide a maladaptive and hazardous motivational background for alcohol use (e.g., due to maladaptive cognitive processes and cognitive biases) (Cooper et al., 2015). To the best of the Author's

knowledge, this was the first time that subgroups of AUD with distinct profiles of co-occurring psychopathological symptoms were compared in terms of drinking motives.

As the classes with at least moderate baseline psychopathological symptom severities also showed higher levels of harmful alcohol consumption, the findings of Study 2 are indirectly in accordance with previous findings, which suggested that coping motives are positively linked to alcohol consumption and problems (Bresin & Mekawi, 2021; Cooper et al., 2015). The classification model in Study 2 was predominantly considered internalizing psychopathological symptoms and subgroups of AUD with moderate and high baseline psychopathological severities were characterized by the highest symptom severity of MDD, GAD, OCD and interpersonal sensitivity. Therefore, the observed significant and positive associations between these latent classes of AUD and coping motives are congruous with previous literature data. For example, higher levels of coping motives were associated with elevated rates of features associated with negative affectivity and internalizing symptomatology, such as neuroticism, anxiety sensitivity, negative urgency, symptoms of MDD, GAD, SAD, and OCD (Allan et al., 2015; Bakhshaie et al., 2021; Bravo et al., 2018; Cooper et al., 2015; Schry & White, 2013). Some theoretical models of AUD can account for the associations between these moderately-highly severe AUD latent classes of psychopathology and coping motives. For example, the self-medication hypothesis proposes that symptoms of MDD and ADs can predict subsequent alcohol use and problems via the alcohol drinking motivation to mitigate and cope with symptoms of MDD and ADs (Anker & Kushner, 2019; Hussong et al., 2011; Morris et al., 2005; Smith & Randall, 2012; Turner et al., 2018). That is, coping motives might mediate the effect of higher overall psychopathological distress among AUD individuals in the more severe psychopathological latent classes on adverse alcohol-related outcomes. Alternatively, based on the allostatic model of AUD, it might be possible that these more severe latent classes of AUD represented more severe forms of AUD which can be characterized by the dominance of negative reinforcement mechanisms and symptoms of MDD and ADs can overlap with withdrawal symptoms of AUD (i.e., drinking to cope with high levels of withdrawal symptoms, distress) (Anker & Kushner, 2019; Koob, 2011; Le Moal & Koob, 2007). Finally, it might be important to consider the function of emotion regulation difficulties as well (e.g., negative urgency, rumination) on the relationship between more severe psychopathological subgroups of AUD and coping motives (i.e., affected individuals might use alcohol to cope with

psychopathological distress in absence of the use of adaptive emotion regulation strategies) (Aldao et al., 2010; Cheetham et al., 2010; Selby et al., 2008).

Existing research also reported that conformity drinking motives are associated with distinct patterns of distal psychopathological antecedents. Namely, significant and positive relationships were shown between conformity motives and anxiety sensitivity, symptoms of SAD and BPD (Cooper et al., 2015; Kaufman et al., 2020; Schry & White, 2013). These relationships might indicate that those individuals who experience more severe difficulties in their interpersonal relationships and show problems related to assertiveness might drink more frequently to avoid social disapproval and rejection. It might be possible that these individuals lack effective interpersonal skills which would be needed for adequate functioning in social gatherings; therefore their alcohol drinking might aim to mitigate distress related social discomfort and anxiety and to enhance their inclusion in a given social group (Kaufman et al., 2020; Schry & White, 2013). In line with this, symptomatic profiles of the classes of 'Moderate severity symptomatic subgroup with strong decrease' and 'High severity symptomatic subgroup with moderate decrease' indicated increased levels of interpersonal sensitivity (e.g., symptoms related to perceived interpersonal rejection) among these individuals with AUD. However, it is important to note that it was not possible to test the assumed mechanisms on the associations between negative reinforcement drinking motives and classes of AUD in Study 2, thus these assumptions should be interpreted cautiously.

Findings of Study 4 highlighted the mediating function of internal drinking motives (i.e., a composite measure comprising enhancement and coping motives) on the relationship between symptoms of EDs and alcohol consumption among adolescents. According to the motivational model of alcohol use, drinking motives with internal sources refer to motives which are characterized by self-directed expected consequences of alcohol use. Individuals drinking because of enhancement and coping reasons are expected to obtain changes in their internal and affective states by using alcohol. In the cases of coping motives, alcohol drinking aims to alleviate and mitigate negative emotional states, whereas enhancement motives cover reasons for alcohol use which aim to experience and enhance pleasurable affective and psychophysiological states (Cooper et al., 2015; Cox & Klinger, 1988; Kuntsche et al., 2005). Previous studies highlighted the role of internal drinking motives on alcohol use-related outcomes: enhancement motives were the most strongly associated with different measures of alcohol consumption, whereas

enhancement and coping motives showed the strongest associations with alcohol use-related problems (Bresin & Mekawi, 2021; Cooper et al., 2015; Kuntsche et al., 2005). However, it is important to note that a specific assessment of the effects of enhancement and coping motives was not possible in Study 4.

In line with the findings of Study 4, existing literature data demonstrated that individuals with different forms of EDs (e.g., BN, BED) consistently showed higher levels of coping motives and enhancement motives of alcohol use (Anderson et al., 2006; Luce et al., 2007; Mikheeva & Tragesser, 2016; Trojanowski et al., 2019). However, to the Author's best knowledge, Study 4 tested and reported for the first time the mediating effect of drinking motives with internal source on the relationship between symptoms of EDs and alcohol use. That is, Study 4 fits and extends those previous research data which suggested the mediating role of drinking motives between distal predictors of psychopathological symptoms and alcohol use-related outcomes (Allan et al., 2015; Bakhshaie et al., 2021; Bravo et al., 2018; Grazioli et al., 2019; Kaufman et al., 2020; O'Hare & Sherrer, 2011; Terlecki & Buckner, 2015). As the design of Study 4 did not allow to empirically explain the background of the observed mediational pathway, theoretical explanations should be only considered cautiously. Previous studies reported shared motivational processes (e.g., alcohol using and eating behavior used as a function of emotional modulation) and psychological and affective characteristics (e.g., negative affectivity, emotion regulation difficulties, reward seeking tendencies) between different forms of EDs and alcohol use which might support the findings of Study 4 (Ferriter & Ray, 2011; Schulte et al., 2016; Trojanowski et al., 2019). In line with this, it was hypothesized that frequent and regular presence of enhancement and coping drinking motives might indicate that alcohol drinking is used as a dominant way to regulate positive and negative emotional states (e.g., to experience positive emotions when the individual shows low levels or lack of positive affective states, to enhance intensity of positive emotions, to cope with negative affectivity) as the alcohol using person might be short of adaptive emotion regulation strategies (Cheetham et al., 2010; Cooper et al., 2015). Alternatively, it is also possible that the symptoms of EDs can contribute to high rates of psychological distress and adverse intra- and interpersonal outcomes; therefore, alcohol use among the affected individuals might serve as a way to cope with these negative consequences which might subsequently lead to elevated rates of alcohol use.

VIII/2/4. Limitations and future directions

The findings of the dissertation should be interpreted cautiously due to various methodological limitations of the included studies. First, samples used in the dissertation might bias the findings. For example, although Studies 1, 3 and 4 used representative samples of the Hungarian adult and adolescent population, it might be possible that prevalence of alcohol use and alcohol problems were underestimated and these studies only reached limitedly specific risk populations with increased rates of risk behaviors. Future studies might consider oversampling specific, at-risk demographic subgroups during representative sampling procedures to ensure more accurate assessment of alcohol use in the adolescent and adult population. Moreover, in Studies 1 and 4 non-alcohol users were excluded from the analyses which also limits the generalizability of the findings. The applied convenience and non-representative sampling in Study 2 might have influenced characteristics of the classes and contributed to limited generalizability of the identified subgroups of AUD (e.g., those with less severe AUD pathology or non-treatment-seeking AUD individuals were underrepresented or not included). Therefore, future studies should consider using large representative samples of individuals with AUD to examine subgroups of co-occurring psychopathological characteristics.

Second, the methodological designs of Studies 1-4 impeded the exploration of causal mechanisms between alcohol use-related variables and psychopathological symptoms. For example, cross-sectional design of Studies 1, 3 and 4 did not allow to investigate bidirectional relationships between psychopathological symptoms and alcohol use and alcohol problems (e.g., it was not possible to determine whether different forms of psychopathological symptoms precede alcohol use or rather induced by alcohol use). Moreover, in the cases of studies which aimed to establish classification models of alcohol use and AUD, the longitudinal stability and prognosis of the identified latent classes were not (i.e., Studies 1 and 3) or only limitedly (i.e., Study 2) examined. The applied before-after design of Study 2 (i.e., absence of control group, blinding, randomization and long-term follow-up) was also insufficient to determine the specific effect of the MM treatment program on psychopathological changes. The application of RCT design can allow to design more tailored treatment programs and to explore more precisely differential treatment responses of latent classes with different characteristics of alcohol use and psychopathological symptoms (Roos et al., 2017). Moreover, Study 2 did not assess factors explaining treatment drop-out (e.g., psychopathological symptoms, drinking motives), whereas in absence of systematic and quantitative long-term follow-

up data it was not possible to examine relapse rates after treatment and predictors of relapse (e.g., psychopathological symptoms, drinking motives). Future studies might also focus to examine these factors regarding the treatment program of the MM. Specifically, future studies might use machine learning-based analytical approaches to prospectively predict dropping out from treatment, relapse or remission after treatment based on initial proximal alcohol use-related variables (e.g., drinking motives), AUD- or psychopathological symptoms and personality characteristics (Kim et al., 2021; Kinreich et al., 2021). Overall, future studies should consider using longitudinal design with multiple measurement points to examine more precisely temporal stability and prognosis of latent classes of alcohol users (e.g., AUD- and other psychiatric disorder-related treatment involvement, maturing out) and to explore causal relationships between alcohol use-related variables and psychopathological symptoms (e.g., patterns of sequential comorbidity). For example, by establishing latent classes with different trajectories of alcohol use (e.g., classes showing stable, increasing or decreasing patterns of high alcohol use and problems) and examining their longitudinal relationships between psychopathological symptom levels and psychopathological symptom changes across these measurements (i.e., Studies 1-3), it would be possible to discriminate and assess bidirectional causal mechanisms between outcomes of alcohol use and psychopathological symptoms (e.g., whether increase in symptoms of depression, hostility or GD lead to harmful changes in alcohol use, or vice versa). Moreover, by using ecological momentary assessment (EMA) it would be possible to assess within-person relationships between alcohol use and problems, drinking motives and psychopathological symptoms, in addition to considering between-person effects (Stevenson et al., 2019; Wycoff et al., 2020). For example, regarding the suggested indirect effect of symptoms of EDs on alcohol consumption via drinking motives (i.e., Study 4), a study with longitudinal design (e.g., with EMA design or using cross-lagged analysis) could provide more detailed insight on the assumed causal pathways (e.g., whether more severe symptoms of AN and BN lead to increased alcohol use via higher use of internal drinking motives, or vice versa). Finally, future longitudinal studies using treatment samples of individuals with AUD might also focus on exploring how changes in drinking motives (e.g., decrease in coping motives might lead to maturing out) and in cognitive-motivational factors of abstinence (e.g., increasing levels of motivation and engagement to change on the problematic alcohol using behavior, different motivations for abstinence [e.g., fear from loss of control, negative consequences], increasing levels

of drinking refusal self-effeicacy) are associated with treatment outcomes and changes in outcomes of alcohol use and psychopathological symptoms (Anderson et al., 2013; de Visser & Piper, 2020; Littlefield et al., 2010).

Third, although the theoretical justification for Studies 1 and 3 was related to the need to explore subgroups of alcohol users in the CEE region (Aims 1/a and 3/a), these findings only included individuals from the Hungarian adult and adolescent populations. Therefore, cross-cultural generalizability of these latent class models was not tested. Future studies might explore similarities and differences among CEE countries to broaden the existing knowledge on latent classes of alcohol use.

Fourth, the measurement of alcohol use- and psychopathology-related constructs was biased in several aspects. It might be possible that self-report measures of alcohol consumption and problems related to alcohol use as well as psychopathological symptoms were confounded by recall and social desirability bias. In all of the studies in the dissertation the measurement of variables regarding alcohol use and psychopathologies was not complete, such as not measuring specific symptoms of AUD (e.g., in Studies 1-4) and problems related to adolescent alcohol use (e.g., in Studies 3 and 4), assessing only a limited number of psychiatric disorders (e.g., in Studies 1 and 2), not covering all aspects and symptoms of psychiatric disorders by the applied measurements (e.g., in Studies 3 and 4), using non-specific constructs of alcohol use, drinking motives and psychiatric disorders (e.g., in Studies 2 and 4). Moreover, all the studies in the dissertation were limited by not including relevant confounding or third variables which can account for the associations between alcohol use-related outcomes and psychopathological symptoms (e.g., impulsivity, sensation seeking, negative urgency, emotion regulation strategies and difficulties, alcohol expectancies). In this regard it is important to highlight that none of the studies in the dissertation assessed and considered the effects of the presence of lifetime and current psychiatric comorbidities (e.g., the presence of gambling disorder in Study 2, ADHD in Study 3, ADs in Study 4). Taken together, future studies might consider using standardized measurements (e.g., clinical interviews) of symptoms of AUD and psychiatric disorders based on the DSM-5 or the ICD-11 as well as aiming to include wider-range of psychiatric disorders and relevant covariates of comorbidity and to measure more specifically alcohol use and drinking motives. In addition to these, it would be also worth to examine further the utility and relevance of broad, transdiagnostic factors of psychopathology regarding comorbidity of

AUD and other psychiatric disorders, investigate shared and specific neurobiological features of AUD and other psychiatric disorders and explore symptomatic level associations between these disorders (e.g., by using network analysis) (Afzali et al., 2017; Anker et al., 2019; Caspi et al., 2014; Kotov et al., 2017).

VIII/3. Conclusions and practical implications

The present dissertation aimed to examine associations between alcohol consumption and various forms of psychopathological symptoms by identifying empirically-based subgroups of alcohol users in clinical and general adult and adolescent samples and examining their associations with various dimensions of psychopathological symptoms and by investigating the role of drinking motives on the relationships between psychopathological symptoms and outcomes of alcohol use. Studies 1-3 identified latent classes of alcohol use and AUD with increasing severity levels and highlighted the role of externalizing and internalizing psychopathological symptoms in alcohol classification models. Moreover, Studies 2 and 4 highlighted the role of negative reinforcement and internal drinking motives on the associations between outcomes of alcohol use and psychopathological symptoms.

By using representative, population-based adult and adolescent samples in Studies 1 and 3, respectively, it was possible to assess and identify individuals who show high-risk and hazardous patterns of alcohol and illicit drug use. Specifically, approximately 9% of the adult alcohol users showed high levels of AUD symptoms, whereas approximately 7% of the adolescent population was characterized by polysubstance use patterns. Exploring prevalence of high-risk alcohol users in the adult and adolescent population and describing their psychological characteristics might be informative for policymakers to design more tailored prevention and intervention programs. For example, it might be worth to consider applying further public health strategies in Hungary to decrease the level of harms related to alcohol use. For example, previous studies reported that increasing excise taxes on alcohol as well as applying restrictions on alcohol availability and marketing can be cost-effective strategies to control alcohol use at population level (Chisholm et al., 2018). Identifying and screening for at-risk individuals with hazardous and harmful patterns of alcohol use and elevated levels of co-occurring psychopathological symptoms is warranted because of the high levels of health and social

burden attributable to alcohol use and to the comorbidity of AUD and other psychiatric disorders as well as due to the high treatment gap for AUD (Griswold et al., 2018; Hjorthøj et al., 2015; Rehm et al., 2013; Samokhvalov et al., 2010; Shield et al., 2020). For example, performing brief screening programs for harmful alcohol use among clients at general practitioner services, emergency departments and school-level might have a potential to identify at-risk individuals who might require assistance regarding their difficulties on alcohol use. Moreover, findings from Studies 3-4 might suggest that those professionals who work with specific adolescent populations showing a risk for EDs or GD (e.g., in child psychiatry or addictology) can consider applying routine screening for harmful and risky alcohol and substance use to identify individuals who might be at-risk for showing co-occurring risk behaviors. Service providers at the abovementioned levels might consider using brief questionnaires to assess harmful alcohol and substance use patterns, such as the AUDIT or the Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) (Saunders et al., 1993; WHO ASSIST Working Group, 2002). Moreover, these screening programs might specifically focus on aspects of alcohol use which are meaningfully differentiate between subgroups of alcohol users with different severity levels (e.g., excessive and episodic alcohol use for classes of alcohol users with moderate severity, whereas alcohol use-related problems or polysubstance use patterns for classes of alcohol users with high severity levels). These subgroups of alcohol users might require differential intervention approaches (e.g., teaching alcohol use-related protective behavioral strategies for less severe users, directing symptomatic alcohol users to AUD-specific treatment programs) (Kenney et al., 2014). Taken together, it might be important to consider selecting appropriate levels of care for latent classes of alcohol users with different severity levels and ensuring to provide treatment forms which are suitable and tailored for the needs of a given subgroup of alcohol users (Magura et al., 2003).

Findings of the present dissertation indicated that higher levels of alcohol use and problems are co-occurring with various psychopathological symptoms. Therefore, intervention and prevention programs regarding alcohol use might consider targeting not only aspects of alcohol use but rater using a broader focus which simultaneously covers domains of substance use and mental health (Teesson et al., 2020). Similarly, existing literature on treatment-related issues for individuals with co-occurring AUD and other psychiatric disorders proposed various treatment models, such as sequential (i.e., AUD and the co-occurring psychiatric disorder is treated one after the other), parallel (i.e.,

simultaneous but distinct treatment of AUD and the co-occurring psychiatric disorder by different treatment approaches) and integrated treatment forms (i.e., simultaneous and unified treatment of AUD and the co-occurring psychiatric disorder by the same treatment team) (Farren et al., 2012; Flanagan et al., 2018; Smith & Randall, 2012; Yule & Kelly, 2019). In the latter case, psychotherapies might consider emphasizing the functional association between the comorbid disorders. These treatment programs might include elements of motivational enhancement therapies (e.g., enhancing motivation to change on current patterns of alcohol use), twelve-step-related approaches (e.g., receiving mutual support by members of a self-help group, increasing abstinence-focused engagement), relapse prevention focused therapies (e.g., recognizing and handling high-risk situations, promoting drinking refusal skills), cognitive behavioral therapies (e.g., techniques to handle MDD and GAD, such as cognitive restructuring, focusing on negative automatic thoughts, teaching effective coping and relaxation methods), contingency management (e.g., reinforcing the presence of positive outcomes and penalize the presence of negative outcomes) as well as address transdiagnostic characteristics which might explain the interrelationship between the comorbid disorders, such as focusing on processes regarding emotion regulation (e.g., teaching to have non-judgmental approach on emotions and to accept the distressing affective states, enhancing the use of adaptive emotion regulation strategies and correcting maladaptive ones, relaxation programs), reward dysfunction (e.g., teaching to reach pleasurable and desired affective states with natural reinforcers), impulsivity and behavioral control, assertive communication and effective social skills (Arias & Kranzler, 2008; Farren et al., 2012; Flanagan et al., 2018; Garofalo & Wright, 2017; Helle et al., 2019; McHugh & Weiss, 2019; Smith & Randall, 2012; Yule & Kelly, 2019). The present findings might also highlight the need for concentrating on drinking motives-related mechanisms which can influence the association between problematic alcohol use and psychopathological symptoms. Moreover, intervention and treatment programs working with adolescents who are at-risk for or showing EDs or GD might implement elements which aim to explore and highlight functional associations between these psychiatric disorders and alcohol use. For example, it might be crucial to assess high-risk situations for alcohol use among these individuals (e.g., screening for concurrent or consecutive presence of these behaviors: alcohol might be used during gaming which subsequently contributes to a more problematic pattern of gaming and alcohol use, or excessive restrictive or dieting behaviors before and after drinking alcohol), present alternative and more adaptive strategies to reach more effective

control over alcohol use (e.g., teaching adaptive emotion regulation and coping strategies to avoid using alcohol as a means for regulating emotional experiences, introducing alcohol use-related protective strategies), exploring motivational similarities and differences between alcohol use and eating behavior or gaming (e.g., overlap between the behaviors as they are used to reach rewarding and pleasurable experiences or to reduce negative affectivity).

IX. References

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