Alcohol and Depression:

Can This Connection be Explained by Cognitive Processes?

Inaugural dissertation

for the degree of Doctor of Philosophy

(Dr. phil.)

Presented at the Faculty of Human Sciences of the University of Cologne

according to the doctoral regulations from December 18, 2018

submitted by

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born June 13, 1989 in Heidelberg

September 2021

This dissertation was accepted by the Faculty of Human Sciences of the University of Cologne in February 2022.

Acknowledgement

A big thank you goes to Prof. Dr. Alexander Gerlach and Prof. Dr. Johannes Ehrenthal for their willingness to review this dissertation. In particular, I would like to thank Alexander Gerlach for "catching me up" with his mentoring and helping me to take new courage. I thank him for his patience, his advice, his seemingly inexhaustible knowledge and his optimism.

Another thank you goes to my colleagues Anna Pohl and Linda Mehrmann, who motivated me, found encouraging, supportive words for me and who always made me believe that I could master all challenges. I would especially like to thank Anna Pohl for the good cooperation in our work and her guidance in handling the biodata.

I would also like to thank Gerhard Mutz, because without his technical knowledge and support I would have been stuck with the programming and evaluation of my experiment.

A big thank you goes to all participants who agreed to support my research project. Without you, the implementation would not have been possible in the first place. I admire your motivation and perseverance and have taken both as a model for my own work.

And I would also like to thank the students Claudia Mueller-Weinitschke, Sarah and Nina Jaspert, who supported me with their great work in the implementation of study 1.

A final thank you goes to all those people close to me who have supported and advised me during this challenging time.

Table of Contents

Summary

Despite the high comorbidity between affective disorders and substance use disorders, such as alcohol use disorder (AUD), and numerous theoretical assumptions attempting to explain this relationship, the underlying mechanisms have received little empirical investigation and consequently have not yet been sufficiently identified.

One way to explain this connection may lie in the functioning of pathological cognitive processes linked to depression. Rumination[1] and metacognitive beliefs about rumination[2] contribute to the maintenance of depression and promote the use of dysfunctional coping strategies, such as the consumption of alcohol, to reduce rumination. Alcohol is considered an easily accessible coping strategy that attempts to reduce negative feelings / emotions such as sadness, thus self-medicating depressive symptoms that have been intensified by rumination. In consequence, this may result in an increase in consumption of alcohol as well as increased alcohol-related problems. Unfortunately, empirical research is lacking to establish (1) whether these mechanisms promote further use and (2) what specific disorder-related processes underlie the increased comorbidity of major depressive disorder (MDD) and AUD.

The two studies conducted in this dissertation address these same potentially underlying mechanisms, particularly the function of pathological cognitive processes in the context of clinical depression and negative alcohol-related consequences:

- Study 1 examined the direct influence of alcohol on disorder-specific cognitive (rumination and metacognitions), emotional symptoms (sadness), and psychophysiological processes (heart rate, heart rate variability, muscle tension) in a population of individuals with MDD.
- Study 2 used structural equation modeling to examine the function of rumination in the context of depressive symptomatology, drinking motives (including drinking to cope), and negative alcohol-related consequences (i.e., alcohol consumption and alcohol-related problems).

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[1] The repetitive negative thinking about past events, possible causes and consequences of negative emotions (Nolen-Hoeksema, 1991). For a detailed definition, see Section 1.1.

[2] This can be, for example, the belief that rumination is helpful. For a detailed definition, see Section 1.4.1.

Study 1 (Gawron et al., in revision) investigated whether and how alcohol consumption affects disorder-specific processes in MDD. Of particular interest was the extent to which alcohol consumption activates and influences metacognitions. To this end, a worry induction paradigm (Borkovec & Inz, 1990) was adapted to a sample of individuals with MDD (N = 65). Specifically, instead of worry, ruminative thinking was induced (for further details, see Chapter 2: Study 1, procedure).

In the first part of the randomized controlled experiment, subjects completed a clinical diagnostic interview. In the second part of the study, they participated in an experiment in which they first consumed either alcohol (vodka with orange juice), placebo (orange juice ostensibly containing vodka) or pure orange juice. This drinking phase was followed by a rumination induction and a relaxation phase. While the subjects were ruminating or trying to relax, psychophysiological parameters were recorded. They also rated their levels of sadness, tension, and rumination, as well as metacognitions before (first measurement point, T1) and after the rumination phase (T2) and at the end of the relaxation phase (T3).

No significant differences were found between the group that consumed alcohol and the other two groups that did not consume alcohol (placebo and control) in terms of self-reports, metacognitions, or psychophysiology at all three measurement points. However, induced rumination led to increased sadness, increased muscle tension, and increased heart rate. These results suggest that the consumption of alcohol has no influence on disorder-specific processes in MDD – i.e., also no influence on cognitions or metacognitions. Thus, the study failed to clarify the extent to which the consumption of alcohol was actually used to cope with cognitive processes in MDD (as postulated by the Metacognitive Model of Rumination and Depression, MCM; Papageorgiou & Wells, 2003). Also, the function of (meta-)cognitions within the comorbidity of AUD and MDD could not be clarified.

In study 2 (Gawron & Gerlach, in revision), a structural equation model was developed for a clinically depressed population (N = 209), trying to explain the relationship between depressive symptoms and negative alcohol-related consequences via ruminative processes and drinking motives.

During an online survey, all relevant variables / indicators of the model were recorded using self-reports. Only individuals who were currently undergoing treatment for depressive symptoms or had

been undergoing treatment participated in the study. First, we followed the model specifications of Bravo et al. (2018): Depressive symptoms were predictors of rumination, with four rumination factors acting as second-order indicators of a rumination factor. Rumination was modeled as a predictor of the four drinking motives (conformity, coping, enhancement, social), which in turn predicted alcohol use and alcohol-related problems (depressive symptoms → rumination → drinking motives → negative alcohol-related consequences). In this model, only a few significant direct associations were found: Between depressive symptoms and rumination, as well as between enhancement and coping drinking motives and negative alcohol-related consequences. In a simplified model, in which only one drinking factor was defined, it was possible to show direct significant associations between the individual factors of the described structure. Furthermore, depression had a significant positive effect on alcohol-related problems and rumination. Rumination, in turn, had both a significant positive effect on drinking motives and a significant negative effect on alcohol-related consequences.

Drinking motives as well as cognitive processes seem to be important for the association of depressive symptoms and alcohol-related consequences. However, based on the present results, the function of rumination remains unclear. It is possible that rumination itself is used as a form of coping with aversive emotional states (cf. Papageorgiou & Wells, 2003), which might make the use of other strategies, such as alcohol consumption, unnecessary and could provide an explanation for the reduced alcohol-related consequences. Considering the direct effects of the simplified model, it may also be hypothesized that depression increases rumination, which leads to the consumption of alcohol to regulate the aversive experience, thus encouraging further consumption. Therefore, there is also the possibility that alcohol consumption is chosen as another form of coping strategy to reduce unpleasant feelings that have been reinforced by repetitive negative thoughts (i.e., rumination; in accordance with the Emotional Cascade Model, ECM; Selby et al., 2008).

1. General Introduction and Integration

"Those who have worries also have liquor."

(Pious Helen, Wilhelm Busch)

1.1 Repetitive Negative Thinking: Rumination

Repetitive negative thinking (RNT) can be perceived as disturbing, time-consuming, and very stressful. Both worries[3] about potentially stressful events in the future and rumination about stresses in the past are among such repetitive cognitions.

According to the *Response Styles Theory of Depression* (Nolen-Hoeksema, 1991), rumination, a hallmark of depressive disorders, is defined as recurrent negative thinking and passively focusing on symptoms of depression and distress (Nolen-Hoeksema, 1991), past events, and possible causes and consequences of aversive feelings / symptoms of distress (Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema et al., 2008).

On the one hand, the decision to ruminate may be quite conscious (Papageorgiou & Wells, 2004), as it is understood as a way of responding to distress (Nolen-Hoeksema, 1991) and may be an attempt to cope with stress and negative feelings (Matthews & Wells, 2004); on the other hand, rumination is a maladaptive form of self-reflection (Nolen-Hoeksema et al., 2008) that leads to the reinforcement of negative affect (Papageorgiou & Wells, 2004), as well as contributes to the development (Huffziger et al., 2009; Weber & Exner, 2013) or intensification of depressive episodes (Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema & Harrell, 2002). The negative effect of rumination is, among other things, attributed to the fact that focusing on negative emotions increases accessibility to mood-congruent cognitions, causing negative thoughts, appraisals, and memories to build up (Nolen-Hoeksema, 1991).

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[3] Worry is regarded as the central defining feature of generalized anxiety disorder (GAD) and is defined as a process involving predominantly verbal thought activity that relates to uncertain / future events with potentially negative consequences, is accompanied by negative affect, and is experienced as uncontrollable (Borkovec et al., 1998).

Moreover, rumination is associated with increased negative thinking, poor problem solving, impaired motivation, concentration, and cognition, inhibits instrumental (coping) behavior, and promotes further stress and social problems (for a review of studies on negative consequences, see Lyubomirsky & Tkach, 2004; Nolen-Hoeksema et al., 2008). Lyubomirsky and Tkach (2004) hypothesize a vicious cycle between rumination, depression, and these same negative consequences, with rumination and depression activating this cycle. The individual parts of the cycle may interact or feedback with each other, and the sequence of these relationships may follow different paths. As an example, the authors cite that people who are depressed and ruminate tend to interpret their circumstances pessimistically and confirm their view by recalling mood-congruent memories. Thus, they think about their problems for too long without developing a solution. Due to a lack of motivation, they do not succeed in putting potentially good solutions into action. As a result, the problems cannot be solved, get worse and reinforce the already existing negative mood.

Individuals with high negative self-attention, that is, individuals who ruminate, are thought to try to suppress this negative self-focus. They use dysfunctional behaviors, such as self-harm, excessive eating, or drinking, to suppress their negative thoughts and feelings (Nolen-Hoeksema et al., 2008). In addition to depression, rumination thus favors the development of other forms of psychopathology, such as bulimic behaviors or substance abuse (Abramson et al., 2006; Nolen-Hoeksema & Harrell, 2002). Further evidence on the association between rumination and alcohol can be found in Section 1.3.

1.2 The General Effects of Alcohol

In order to understand why alcohol is used, for example, to cope with rumination and depression, it is also necessary to understand the general effects of alcohol. Therefore, some key theories of alcohol effects are summarized in the following (for an overview, see Battista et al., 2010; Sayette, 2017).
With regard to the effects of alcohol, a distinction can be made between two effects (Sayette, 2017):

- Alcohol has a negative reinforcing effect (a negative state is diminished).
- Alcohol has a positive reinforcing effect (a pleasant state is intensified); although the empirical evidence on this is inconsistent (Sayette, 2017) and therefore not discussed in detail below.

An early approach, the *Tension-Reduction Hypothesis* (TRH; Conger, 1956), assumes that alcohol is a negative reinforcer consumed for its tension- and anxiety-reducing effects on the central nervous system. While “no simple direct relation between the pharmacologic effect of alcohol and its behavioral consequences” (Wilson, 1978, p. 317) has been found, it has encouraged the development of further theories incorporating additional moderating factors, such as gender, learned expectations about the effects of alcohol, or setting in which consumption occurs (Battista et al., 2010; Sayette, 2017).

Another key model is the *Stress-Response Dampening Model* (Sher & Levenson, 1982), which postulates that alcohol consumption does not generally reduce stress, but does improve the stress response, while accounting for personality traits and individual differences. For example, it is hypothesized that individuals with factors indicative of high trait social anxiety should experience greater reductions in state anxiety through the consumption of alcohol than individuals with lower levels of these trait factors (Battista et al., 2010).

Furthermore, cognitive factors appear to mediate the anxiolytic effects of alcohol (Sayette, 2017). Three theories address these same cognitive processes: The *Self-Awareness Model* (SAM; Hull, 1981), the *Attention Allocation Model* (AAM; Steele & Josephs, 1988), and the *Appraisal-Disruption Model* (ADM; Sayette, 1993). The commonality of the models is that they address social anxiety and assume that the cognitive processing of social anxiety-related information is disrupted by alcohol, resulting in a reduction of anxiety. Thus, consumption functions as a short-term coping strategy for individuals who are socially anxious (Battista et al., 2010).

In the following section, these different approaches are briefly explained: The SAM assumes that the consumption of alcohol can interfere with the encoding of self-relevant information, thereby reducing performance-related self-evaluation. Especially in unpleasant situations with negative self-evaluation, the stress response is reduced and, conversely, the probability of further consumption is increased.

In the course of the AAM it is assumed that the perceptual ability is impaired by alcohol, whereby the focus of attention is directed exclusively to the most salient cues of the environment (called "alcohol myopia"). Individuals with social anxiety are thought to find it quite rewarding to have their

attention narrowed to other salient stimuli, rather than to the anxiety-related stimuli that are normally in focus (Battista et al., 2010).

The ADM postulates that alcohol disrupts the initial appraisal of information by weakening the link between new and old information. Accordingly, a stressful social situation experienced in an intoxicated state is likely to be interpreted as less threatening because other situations / information stored as threatening cannot be retrieved in long-term memory, leading to a reduction in social anxiety.

It can be assumed that the consumption of alcohol also influences cognitive processes in individuals with MDD and furthermore favors a reduction of depressive symptoms. Theoretical considerations on the significance of cognitive processes in the connection between MDD and alcohol use (disorder) as well as assumptions about the emotion-regulating effect of alcohol can be found in Section 1.4.

1.3 Rumination, Depression, and Alcohol

Rumination is generally associated with increased negative mood (Papageorgiou & Wells, 2004; Simons et al., 2016) and specifically contributes to the maintenance of depression (Nolen-Hoeksema et al., 2008). Unhelpful, and in some cases, even harmful, strategies are often used to cope with these recurring unpleasant thoughts, such as smoking, unhealthy eating (Clancy et al., 2016), the use of drugs, or the consumption of alcohol (Clancy et al., 2016; Nolen-Hoeksema & Harrell, 2002). Previous studies have shown that rumination is associated with increased alcohol use (Bravo et al., 2017; Ciesla et al., 2011; Heggeness et al., 2021; Simons et al. 2016) and alcohol-related problems (Bravo et al., 2018; Sorid et al., 2021). Accordingly, it is reasonable to assume that rumination is associated with both negative feelings, such as depressed mood, and negative alcohol-related consequences.

With this in mind, it is not surprising that the presence of a depressive disorder increases the risk of developing AUD (Boschloo et al., 2011; Kessler et al., 1997), and that the comorbidity between affective and substance use disorders is accordingly elevated (Boden & Fergusson, 2011; Grosshans & Mann, 2012; Wolitzky-Taylor et al., 2011).

Despite the multitude of different theories explaining the association between alcohol use (disorder) and other mental disorders (cf. Battista et al., 2010), the underlying mechanisms of this high

comorbidity have rarely been empirically investigated – especially with regard to the link between AUD and MDD.

The following hypotheses and models describe some key notions trying to understand this connection, including a discussion of the function of pathological cognitive processes such as rumination.

1.4 Depression and Alcohol: Alcohol as a Coping Strategy

1.4.1 Alcohol for Coping With Cognitions: The Metacognitive Model of Rumination and Depression

The *Metacognitive Model of Rumination and Depression* (MCM; Papageorgiou & Wells, 2003) emphasizes the function of thought processes in the context of depressive symptomatology and alcohol (use): Certain triggers (e.g., unpleasant thoughts) initially activate positive metacognitive beliefs about the benefits of rumination (e.g., "To understand my depressive feelings, I need to think about my problems."). Such assumptions motivate individuals to engage in rumination and to continue to do so. However, rumination may also activate negative metacognitive beliefs regarding the uncontrollability and danger of rumination (e.g., "I can't stop myself from ruminating.") as well as regarding negative social consequences of rumination (Papageorgiou & Wells, 2004). Also, these negative metacognitive beliefs may contribute to the maintenance of depression by eliciting additional feelings, such as hopelessness (Huntley & Fisher, 2016). Moreover, negative metacognitions possibly promote the use of dysfunctional coping strategies to reduce or avoid rumination. The use of alcohol represents one such strategy (Mollaahmetoglu et al., 2021; Wells et al., 2011).

Clinical (e.g., Papageorgiou & Wells, 2003) and nonclinical studies (see, e.g., Solem et al., 2016; Weber & Exner, 2013; Yilmaz et al., 2015) have shown that metacognitive beliefs about rumination are significant for the onset (Faissner et al., 2018) and maintenance (Huntley & Fisher, 2016; Solem et al., 2016) of depressive states / depression. Metacognitions are furthermore associated with other psychopathologies, such as generalized anxiety disorder (GAD), obsessive-compulsive disorders (OCD), or eating disorders (see review by Sun et al., 2017), as well as problematic alcohol use (Spada & Wells, 2005; Spada et al., 2007). For example, it was shown that in unpleasant emotional states, less alcohol is consumed in the presence of adaptive metacognitions than in the presence of maladaptive

metacognitions (Moneta, 2011), such as the negative belief that one must control thoughts (Spada et al., 2007).

In addition, RNT, such as rumination and worry, are associated with increased use in individuals with AUD (Devynck et al., 2019). Moreover, as mentioned earlier in Section 1.3, rumination is associated with alcohol consumption (Caselli et al., 2008; Caselli et al., 2010; Riley et al., 2019) and negative alcohol-related problems (Aldao et al., 2010; Nolen-Hoeksema & Harrell, 2002; Willem et al., 2011), as well as unhealthy behaviors (Clancy et al., 2016). However, these studies often focus on the drinking to cope approach (e.g., Heggeness et al., 2019; Thornton et al., 2012; see also Section 1.4.2), typically use self-reports (e.g., Bravo et al., 2018), and examine nonclinical samples (e.g., Bravo et al., 2017).

1.4.2 Alcohol for Coping With Emotions: The Self-Medication Hypothesis

The *Self-Medication Hypothesis* (SMH) assumes a direct causal relationship between two disorders, i.e., the presence of one, e.g., anxiety disorder, favors the development of the other, e.g., AUD (Bolton et al., 2009). According to this hypothesis, individuals with anxiety states / anxiety disorders tend to abuse alcohol or drugs in order to reduce their emotional stress and negative affect (Quitkin et al., 1972). These considerations are in turn based on the work on the tension-relieving and anxiety-reducing effects of alcohol (TRH), which in the sense of negative reinforcement favors further consumption in situations associated with stress and anxiety (Conger, 1956).

Previous research mostly focused on self-medication with alcohol in anxiety states or anxiety disorders (see, e.g., Bolton et al., 2006; Carrigan & Randall, 2003; Gerlach et al., 2006; Robinson et al., 2009; Thomas et al., 2003) rather than affective disorders (Bolton et al., 2009). However, findings on self-medication in this area also suggest that substances are used to alleviate mood problems, particularly depressive mood (e.g., Bolton et al., 2009; Weiss et al., 2004).

An approach very similar to the SMH, namely *drinking to cope*, describes the use of alcohol for negative reinforcement, that is, to escape, avoid, or otherwise regulate negative feelings (Abbey et al., 1993; Cooper et al., 1995). Drinking to cope has been associated with tension reduction expectancies (Cooper et al. 1995), alcohol use and alcohol-related problems (see, e.g., Abbey et al., 1993; Bravo et

al., 2017; Bravo et al., 2018; Cooper et al. 1995; Grunberg et al., 1999), as well as depressed mood (Holahan et al., 2003), and rumination (Bravo et al., 2017; Bravo et al., 2018).

Using structural equation modeling, it was shown that depressive symptoms lead to increased rumination, which in turn is associated with more coping drinking motives and then results in increased alcohol consumption as well as alcohol-related problems (Bravo et al., 2018). The same research group also addressed the question of whether, in addition to coping motives, other drinking motives[4] (enhancement, conformity, social) influence the decision to consume alcohol, but found no significant effects in this regard. The relationships postulated by the authors (Bravo et al., 2018) were found to be significant for the most part, but the model assumptions have so far only been tested in a nonclinical population (college students).

1.4.3 Alcohol for Coping With Rumination-Induced Emotional States: The Emotional Cascade Model

The *Emotional Cascade Model* (ECM; Selby et al., 2008), originally developed in the context of impulsive behaviors, postulates that negative emotions and ruminative thinking influence each other in a self-reinforcing manner. In order to break this vicious cycle of intense negative feelings and rumination ("emotional cascade"), the use of impulsive behaviors may occur, such as alcohol use / binge drinking, binge eating, or self-harm (Selby et al., 2016). In addition, the presence of emotional cascades may increase the risk of developing a mental disorder, such as borderline personality disorder (BPD; Selby & Joiner, 2009).

For different forms of impulsive behaviors in comorbid mental disorders, like MDD, GAD, and social phobia (Selby et al., 2016), BPD (Selby et al., 2009) and OCD (Jungmann et al., 2016), as well as a nonclinical population (Selby et al., 2008) the assumptions of the model have been empirically confirmed.

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[4] The theoretical considerations of the four drinking motives are provided by a motivational model of alcohol consumption (Cox & Klinger, 1988) that has already been empirically validated (Cooper, 1994).

Mollaahmetoglu and colleagues (2021) examined how alcohol consumption affects rumination in a group of individuals with risky consumption and to which extent this effect can be explained by a decrease in negative mood. They discovered that alcohol reduced rumination at a low dose and also influenced rumination by altering mood. More precisely, people who ruminate are at risk of consuming alcohol to reduce aversive feelings that have been intensified in advance by ruminative thinking (Mollaahmetoglu et al., 2021). Thus, the consumption of alcohol may represent a strategy for coping with rumination-induced emotional cascades (Selby et al., 2008).

1.5 Summary of the Empirical Work

The goal of this cumulative dissertation was to gain a better overall understanding of the underlying mechanisms linking negative alcohol-related consequences to depression-specific processes. For this reason, two studies investigated the relationship between alcohol consumption and pathological cognitive processes.

Based on the findings presented in the previous section, the following questions arose for the studies:

Study 1: How does alcohol consumption affect disorder-specific cognitive, emotional, and psychophysiological processes in individuals with MDD?

Study 2: How are depressive symptoms, rumination, drinking motives, and negative alcohol-related consequences connected?

A brief summary of each study is provided below. The respective publications can be found in Chapter 2.

1.5.1 The Influence of Alcohol on Rumination and Metacognitions in Major Depressive Disorder

The comorbidity between MDD and AUD is highly prevalent, but the reasons for this association are not well understood yet. Rumination can activate metacognitive beliefs, which in turn contribute to the development and maintenance of rumination and depressed mood. Negative metacognitions may in turn lead to other dysfunctional coping strategies, such as alcohol use. This study examined whether the consumption of alcohol reduced metacognitions, rumination, and other disorder-specific processes in a group of individuals suffering from MDD. This "beneficial" effect of alcohol on these processes could then potentially provide an explanation for the association between MDD and AUD.

Methods: The experiment consisted of three randomized conditions: Alcohol (experimental condition, n = 22), placebo (n = 22), orange juice (control condition, n = 21). While participants ruminated or relaxed over a period of time, the effect of alcohol, placebo, or no alcohol (orange juice) consumption on (meta-)cognitions, depressive symptoms, and psychophysiological variables was examined.

Results: Voluntary rumination increased depressive symptoms, facial muscle tension, and heart rate, but had no effect on metacognitions or heart rate variability. Alcohol consumption had no effect on metacognitions, rumination, sadness, self-reported tension, or psychophysiological measures.

Conclusions: No evidence was found that alcohol consumption influenced disorder-specific processes in MDD. However, rumination had a negative effect on several depression-specific processes, although it did not activate (negative) metacognitions.

1.5.2 Linking Depression With Negative Alcohol-Related Consequences: A Structural Equation Model Among a Clinically Depressed Population

Results of previous studies suggest that rumination acts as a mediating mechanism between depression, drinking motives (drinking to cope), and negative alcohol-related consequences. In the course of this study, the associations between depression, rumination, drinking motives, alcohol consumption, and alcohol-related problems were examined for the first time in a clinically depressed population ($N = 209$).

Methods: Using structural equation modeling, two models of varying complexity were tested, whose specifications were based on the results of previously evaluated models on nonclinical samples: Depressive symptoms → rumination → drinking motives → alcohol consumption and alcohol-related problems.

Results: Our complex model showed a significant positive effect between depressive symptoms and rumination. In addition, drinking motives (enhancement and coping) led to increased negative alcohol-related consequences. In our simplified model, pronounced depressive symptoms were associated with both increased rumination and increased alcohol-related problems. Rumination was in turn associated with drinking motives, which in turn promoted alcohol consumption and alcohol-related problems. However, rumination itself was also negatively associated with alcohol-related consequences, i.e., a decrease in rumination was associated with an increase in consumption as well as alcohol-related problems.

Conclusions: In a clinically depressed sample, an association was found between depressive symptoms and negative alcohol-related consequences. This association appeared to be partially explained by drinking motives and rumination. However, the function of rumination, partly because of the direction of its association with alcohol-related consequences, has not yet been clearly elucidated.

1.6 Conclusion and Implications

The aim of this dissertation was to gain a better understanding of the mechanisms underlying the high comorbidity of AUD and MDD. First, it can be stated that RNT in the form of rumination appears to be relevant as a mechanism for understanding this association, although it cannot fully explain it – at least in this work.

Rumination is associated with a number of different processes and behaviors. It increases muscle tension and heart rate (study 1), enhances negative affect such as sadness (studies 1 and 2), and influences the decision to consume alcohol to regulate the aversive emotional experience (depression → rumination → drinking motives → consumption; study 2).

At the same time, rumination can be understood as an independent form of (short-term dysfunctional) coping (Papageorgiou & Wells, 2003; cf. Section 1.4.1: MCM), which is why the use of further coping strategies, such as alcohol consumption, may no longer be necessary. Should rumination be "successful" in this function, further consumption as well as the development of a comorbid disorder with AUD is rather unlikely. However, if rumination is accompanied by an intensification of negative affect and physical symptoms, it can be assumed, both in terms of the SMH (cf. Section 1.4.2) and the ECM (cf. Section 1.4.3), that alcohol is used to reduce the aversive emotional experience, making further consumption more likely and also increasing the risk of developing AUD.

In this context, a closer look at drinking motives should be considered. In study 2, drinking motives were assessed by the Drinking Motives Questionnaire (Kuntsche & Kuntsche, 2009). The wording of the items refers to emotional rather than cognitive content, such as "I drank alcohol because it helped me when I was down or irritable.". Although emotional content is involved, the association between depressive symptoms and drinking motives is not significant. Drinking motives correlate exclusively with rumination, alcohol consumption, and alcohol-related problems. It is possible that emotional activation is intensified by rumination, which could then in turn motivate to reduce / change this emotional state by consuming alcohol. Ultimately, an emotional state would then be managed after all, rather than recurrent negative thoughts. The results of a study by Simons et al. (2016) support this possible interpretation. Here, rumination was shown to increase depressive symptoms and alcohol consumption was shown to decrease depression. The authors assume that rumination counteracted the

"positive", dampening effect of alcohol, requiring even more consumption to achieve the same effects. In short, participants drank to regulate their feelings, but these were intensified by repetitive thoughts.

By influencing emotions, alcohol consumption also has an indirect effect on rumination, i.e., via a reduction in negative mood, a reduction in ruminative thinking occurs (Mollaahmetoglu et al., 2021). Thus, the consumption of alcohol may function as a maladaptive coping strategy of rumination-induced emotional states (Selby et al., 2016; Jungmann et al., 2016; Mollaahmetoglu et al., 2021; cf. Section 1.4.3: ECM).

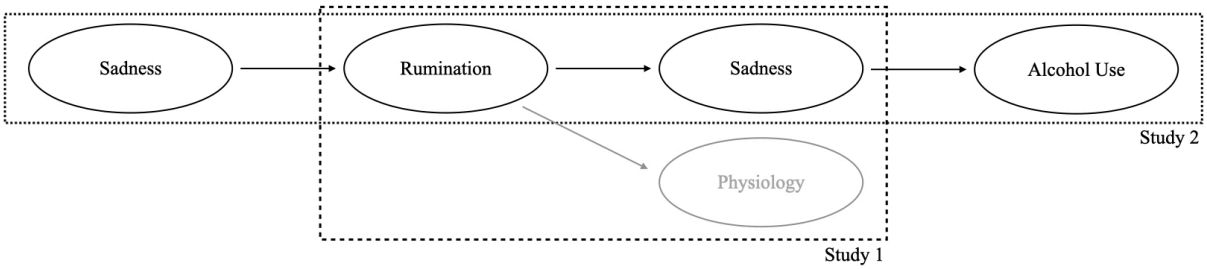
1.6.1 Intensifying Interaction of Cognition and Emotion as Underlying Mechanism of Major Depressive Disorder and Alcohol Use Disorder?

The following may be important in understanding the comorbidity of MDD and AUD: Rumination is present in MDD and, whether helpful in the short term or not, is a dysfunctional cognitive process that leads to the maintenance and reinforcement of depression in the long term. Although the MCM emphasizes the importance of cognitive processes, depressive experience is defined holistically at the levels of behavior, thoughts, and emotions (Wells, 2011). Consequently, the use of other strategies (i.e., alcohol consumption) in dealing with depressive symptomatology could also represent a form of self-medication within the model. Therefore, a conclusion might be to use a combination of the MCM and the SMH to explain comorbidity and not to consider them separately. This would also imply drawing on two mechanisms for understanding the link between MDD and AUD, i.e., both (repetitive) negative cognitions and (aversively experienced) emotions. An interaction of both, in terms of a dysfunctional intensification of cognitions and emotions (and / or bodily processes), could ultimately promote the use of alcohol as a coping strategy for aversive experiences (see Figure 1).

The idea of an intensifying interaction and the resulting use of impulsive behaviors has already been taken up in the ECM and applied to different mental disorders, such as BPD (Selby et al., 2009) or OCD (Jungmann et al., 2016). The study results of this dissertation suggest that the assumptions of this model can also be transferred to a depressive population.

Figure 1

Interactive Model of Emotions and Cognitions in MDD and AUD



Note. Aversively experienced emotions, such as depressed mood or sadness, are accompanied by negative cognitions, such as increased rumination. Rumination, in turn, intensifies negative emotions and can also adversely affect physical processes. The intensified negative emotional state favors the use of dysfunctional coping strategies, such as the consumption of alcohol.

The dashed boxes indicate how the results of studies 1 and 2 could be placed in the model.

1.6.2 Metacognitions in the Context of Depression und Alcohol Use (Disorder)

As feasible and straightforward as this model appears to be for explaining the comorbidity of MDD and AUD, it fails to do justice to part of the study findings as well as part of the theoretical assumptions of the MCM. Although the model seizes on cognitive processes to explain the maintenance and reinforcement of depressive symptomatology, it limits itself to rumination and neglects the presence / activation of metacognitive assumptions. As a consequence, the central mechanism considered by the MCM to be crucial for the exacerbation of depressive symptomatology is missing. At the same time, this aspect was deliberately excluded from the modeling because it was not possible to activate metacognitions about rumination in the course of the experimental investigation (cf. Chapter 2: Study 1, results).

This fact raises several important follow-up questions and at the same time provides inspiration for further research approaches, which will be discussed in the following. First, it raises the question to what extent rumination can actually activate metacognitions in individuals with MDD (see Section 1.6.3), and if so, which metacognitive beliefs (positive vs. negative vs. both) are present and to what

degree (see the following paragraph). This is followed by the final, still unresolved question of the potential influence of alcohol consumption on these beliefs (see Section 1.6.4). Although study 1 did not succeed in activating metacognitions through induced rumination and in sufficiently clarifying these very questions, the novelty of the research approach should nevertheless be emphasized (see Chapter 2: Study 1, discussion). Thus, these questions require further clarification in future studies.

The failed activation of metacognitions in individuals with MDD does not seem entirely surprising, as previous studies on this topic have also failed to provide conclusive results. Even in the first studies on the MCM (Papageorgiou & Wells, 2003), it proved difficult to determine a clear model structure. On the one hand, a good model fit was obtained in a sample with individuals who were clinically depressed (Papageorgiou & Wells, 2003, study 1), i.e., the statistical data were consistent with the theoretical assumptions of the MCM (Papageorgiou & Wells, 2004). On the other hand, during the same study, evidence was found that the model structure differed between clinical and nonclinical samples (Papageorgiou & Wells, 2003, study 2; Papageorgiou & Wells, 2004). Subsequent nonclinical studies have confirmed the assumptions of the MCM (e.g., Roelofs et al., 2007; Solem et al., 2016), whereas activation of metacognitions failed in a depressed sample (Roelofs et al., 2010). In a longitudinal study of university students, the assumptions of the MCM could also be confirmed. The presence of positive metacognitions predicted the extent of rumination at a later point in time, which in turn made negative mood more likely. Moreover, an indirect effect was found between positive metacognitions and depressive symptoms via rumination (Weber & Exner, 2013). Negative beliefs about rumination also appear to be relevant over time in predicting depressive symptoms (Papageorgiou & Wells, 2009). In turn, the results of a meta-analytic review suggest that although negative metacognitions are present in all of the compared psychopathologies, they are weaker in MDD than in other disorders, such as GAD, OCD, or eating disorders (Sun et al., 2017).

These conflicting findings on metacognitions in depression suggest that future studies should further investigate the relationship between positive and negative metacognitions, rumination, and depressive symptomatology, in both clinical and nonclinical studies and preferably in a longitudinal design.

1.6.3 Repetitive Negative Thinking in the Context of Depression and Alcohol Use (Disorder)

The results of a systematic review by Devynck and colleagues (2019) suggest that all forms of RNT (i.e., depressive rumination, ruminative brooding, ruminative reflection, worry, and abstract-analytic thinking) predict alcohol use in individuals with AUD. However, because of the small number of studies, gender differences, and inconclusive results with respect to nonclinical samples, the authors also emphasize the need for further research to draw firm conclusions about the association of RNT and alcohol use. In addition, they suggest less focus on subtypes of RNT and rather assume a single process in a transdiagnostic understanding. More generally, these findings emphasize the importance of RNT as an underlying mechanism for linking psychopathology and alcohol use, and may also provide specific clues to the comorbidity of MDD and AUD. For example, to test these indications in the context of the present work, it could have been investigated whether there are also gender differences in rumination as well as in alcohol use and alcohol-related problems. Furthermore, it could have been considered in advance whether only one subtype of rumination should be investigated or whether the assessment of different subtypes of RNT and / or a transdiagnostic process of RNT could have been interesting for the research question.

Also, when considering the results from study 2, it is important to examine the extent to which the association between depressive symptomatology and negative alcohol-related consequences can be explained at all by RNT, more specifically rumination.

Since, despite the significant positive direct effects and corresponding linkage of the variables with each other (for details see Chapter 2: Study 2), the function of rumination still cannot be clearly clarified. For one thing, rumination has a significant positive effect on drinking motives, which in turn are associated with negative alcohol-related consequences; for another, significant negative effects of rumination on alcohol consumption as well as alcohol-related problems are found.

These effects can be interpreted in different ways: On the one hand, it could mean that the form of ruminative thinking assessed in the study is not associated with an increase in negative alcohol-related consequences, but possibly a different subtype of RNT (e.g., reflective pondering; Heggeness et al., 2021). On the other hand, as already mentioned, if the negative effect of rumination on alcohol-related variables does indeed exist, this could mean that individuals with MDD whose rumination is more

pronounced are less likely to use alcohol as a strategy to deal with repetitive thoughts. Thus, it could be further hypothesized that depressed mood motivates individuals to cope with their negative feelings either by ruminating or, alternatively, by consuming alcohol. The former could either be understood as coping in terms of emotional avoidance, i.e., feelings are suppressed through rumination (Liverant et al., 2011). Alternatively, it could suggest that positive metacognitions about the "usefulness" of rumination are present (Watkins & Moulds, 2005), whose activation promotes further rumination as a coping mechanism.

One way to find out whether rumination activates metacognitions in MDD could be to assess them after successful rumination induction, e.g., with self-reports such as the Positive and Negative Beliefs about Rumination Scales (Papageorgiou & Wells, 2001a; Papageorgiou & Wells, 2001b). In order to ultimately determine which coping strategy is preferred, it should be investigated whether rumination continues in the presence of metacognitions or whether other dysfunctional behaviors are used instead to cope with depressive symptomatology, such as the consumption of alcohol. This, in turn, could be helpful in understanding comorbidity: Those who "only" ruminate in a depressive state are unlikely to develop a problematic use of alcohol, but those who use consumption for regular coping with rumination-induced emotional states are more likely to develop comorbid AUD. Gender differences and population differences may also be present here, which should be taken into account in future studies.

1.6.4 Alcohol Effects and Use in Depression

Despite some promising associations between rumination, depression, and alcohol consumption (especially in study 2), the present work could not clearly clarify how the consumption of alcohol affects disorder-specific processes in depression.

One possible reason for this could be that alcohol was not used as a preferred coping strategy in either sample and was thus consumed infrequently. This is because both studies showed that some of the participants consumed little to no alcohol. In contrast, Bravo and colleagues (2018) specifically recruited students for their research who regularly consumed (a larger amount of) alcohol. As a result, only a limited number of significant associations may have been found between depression, rumination, drinking motives, and negative alcohol-related consequences. This may also explain the partially

contradictory results with regard to rumination. Since the participants in study 2 consumed less alcohol and less frequently, they probably expected fewer positive effects of alcohol (drinking motives) and used it correspondingly less frequently to cope with aversive states – or, as already mentioned, preferred other strategies, which, however, were not examined in the course of the study. A further investigation with a larger sample of individuals suffering from depression, who ruminate and have a high to problematic alcohol consumption would be desirable to further test the hypotheses or model assumptions of study 2. In this context, it might also be interesting to assess metacognitive beliefs about rumination, i.e., they could be included as another factor in the model equation to examine how they influence, e.g., rumination or promote consumption (depression → rumination / metacognitions → drinking motives → alcohol use).

Another reason could be that the amount of alcohol administered in study 1 was not appropriate for the sample. There may be a difference in the effects of alcohol in MDD compared to anxiety disorders. For example, the AAM (Steele & Josephs, 1988) suggests that "alcohol myopia" (alcohol's impairment of perception and thought) increases with the amount of alcohol (Steele & Josephs, 1990). In the case of social anxiety disorder, the (larger) amount administered in study 1 achieved the desired effect, i.e., a "positive" influence on anxiety symptoms (Gerlach et al. 2006; Stevens et al., 2008). In contrast, such an effect has been demonstrated with a low to moderate amount of alcohol in individuals with negative mood (Mollaahmetoglu et al., 2021). Consequently, it needs to be considered to what extent the models on the effect of alcohol on anxiety (cf. Section 1.2) can be applied to affective disorders at all. After empirical testing of the model assumptions regarding the effects of alcohol in the context of MDD, a revision of these might be indicated.

To the best of my knowledge, there have been only two empirical studies to date (Mollaahmetoglu et al., 2021; Gawron et al., submitted) that have examined the direct effects of alcohol on cognitive processes such as ruminative thinking. And there is only *one* study to date that has examined the direct effects of alcohol on metacognitions (Gawron et al., submitted). Due to the small number of studies, both the power and generalizability of the results are limited. Further replication of the study design is needed to improve these parameters and the understanding of the underlying mechanisms of MDD and AUD.

1.6.5 Conclusion With Wilhelm Busch

The studies conducted in the context of this dissertation were able to partially answer the question of the function of cognitive processes in explaining the comorbidity of MDD and AUD. The presence of negative cognitions in the form of ruminative thoughts seems to intensify unpleasant emotions / states and thus to favor the use of alcohol as a coping strategy for this aversive experience. In study 1, there is a significant increase in depression scores (self-reports) after the rumination induction; in study 2, there is a significant positive correlation between depressive symptoms and rumination as well as between rumination and drinking motives (involving the manipulation of emotional states with the aid of alcohol consumption).

Thus, these findings are best interpreted in terms of the ECM: Due to the reciprocal feedback and synergistic effects of emotions and cognitions, exacerbation occurs. To break the vicious circle, an intensive form of dysfunctional emotion regulation is often used, such as the consumption of alcohol (Selby et al., 2016). An important implication for the treatment of MDD and AUD comorbidity may lie in the teaching of appropriate emotion regulation strategies. The results of a recently published study by Sorid and colleagues (2021) also point in this direction. The consumption of alcohol is intended to reduce emotional discomfort that appears to be exacerbated by negative thoughts. Conversely, appropriate regulation of emotions could reduce the discomfort caused by rumination and thus the tendency to use alcohol for coping (Sorid et al., 2021).

Unfortunately, the present work was unable to clarify the function of metacognitions in the context of MDD and AUD and the impact of acute alcohol consumption on depression-specific processes. As noted above, further replication of the study design is needed to draw firm conclusions about the effects of alcohol on these processes and the function of metacognitions.

Finally, what can be said in conclusion about Wilhelm Busch's statement on worries and liqueur? In my opinion, the following can be stated: "Those who have worries also have liquor, but also have unpleasant feelings.". In fact, findings from previous studies in the field of anxiety disorders suggest that individuals with GAD suffer from worries associated with negative feelings (e.g., Andor et al., 2008; Llera & Newman, 2014) and consume alcohol to reduce these same anxiety symptoms (e.g.,

Bolton et al., 2006; Grant et al., 2005; Robinson et al., 2009). In addition, so-called worry-reduction alcohol expectancies may be present, i.e., the belief that consuming alcohol will reduce worry (Smith & Book, 2010), which encourage further consumption. However, in the field of GAD, there is also a lack of studies examining the direct effects of alcohol on disorder-specific processes. For the most part, findings on comorbidity are based on symptom assessment via questionnaires and self-reports. It is difficult to draw conclusions about the underlying mechanisms of this high comorbidity (Burns & Teesson, 2002) on the basis of the available studies. For impulsive behaviors in comorbid mental disorders, including individuals with GAD (Selby et al., 2016), and OCD, the assumptions of the ECM could be successfully replicated (Jungmann et al., 2016). Potentially, this could also be a promising approach for GAD to explain the association of negative feelings, worry, and alcohol use. After all, worry and rumination are both forms of RNT that are associated with worsening affectivity.

Considering the results of both studies and including the assumptions on the interaction of emotions and cognitions in MDD (cf. Sections 1.4.3 and 1.6.1), a very similar statement can be made in the context of depression, rumination, and alcohol: "Those who ruminate also have liquor, but also have unpleasant feelings.".

However, similar to GAD, further research on the underlying mechanisms is needed before the variation of Wilhelm Busch's quote can be unequivocally agreed with, and that is research that considers (meta-)cognitive and emotional processes in MDD differentiated as well as in interaction, and examining the direct effect of alcohol on these same processes.

1.7 References

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2. Original Publications

Study 1

Gawron, L., Pohl, A., & Gerlach, A. L. (in revision). The Influence of Alcohol on Rumination and Metacognitions in Major Depressive Disorder. *Clinical Psychology in Europe*.

Study 2

Gawron, L., & Gerlach, A. L. (in revision). Linking Depression With Negative Alcohol-Related Consequences: A Structural Equation Model Among a Clinically Depressed Population. *Substance Use & Misuse*.

Individual Author Contributions

My own contribution to the studies conducted in this work is as follows (based on the CRediT author statement): Conceptualization, methodology, formal analysis, data curation, writing - original draft, writing – review and editing, and visualization.

Dr. Anna Pohl and Prof. Alexander Gerlach also contributed with conceptualization, methodology, discussing data analysis, and writing – review and editing. Prof. Alexander Gerlach also contributed with project administration and supervision.

The Influence of Alcohol on Rumination and Metacognitions in Major Depressive Disorder

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This article is the manuscript version.

The article was revised and resubmitted to *Clinical Psychology in Europe* for a re-review on June 20, 2022.

Abstract

Background and Objectives: Comorbidity between major depressive disorder (MDD) and alcohol use disorder (AUD) is highly prevalent but reasons for this association are unclear. Rumination may activate metacognitive beliefs that contribute to the development and maintenance of rumination and depression. Negative metacognitions can further lead to other dysfunctional coping strategies (i.e., consumption of alcohol). We examined whether alcohol reduces metacognitions, rumination and other disorder-specific processes in a group of individuals suffering from MDD.

Methods: In an experiment with three randomized conditions we investigated whether the consumption of alcohol, placebo or no alcohol (orange juice) affects (meta-)cognitions, depressive symptoms and/or psychophysiological variables while participants ruminate.

Results: Voluntary rumination increased self-reported sadness, tension and rumination, tensed facial muscles and increased heart rate, but did not affect metacognitions and heart rate variability. The consumption of alcohol did not influence rumination, metacognitions, depressive or psychophysiological measures.

Limitations: We recruited a depressed population but excluded pathological alcohol use due to ethical considerations.

Conclusions: We found no evidence that alcohol consumption affects rumination, metacognitions and other disorder-specific processes in MDD. However, rumination had a negative effect on various depression-specific processes, although it did not activate (negative) metacognitions.

Keywords: major depressive disorder, rumination, metacognitions, alcohol consumption, self-medication, alcohol use disorder.

The Influence of Alcohol on Rumination and Metacognitions in Major Depressive Disorder

Rumination, the repetitive negative thinking about past events, possible causes and consequences of negative emotions (Nolen-Hoeksema, 1991), contributes to the development (e.g., Huffziger et al., 2009) as well as maintenance and severity of depressive episodes (e.g., Nolen-Hoeksema & Harrell, 2002; Nolen-Hoeksema et al., 2008). Moreover, rumination has negative effects on somatic health, as illustrated by a number of psychophysiological changes such as decreased heart rate variability (HRV; Woody et al., 2014; Ottaviani et al., 2015), increased heart rates (HR; Ottaviani et al., 2016) and changes in muscular tension, e.g., in the corrugator EMG (Teasdale & Rezin, 1978).

According to the metacognitive model of rumination and depression (MCM), rumination is maintained by metacognitions reflecting on this type of perseverative thinking (Papageorgiou & Wells, 2003). Negative thoughts or other triggers initially activate positive metacognitive beliefs about the usefulness of rumination (e.g., "In order to understand my feelings of depression, I need to ruminate about my problems."), and motivate further rumination. However, rumination prevents effective problem solving and intensifies negative affect. As a result, negative metacognitive beliefs emerge regarding the uncontrollability and harmfulness of rumination and its social consequences (e.g., "I cannot stop myself from ruminating."; "People will reject me if I ruminate."), thereby increasing the accessibility of negative and threatening information (e.g., negative thoughts or emotions), and thus exacerbating and maintaining depressive symptoms as well as promoting further rumination (Papageorgiou & Wells, 2004).

Both, clinical (e.g., Papageorgiou & Wells, 2003) and nonclinical studies (e.g., Solem et al., 2016; Roelofs et al., 2007; Weber & Exner, 2013; Yilmaz et al., 2015) have shown that metacognitive beliefs about rumination are significant for the onset (Faissner et al., 2018; Papageorgiou & Wells, 2009) and maintenance (e.g., Huntley & Fisher, 2016; Solem et al., 2016) of depressive states / depression.

Negative metacognitions may also promote the use of dysfunctional behavioral strategies, such as the use of alcohol, to control or avoid recurrent negative thoughts. In the long term, however, these strategies may maintain negative metacognitions (cf. metacognitive model of generalized anxiety disorder; Wells, 2005; Wells, 2011). Although the MCM of generalized anxiety disorder focuses on worry and meta-worry, we assume that the assumptions regarding the use of other coping strategies can

also be applied to the MCM for depression and rumination. Thus, we take a step beyond the original model by postulating that alcohol use functions as a cross-model coping strategy that can reduce rumination (see, e.g., Mollaahmetoglu et al., 2021) and possibly negative metacognitions (in the short term), making these processes seem less uncontrollable and threatening.

According to the appraisal disruption model, alcohol can disrupt the appraisal of threatening information (i.e., cognitions; Sayette, 1993). More specifically, alcohol may interfere with the initial perception of stressful information by preventing negative memories and associated stressful concepts from being activated. Moreover, cognitive abstraction capacity is supposed to be reduced by alcohol (Sayette, 1933), which may also impede perseverative thinking and related metacognitions. Finally, when intoxication precedes a stressor, it can buffer the stress by attenuating appraisal, thereby protecting the person drinking from fully experiencing the stressor (Sayette, 2017). Applied to the context here, negative metacognitions can also be defined as a type of threatening information whose appraisal can be attenuated by alcohol consumption. Furthermore, intoxication could prevent concepts associated with negative metacognitions, such as ruminative thoughts, from being activated, possibly leading to relief in terms of less threatening rumination or generally less aversive emotional states. Since this dysfunctional coping strategy is only helpful in the short term, alcohol may be consumed repeatedly in order to feel a facilitating effect (negative reinforcement). This could then lead to the development of a problematic drinking pattern or an alcohol use disorder (AUD).

Empirical evidence suggests that these negative metacognitions are in particular associated with problematic alcohol use (Spada & Wells, 2005; Spada et al., 2007). The higher the levels of maladaptive metacognitions are, the more likely alcohol is consumed in response to unpleasant aversive states (Moneta, 2011). In line with this, rumination is associated with alcohol consumption (Caselli et al., 2008; Ciesla et al., 2011; Devynck et al., 2019) and with increased alcohol-related problems (Nolen-Hoeksema & Harrell, 2002; Willem et al., 2011). In a group of individuals with risky consumption, the direct effects of alcohol on rumination and mood were examined and it was found that alcohol reduced rumination directly and also indirectly by changing mood (Mollaahmetoglu et al., 2021).

Apart from the study of Mollaahmetoglu et al. (2021), most empirical evidence for the association of rumination, depressed mood and alcohol use (disorder) is correlative (e.g., Bravo et al.,

2018; Heggeness et al., 2019; Thornton et al., 2012). Moreover, these relationships have mostly been examined in analogue samples (e.g., Bravo et al., 2017; Bravo et al., 2018; Ciesla et al., 2011), and metacognitions have been assessed as a *trait* variable (e.g., Papageorgiou & Wells, 2003; Faissner et al., 2018; Papageorgiou & Wells, 2009; Weber & Exner, 2013). However, it has been argued that mimicking typical problematic situations may also provoke the presence of *state*-dependent metacognitive beliefs about perseverative cognitions as well as their consequences, especially in clinical populations (Andor et al., 2008). Consistent with this, negative metacognitions following worrying, so negative state metacognitions, were more pronounced in patients with generalized anxiety disorder compared with control participants when they received feedback that indicated arousal while being asked to relax (Andor et al., 2008).

In light of previous findings, we believe it is important to examine the direct effects of alcohol consumption on perseverative cognitions, such as rumination, and negative metacognitions in an experimental setting: indeed, if it is shown that people with depression can alter cognitive processes with the help of alcohol, this could provide a significant clue to the mechanisms underlying the high comorbidity of major depressive disorder (MDD) and AUD (e.g., Boschloo et al., 2011; Brière et al., 2014; Lynskey, 1998), with, for example, odds ratios between 2.0 (Kessler et al., 1997) and 3.8 (Grant & Harford, 1995).

Namely, depression-related cognitive / ruminative and metacognitive processes that appear uncontrollable and threatening may erroneously appear controllable and less threatening after alcohol consumption, which may be relieving in the short term, thus promoting further consumption and the development of AUD.

To our knowledge, no study has yet examined the direct effects of alcohol on negative (meta)cognitions and depression in a clinically depressed sample. Our aim was therefore to examine these effects on rumination and metacognition in MDD. We specifically focused on (negative) state metacognitions (cf. Andor et al., 2008). The negative appraisal of these state metacognitions may be interrupted by alcohol consumption and consequently appear less threatening (cf. Sayette, 1993). For a holistic understanding of the effects of alcohol on disorder-specific processes, we also wanted to investigate the influence of alcohol on emotional states and psychophysiology (heart rate, heart rate

variability, muscle tension). According to some studies, alcohol can lead to an increase in heart rate (Weise et al., 1986), a reduction in HRV (Koskinen et al., 1994), and a decrease in muscle tension (Stockwell et al., 1982).

Our hypotheses were as follows: given that rumination has an unfavorable impact on negative affect and psychophysiology (see, e.g., Ottaviani et al., 2015; Ottaviani et al., 2016; Simons et al., 2016), we hypothesized that (H1) induced rumination has a negative effect on sadness, tension, and on the extent of rumination itself, as well as on psychophysiological processes. We also hypothesized that (H2) alcohol consumption reduces rumination, (H3) alcohol consumption reduces negative (state) metacognitions about rumination that, according to the MCM rumination and depression, should be triggered by induced rumination, and (H4) alcohol consumption reduces negative emotions such as sadness and experienced muscle tension intensified by rumination. Finally, in addition to rumination, alcohol consumption may also affect psychophysiology, although the direction of the effect in MDD is still unclear. We assumed an increase in HR and a decrease in HRV and muscle tension in individuals with depression (H5).

Method

2.1 Recruitment

Participants were recruited online (e.g., via facebook), with publicly distributed leaflets, posters and at the outpatient treatment centre for psychotherapy. All participants received a compensation of 8.50 euros per hour and were offered counselling. Exclusion criteria were current or past substance use disorder or AUD, complete abstinence of alcohol, GAD, current use of psychoactive medication, liver damage, current or past psychotic episodes, and pregnancy. GAD was excluded to ensure that the main problem with repetitive negative content was rumination and not worrying. All participants signed an informed consent. The ethics committee of the German Psychological Association approved this study (SS 042017).

2.2 Participants

Sixty-five participants (46 women) diagnosed with current MDD using a structured clinical interview (see 2.3) completed the study. Thirty-nine participants (40.5 %) were diagnosed with additional comorbid disorders. Twenty-seven suffered from anxiety disorders (41.5 %), ten from posttraumatic stress disorder (15.4 %), three from obsessive compulsive disorder (4.6 %), three from an eating disorder (4.6 %), and five from somatic symptom disorders (7.7 %). Sociodemographic data is presented in Table 1. Further characteristics can be found in Appendix Table A1. Power analyses according to G*Power 3 (Faul et al., 2007) indicated a required sample size of at least 54 participants, expecting a medium effect size f = .25 for the analysis of a repeated measures ANOVA (within-between interaction) at an alpha level of .05 and 95% power (cf. Andor et al., 2008; Stevens et al., 2017).

Table 1

Demographic Data of all Participants Separated by Group

Variable	AC (n = 22)	PC (n = 22)	OC (n = 21)
Mean Age (*SD*)	33.6 (11.5)	30.2 (11.8)	30.7 (12.9)
Sex, *n* (*%*)			
Women	15 (68.2)	16 (72.7)	15 (71.4)
Men	7 (31.8)	6 (27.3)	6 (28.6)
Education, *n* (*%*)			
O level	4 (18.2)	16 (72.7)	1 (4.8)
Specialized A level	1 (4.5)	3 (13.6)	6 (23.8)
A level	15 (68.2)	3 (13.6)	15 (71.4)
Still attending school	2 (9.1)	-	-
Family status, *n* (*%*)			
Unmarried	17 (77.3)	20 (90.9)	17 (81.0)
Married – living together	1 (4.5)	1 (4.5)	3 (14.3)
Divorced	3 (13.6)	1 (4.5)	-
registered civil partners	-	-	1 (4.8)
Widowed	1 (4.5)	-	-
Treatment, *n* (*%*)			
Current outpatient treatment	4 (18.2)	2 (9.1)	4 (19.0)

Past outpatient treatment	16 (72.7)	15 68.2)	12 (57.1)
Past psychiatric inpatient treatment	7 (31.8)	7 (31.8)	5 (23.9)
Past antidepressant medication	5 (22.6)	7 (31.8)	8 (38.1)

Note. AC = alcohol condition, PC = placebo condition, OC = control / orange juice condition. O level = ordinary level high school certificate, A level = advanced level high school certificate. The groups did not differ significantly.

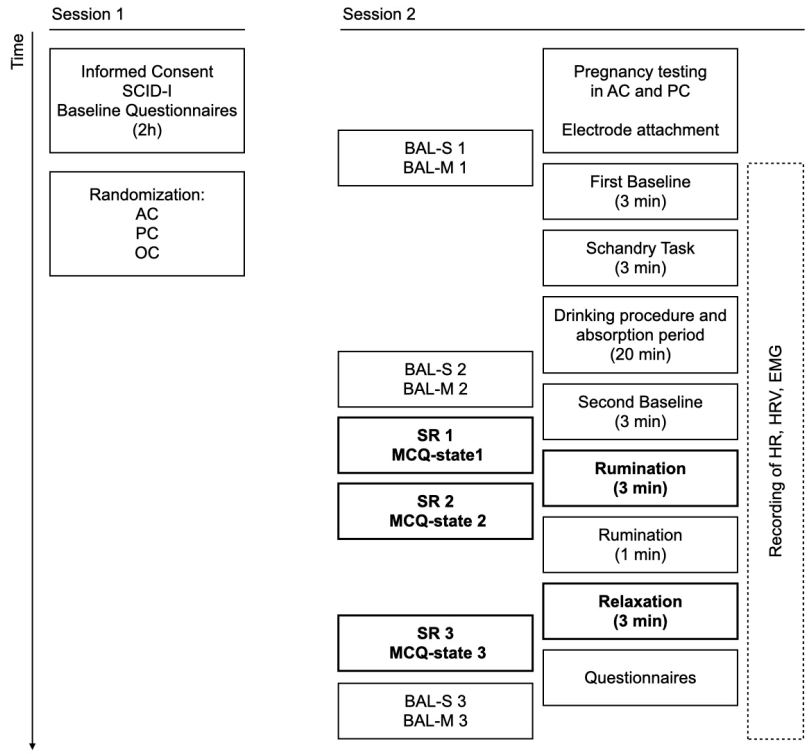
2.3 Procedure (Figure 1)

Participants were telephone screened and then received information about the experiment. They had to agree to participate in the study irrespective of whether they would receive alcohol or not. Participants with depressive symptoms were invited for a 2 h diagnostic session using the German version of the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th version (SCID-I; Wittchen et al., 1997). A trained clinical psychologist conducted the interviews. Participants with MDD then completed several questionnaires (see 2.5.1) and were invited for a laboratory session. At this point, participants were fully randomized to three conditions (see 2.4). At the beginning of the laboratory session, electrodes for physiological measurement were attached and participants estimated their blood alcohol level (BAL). Then the BAL was measured. A three-minute resting period (first baseline) and an additional three-minute task (Schandry, 1981) followed, which will not be reported here. Then, a drinking phase of 15 minutes drinking and a five-minute break allowing for absorption of the alcohol followed. Participants again estimated their BAL and it was also measured. After a second three-minute resting period (second baseline), participants estimated their level of rumination, sadness and tension, and completed the state metacognitions questionnaire (MCQ-state; Andor et al., 2008). The *rumination induction procedure* (a variant of the worry induction procedure; Borkovec & Inz, 1990) followed. Participants were asked to write down three topics they regularly ruminated about and were to choose the currently most troubling one. They were then instructed to ruminate about this topic "like they normally did". After ruminating for three minutes (rumination episode), participants reported their rumination, sadness and tension again and completed the MCQ-state. They were instructed to ruminate for another minute and then asked to relax for three minutes

(relaxation episode). Following the relaxation, participants completed the self-reports and MCQ-state a third time as well as the WBSI, TCQ-R and CAS-I (see 2.5.2). In the end, they estimated their BAL and the BAL was measured one last time. After the experiment, participants were debriefed.

Figure 1

Procedure



Note. Timing and overview of the two sessions. The blood alcohol level (BAL) was measured at the beginning, after the phase of drinking and at the end of the experiment. Self-reports (SR) and MCQ-state were assessed at three time points: before rumination, after rumination and after relaxation. An overview of all baseline questionnaires and all questionnaires used during the experiment can be found in section 2.5. AC = alcohol condition (n = 22); PC = placebo condition (n = 22); OC = control condition / orange juice (n = 21). BAL-S = participants' estimated BAL before each measurement of BAL; BAL-M = measured breath alcohol level. SR = self-reports, i.e., estimated levels of sadness, rumination, and tension. MCQ-state = two subscales of the Metacognitions Questionnaire, German version. HR = heart rate; HRV = heart rate variability; EMG = facial electromyography.

2.4 Drinking Procedure

All participants were asked to eat a light meal, specified in a handout, four hours prior to the experiment and to forego food and drinks containing caffeine from then on. They were requested to abstain from alcohol 24 hours prior to the experiment. Participants in the control condition (OC) were told that they would receive orange juice. Participants in the alcohol (AC) and placebo condition (PC) were both given the information that they would receive alcohol and that they would have to be picked up or wait until their BAL decreased below 0.3 ‰. All participants were tested at 4:00 pm. Female participants in the AC or PC were pregnancy tested. None of the participants tested positive. Finally, height and weight were measured.

Participants in the AC consumed a drink of 1:2 vodka and orange juice. Following a modified version of the Widmark formula, participant's sex, weight, height and age was used to estimate the necessary amount of alcohol to reach a blood alcohol level of about 0.6 ‰ (Gerlach et al, 2006). The nonalcoholic beverage in the OC and PC was orange juice in comparable drinking quantity. In the PC, immediately before serving the beverages, a few milliliters of vodka were dropped on the orange juice and applied along the rims using a pipette (Stevens et al., 2014). Participants received three glasses with equal amounts of chilled beverage, each to be finished within five minutes. After drinking, participants waited five minutes.

Breath alcohol concentration was assessed by breathalyzer with an accuracy of +/- 0.03 mg/L (Dräger, Alcotest, 7410 plus). In the PC, the first measurement used a standard breathalyzer to ensure a BAL of zero. Then, a rigged breathalyzer with identical built was used giving a false feedback of 0.6 ‰ and then 0.7 ‰ BAL.

2.5 Measurements

2.5.1 Baseline Questionnaires

Alcohol Use Disorder Identification Test (AUDIT; Dybek et al., 2006). The AUDIT is a brief screening scale developed by the World Health Organization (WHO) for early detection of problematic drinking. The original as well as the German version includes 10 questions regarding alcohol consumption, dependency symptoms and alcohol related problems. For each question, one of five

statements related to alcohol use in the past year can be selected on a 5-point Likert-type scale ranging from 0 ("never") to 4 (e.g., "daily or almost daily"). Cronbach's α = .76.

Simplified Beck Depression Inventory (BDI-S*;* Schmitt et al., 2003). The BDI-S assesses current depressive symptoms with 20 items on a 6-point Likert-type scale ranging from 0 ("never") to 5 ("almost always"), for example, "I feel sad.". Cronbach's α = .87.

Metacognitions Questionnaire 30 (MCQ-30; Arndt et al., 2011). The German version of the MCQ-30 (a shortened version of the original Metacognitions Questionnaire; Cartwright-Hatton & Wells, 1997) is used to assess thoughts and beliefs (metacognitions) about worry. The questionnaire consists of five subscales (positive worry beliefs, beliefs about uncontrollability and danger, metacognitive efficiency, general negative beliefs, cognitive self-consciousness) assessed by 30 items (e.g., "Not being able to control my thoughts is a sign of weakness."). Items/statements can be rated on 5-point Likert-type scales ranging from 1 ("not agree") to 4 ("agree very much"). Cronbach's α = .84.

Penn State Worry Questionnaire (PSWQ; Stöber, 1995). The German version of the PSWQ is a 16-item questionnaire assessing intensity, excessiveness and uncontrollability of worry (e.g., "I worry all the time.") on a 5-point Likert-type scale ranging from 1 ("not at all typical of me") to 5 ("very typical of me"). Cronbach's α = .89.

Response Styles Questionnaire (RSQ; Kühner et al., 2007). The German version of the RSQ assesses people's cognitive and behavioral strategies to cope with depressed mood with 32 items on 4-point Likert-type scales ranging from 1 ("almost never") to 4 ("almost always"). The RSQ consists of the subscales rumination with 21 items (e.g., "When I am sad, I think about how sad I feel.") and distraction with 11 items (e.g., "When I am sad, I go to my favorite place to get my mind off my feelings."). Cronbach's α = .69.

2.5.2 Questionnaires Used During the Experiment

Assessment of State Metacognitions (MCQ-state; Andor et al., 2008). Since state-dependent changes in metacognitions can be assessed using the MCQ (cf. Andor et al., 2008), two subscales of the MCQ-30 (beliefs about uncontrollability and danger, general negative beliefs) were adapted to the experiential situation. An example is "My ruminating could make me go mad." Cronbach's α = .97.

Rumination Score (RS). The levels of sadness, tension and rumination were assessed with rating scales ranging from zero (“absolutely not”) to 100 (“extremely so”) and then averaged. Cronbach’s α = .83.

White Bear Suppression Inventory (WBSI; Fehm et al., 2000). The German version of the WBSI measures thought suppression with 15 items (e.g., “There are things I prefer not to think about.”) on a 5-point Likert-type scale ranging from 1 (“strongly disagree”) to 5 (“strongly agree”). Cronbach’s α = .85.

Thought Control Questionnaire (TCQ; Fehm & Hoyer, 2004). The German version of the TCQ is a 30-item self-report measure assessing rumination, intrusive and unwanted thoughts. Items can be rated on 4-point Likert-type scales ranging from 1 (“never”) to 4 (“almost always”). Cronbach’s α = .67.

Cognitive Attentional Syndrome-Inventory (CAS-I; Wells, 2011). The German version of the CAS-I assesses maladaptive coping strategies (e.g., worrying, avoidance, use of alcohol/drugs) for dealing with negative thoughts, and negative and positive metacognitive beliefs. In total, the CAS-I consists of four questions. The first three questions are answered using a scale from 0 ("not at all") to 8 ("all the time") and refer to how much dealing with problems or worries about problems was done in the past week and how it was dealt with. The fourth question refers to positive and negative metacognitions, answered using a scale from 0 ("I do not believe in this belief at all.") to 100 ("I am absolutely convinced that this belief is true."). Cronbach’s α = .75.

2.6 Psychophysiological Data Recording, Sampling and Analysis

Psychophysiological data (heart rate, respiration and facial muscle tension) were recorded using the Varioport (Becker Meditec, Karlsruhe, Germany). ECG was recorded at 512 Hz sample rate from three electrodes. The active electrodes were placed on the lowest left rib and on the right collarbone. Ground was affixed to the left collarbone. Respiration was assessed with a respiratory belt (128 Hz sample rate). Facial electromyography (EMG) was recorded in mV at 256 Hz sample rate over the *corrugator supercilii* on the left side of the face with two electrodes (TIGA-MED, Germany Ltd.). The EMG signal was preprocessed using an infinite impulse response high pass filter at 10 Hz. It was notch

filtered at 50 Hz with a width of 3 Hz and rectified and smoothed using a two-step low pass filter with eight point moving average. For HRV, the root mean square successive differences (RMSSD) was calculated (cf. Task Force, 1996; Bertsch et al., 2012). Mean values were computed for each experimental 3-minute episode (baseline 2, rumination, relaxation).

2.7 Data Analysis

Group differences concerning sociodemographic characteristics and self-reported BAL were tested using an ANOVA[1] and Bonferroni-corrected post-hoc tests. Group differences concerning psychopathological variables (questionnaires) were analyzed using a MANOVA. A Pearson correlation was performed between problematic alcohol consumption (AUDIT) and the level of alcohol as a coping strategy (CAS-I). To test our hypotheses, we conducted several repeated measures ANOVAs[2] with Bonferroni-corrected post-hoc tests. Each ANOVA was analyzed by group (alcohol, placebo, orange juice).

To test H1 (rumination increases sadness, tension, rumination, and worsens psychophysiology) the measurement time points of all variables from "second baseline" to "rumination" were examined. H2 (alcohol reduces rumination) and H4 (alcohol reduces sadness and tension intensified by rumination) were tested in one model: for this, RS over time were analyzed. To test H3 (alcohol reduces negative state metacognitions), metacognitions ratings (MCQ-state) were analyzed. To test H5 (alcohol influences psychophysiology), EMG, HR and HRV over time were analyzed. In case sphericity was violated, the Greenhouse–Geisser adjustment was used.

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[1] Initial exploratory analyses revealed a few outliers. However, there was no relevant change in the pattern of results when including vs. excluding outliers. Thus, results from the complete data set are reported. Deviations from the original data set are indicated in the data analysis (e.g., MCQ-state ratings).

[2] The assumption of normality (ANOVA) or the equality of variances (repeated measures ANOVAs) was not met. Since the F-Test is relatively robust for violation of assumption (Finch, 2005; Tabachnick & Fidell, 2007), the ANOVA and the repeated measures ANOVAs were nevertheless conducted and results reported. Because the number of subjects varied across the variables, no repeated measures MANOVA could be calculated for the self-reports or for the biodata. Instead, several repeated measures ANOVAs were conducted with Bonferroni-corrected post-hoc tests.

Results

3.1 Manipulation Check

3.1.1 Coping Strategies

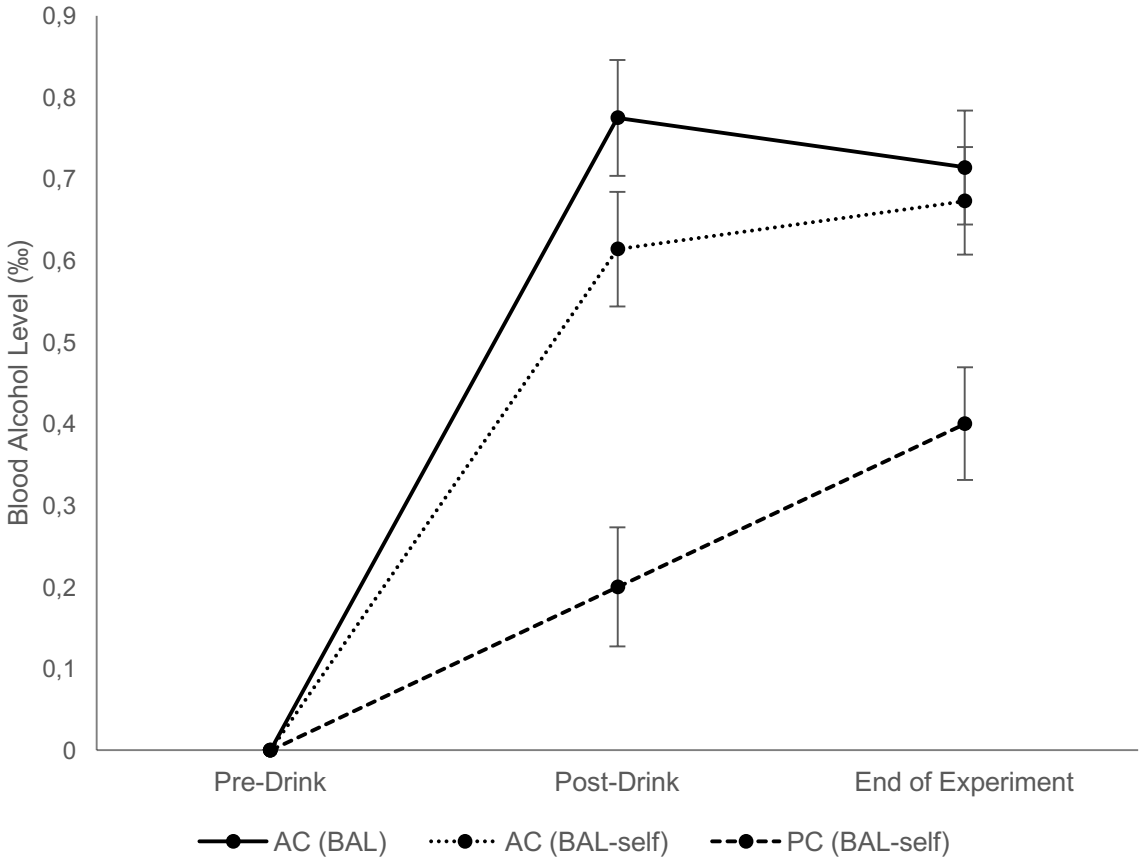
The correlation of AUDIT and CAS-I was significant (r = .46, p < .001). The most frequent used coping strategy was “to control emotions” (M = 6.0, SD = 2.0), followed by “the attempt not to think about anything” (M = 5.2, SD = 2.2), “to avoid situations” (M = 2.8, SD = 2.5), “to control symptoms” (M = 4.2, SD = 2.2), “to seek reassurance” (M = 3.5, SD = 2.3). The least used strategy was “to consume alcohol or drugs” (M = 2.5, SD = 2.0).

3.1.2 Self-Reported Alcohol Level and Measured Blood Alcohol Level

Compared to baseline, in both AC and PC self-estimated alcohol levels (in ‰) were higher after drinking (M_{AC} = 0.6, SD = 0.2, M_{PC} = 0.2, SD = 0.1) and after finishing the experiment (M_{AC} = 0.7, SD = 0.2, M_{PC} = 0.4, SD = 0.2). The manipulation in the PC can be considered successful: 20 of 22 participants believed that they had been given alcohol. Two subjects (PCs) were excluded because their self-estimated BAL was 0.0 ‰ at all measurement points and then assigned to the control condition for subsequent analyses. In the AC, the measured BAL was 0.8 ‰ (SD = 0.2) after the drinking period and 0.7 ‰ (SD = 0.2) at the end of the experiment (see Figure 2).

Figure 2

Blood Alcohol Level



Note. Measured and estimated BAL. Data points represent values before and after the drinking procedure and at the end of the experiment; error bars depict 95% CI. AC = alcohol condition (n = 22); PC = placebo condition (n = 20). BAL = measured breath alcohol level in AC; BAL-self = participants' estimated BAL before each measurement of BAL. Control condition is not included.

3.1.3 Rumination Induction Procedure (H1, H5)

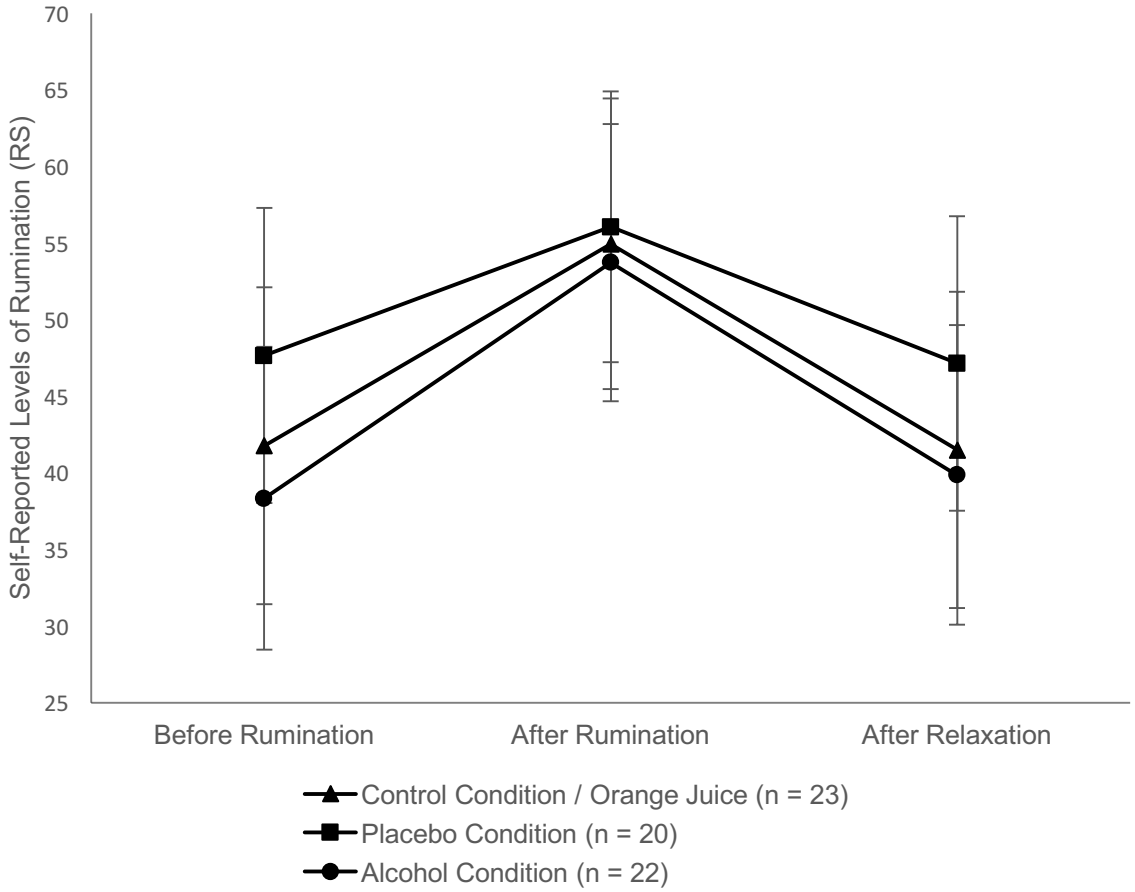
Self-report: An initial univariate ANOVA revealed no significant group differences in the self-reports (F(2, 62) = .86, p = .427) and MCQ-state-ratings[3] (F(2, 42) = .26, p = .772) before rumination induction. After rumination, RS were significantly higher (see Table 2 and Figure 3a), whereas MCQ-state-ratings did not change (see Table 2).

[3] Since the first measuring time of the MCQ-ratings was subsequently integrated into the experiment, the repeated measures ANOVA was conducted with only n = 45.

Psychophysiological measures: An initial univariate ANOVA[4] revealed no significant group differences in HR ($F(2, 61) = .37$, $p = .690$), HRV ($F(2, 61) = 1.46$, $p = .240$) or EMG ($F(2, 58) = .36$, $p = .702$) before rumination. HR and EMG increased significantly with rumination. Regarding HRV, there was no significant change in RMSSD during rumination or relaxation (see Table 2 and Figures 3b, c).

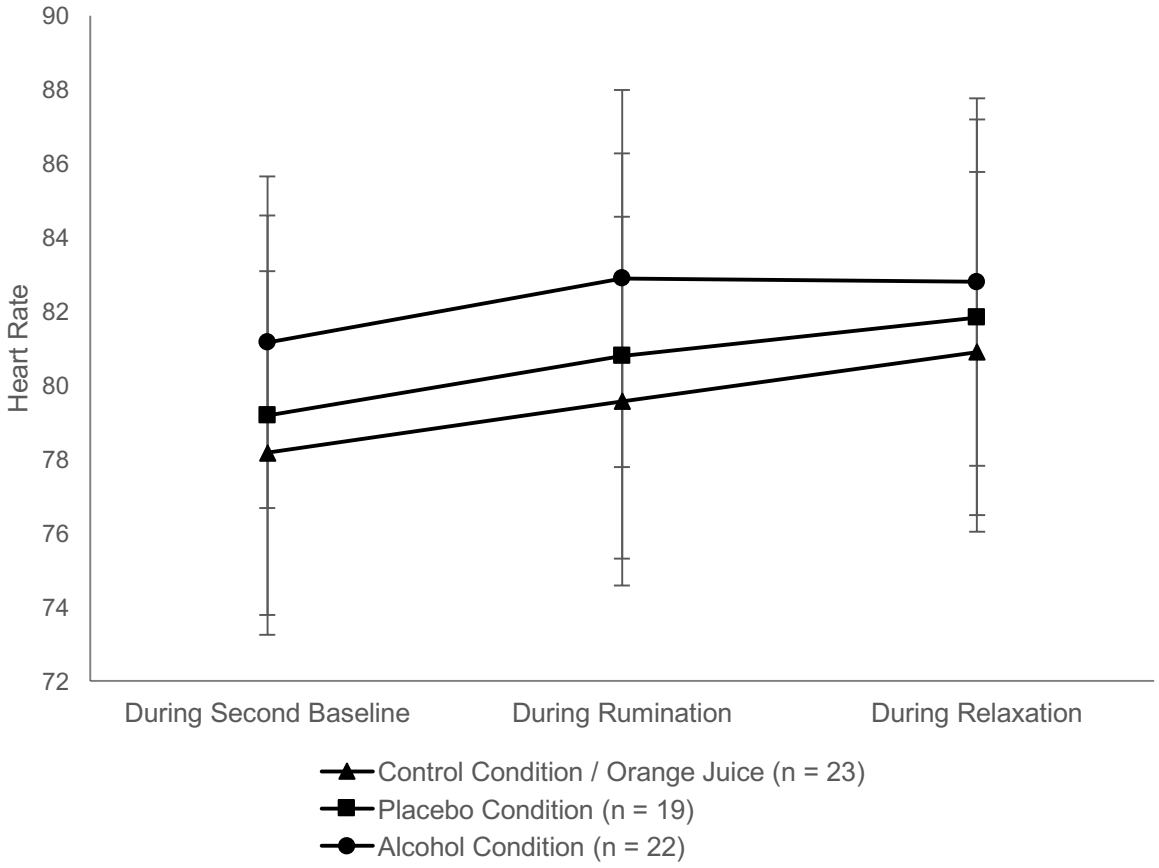
Figure 3

Results Over Time Separated by Group: a) Rumination Score, b) Heart Rate, c) EMG

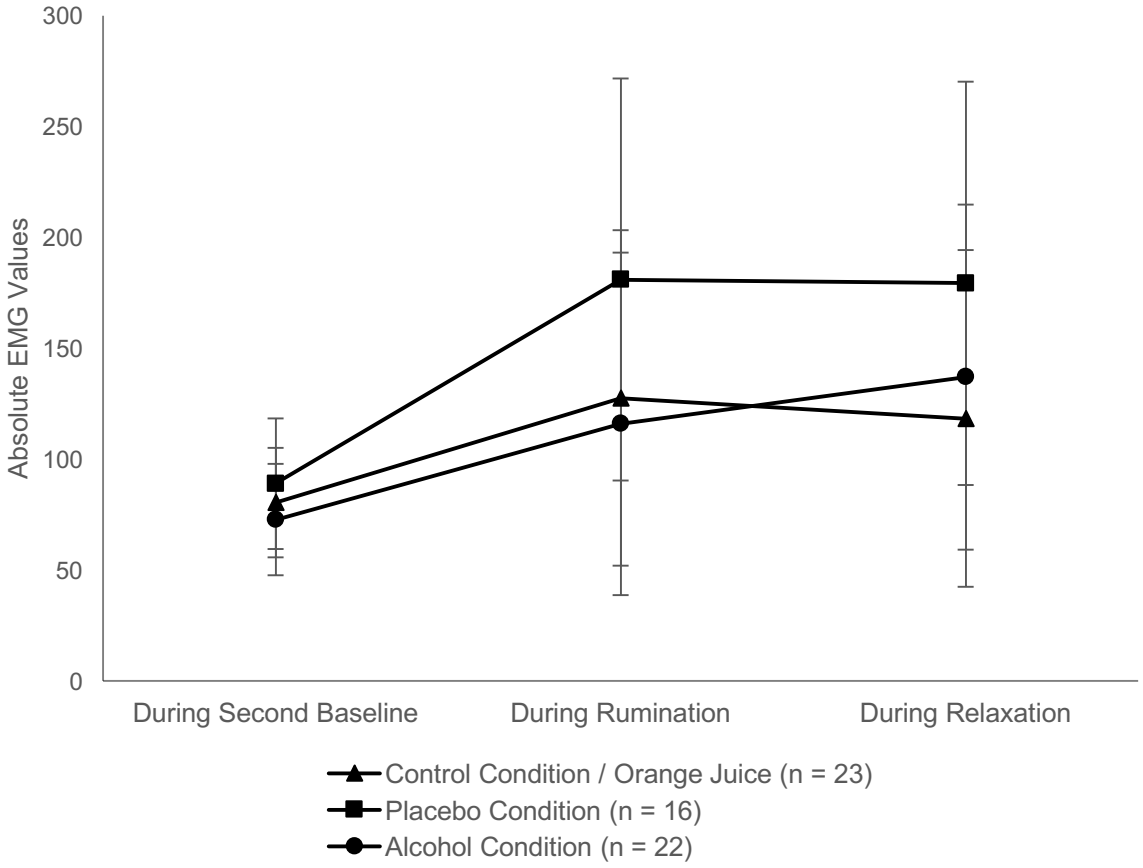


Note. a) Rumination score (RS) over time separated by group. Data points represent the mean values before, after the rumination induction and after relaxation; error bars depict 95% CI. Estimates of depression (sadness, rumination, tension) were rated on a scale from 0 to 100.

[4] Regarding EMG, three subjects (PC) were excluded from further analyses because they were identified as outliers in at least four of five relevant time intervals. Another subject was excluded because the recording of biodata failed. See Table A3 for an overview of all participants per condition.



Note. b) Heart rate over time separated by group. Data points represent the mean values of three time intervals: during second baseline, rumination and relaxation; error bars depict 95% CI.



Note. c) EMG over time separated by group. Data points represent the mean values of three time intervals: during second baseline, rumination and relaxation; error bars depict 95% CI.

3.2 Repeated Measures ANOVAs (H2-H5)

ANOVAs revealed a significant main effect of time for RS ($F(1.52, 94.38) = 16.45$, $p = < .001$, $\eta_p = .21$), HR ($F(2, 122) = 14.12$, $p = < .001$, $\eta_p = .19$), and EMG ($F(2, 116) = 5.41$, $p = .006$, $\eta_p = .09$). From "second baseline" (T1) to "rumination episode" (T2) there was a significant increase in RS, HR and EMG. From T2 to "relaxation" (T3) there was a significant decrease in RS (see Figure 3a). From T2 to T3 there was no significant change in HR and EMG (see Figures 3b, c). No significant effect for group and no interaction effect for time × group was found in any variable (see Table 2 and A2 for all significant and nonsignificant effects, Table A3 for mean values).

Table 2

Repeated Measures ANOVAs Results

Effects	Measures	*F*	*df*	*p*	η^2_{part}
Time	RS	16.45	1.52, 94.38	< .001	.21
	MCQ-state	.32	2, 84	*ns*	.01
	HR	14.12	2, 122	< .001	.19
	HRV	1.57	1.50, 91.26	*ns*	.03
	EMG	5.41	2, 116	.006	.09
Group	RS	.57	2, 62	*ns*	.02
	MCQ-state	.31	2, 42	*ns*	.02
	HR	.32	2, 61	*ns*	.01
	HRV	1.08	2, 61	*ns*	.03
	EMG	.74	2, 58	*ns*	.03
Time × Group	RS	.38	3.05, 94.38	*ns*	.01
	MCQ-state	.15	4, 84	*ns*	.01
	HR	.56	4, 122	*ns*	.02
	HRV	1.69	2.99, 91.26	*ns*	.05
	EMG	.37	4, 116	*ns*	.01

Note. RS = Rumination score. MCQ-state = state version of the Metacognitions Questionnaire. HR = Heart rate, beats per minute (bpm). HRV = Heart rate variability, RMSSD. EMG = facial electromyography, absolute EMG values (uV). *ns* = nonsignificant.

Discussion

We directly studied if alcohol affects disorder-specific processes in individuals suffering from MDD. In particular, we wanted to understand whether and how alcohol affects rumination and metacognitions about rumination. In addition, we were interested in determining the extent to which rumination negatively affects other disorder-specific processes, such as intensifying sadness, and in terms of the MCM, is associated with negative metacognitions.

The rumination induction was successful: self-reported levels for rumination, tension, and sadness increased, as did HR and muscle tension. However, HRV and state metacognitions did not change. We were able to successfully establish a placebo condition (i.e., induce the belief of having consumed alcohol) in almost all participants. In addition, participants who reported higher alcohol

consumption were more likely to report using alcohol for coping. Yet, alcohol use was the least reported coping strategy for aversive states in our sample.

In contrast to our first hypothesis, we did not find an increase in negative metacognitions after rumination. It is possible that the type and implementation of the rumination induction procedure influenced our result. The procedure was originally developed for the induction of worry (Borkovec & Inz, 1990). Given, however, that worry and rumination are often transdiagnostically conceptualized as two forms of perseverative negative cognitions (McEvoy et al., 2013; Watkins, 2008), the procedure for inducing rumination should have been sufficient to induce metacognitions about rumination, just as inducing worry was sufficient to induce metacognitions about worry (Andor et al., 2008). Yet, Andor and colleagues (2008) studied individuals with generalized anxiety disorder whose negative (trait) metacognitions are more pronounced than in individuals with MDD (Sun et al., 2017). Participants in the Andor study received false arousal feedback during the relaxation phase, making it more likely to experience worry and relaxation as uncontrollable. In other words, it was directly suggested to the participants in this study that their condition was not controllable. It is likely that both the type of disorder and the type of manipulation influenced the intensification of metacognitions. One approach for future studies might be to examine both state and trait metacognitions in relation to rumination and depressive symptomatology and to directly induce a sense of uncontrollability to participants.

However, another consideration against the background of the MCM is conceivable. In the Andor study as well as in our experiment, negative metacognitions were measured via two subscales of the MCQ-30. These scales assess the uncontrollability and danger of worry (reworded to rumination in our study), but not negative metacognitions with regard to social consequences of rumination, which, in terms of the MCM, are also typical for the perpetuation of depression. After successful induction, we did not find more pronounced metacognitions in terms of uncontrollability and danger, but we might have found changes in terms of metacognitions related to the social consequences of rumination. One way to measure both types of negative metacognitive beliefs about rumination would have been to include the Negative Beliefs About Rumination Scale (NBRS; Papageorgiou & Wells, 2001) in our experiment. In this way, we would have been even closer to the original model and the respective measurement methods (cf. Papageorgiou & Wells, 2003).

Also, it is possible that negative metacognitions do not need to be reinforced in certain situations to have a negative effect on perseverative thinking. It may be sufficient that these assumptions exist in the first place to maintain depressive states (e.g., Papageorgiou & Wells, 2009). If negative (state) metacognitions cannot be intensified even with the use of other experimental procedures, we nonetheless consider it advisable to reassess the long-term effects of negative metacognitions on the development of depression in a vulnerable group of participants. This would allow to further investigate the extent to which negative metacognitions are causal in the development and maintenance of depression.

Contrary to our hypotheses (H2-H5), we could neither show that alcohol consumption reduced experienced rumination, sadness, or muscle tension, nor that it reduced metacognitions about rumination. The three groups did not differ regarding their RS nor in their ratings of metacognitions. There were also no differences between groups in terms of psychophysiological data. Alcohol did not change the negative effect of rumination on psychophysiological variables, nor did it increase physiological reactivity. Thus, surprisingly, we did not find evidence of alcohol effects on any process potentially relevant for the formation and maintenance of depression.

Conger (1956) suggested that alcohol may be used because it reduces muscular tension. However, alcohol did not reduce muscle tension nor change other measures of arousal. Whereas Conger's notion can be found in many textbooks, the pharmacological (stress-reducing) effects of alcohol have only rarely been illustrated. According to a review of studies in social anxiety, for example, alcohol expectancy effects were more likely to be responsible for a reduction of aversive states such as anxiety than alcohol's pharmacological properties (Battista et al., 2010). Thus, people who consume alcohol and expect a stress and tension-relieving effect, may experience such an effect regardless of pharmacological effects. Such positive alcohol expectancies should have been evident in both the AC and PC in comparison to the OC. Yet, in both self-reports and EMG the numerically highest values (indicating distress) were found in the PC. Since Conger's hypothesis refers mainly to anxiety-provoking situations, it should be noted that these assumptions may not apply in situations where other emotions, such as depression or sadness, are prominent. Or possibly, individuals might assume that alcohol is a helpful strategy, but notice when drinking that the strategy proves unsuccessful.

Significant positive correlations have previously been found between metacognitions and alcohol consumption as well as between anxiety, depression and alcohol consumption (Spada & Wells, 2005; Spada et al., 2007). The consumption of alcohol can therefore be regarded as a conscious strategy for dealing with aversive states (Quitkin et al., 1972). In the AC, however, alcohol consumption did not result in feeling less emotionally distressed than in the other two groups. Thus, we found no evidence that alcohol consumption reduces rumination, metacognitions, or sadness in depressed individuals. Interestingly, our findings are consistent with those of a recent study on social anxiety, in which alcohol consumption had no attenuating effect on negative (post-event) rumination (Hagen et al., 2020), although consumption reduced (social) anxiety (Stevens et al., 2014). Mollaahmetoglu and colleagues (2021) found that alcohol had an effect on ruminative thoughts and mood at a low dose (about 0.2 mg/L) but not at a high dose (about 0.6 mg/L). It is therefore worth considering whether the desirable effects of alcohol in our study would also have been observed if we had used a lower dose. A promising approach for further studies could be to examine alcohol effects on rumination, metacognitions and depressive mood depending on the dose administered. Also, the question arises to what extent the model assumptions on alcohol effects (for a review see Sayette, 2017), which were investigated in the context of anxiety (disorders), can be transferred to other disorders and / or other emotional states, such as depression. It should be noted, however, that according to Sayette's model (1993), appraisal disruption is expected only at higher levels of alcohol (i.e., at an amount of alcohol sufficient to cause cognitive impairment), and that we based our hypotheses on this model. Nonetheless, if alcohol may not be the usual choice for our participants, e.g., to control unpleasant cognitions, metacognitions or emotions, it simply may not have this effect in the present sample due to selection bias. In order to ensure that alcohol is a preferred coping strategy, it would have been necessary to pre-screen, for example with the CAS-I (Wells, 2011).

Regarding the effects of alcohol consumption on (meta-)cognitive, emotional, and psychophysiological processes and its function in coping with depression, it can be stated that further research is needed to investigate these relationships in more detail.

Limitations

One limitation of our study relates to the sample size, due to which only moderate effects could be detected. However, compared to the results of other clinical studies dealing with the effects of alcohol (e.g., in social anxiety disorder), the sample size we recruited can be considered sufficient (cf. Stevens et al., 2017). Another limitation relates to our procedure, which can be considered rather exploratory, as the direct effect of alcohol on metacognitions has not been investigated before and therefore we could only assume that alcohol consumption may prevent negative state metacognitions from being appraised as threatening (cf. Sayette, 1993). In addition, it would have been helpful to assess the expected effects of alcohol on rumination or metacognitions before or during the experiment to include trait and actual expectancies of alcoholic effects into statistical analyses. A final limitation relates to the assessment of rumination. Here, for example, a rumination-related questionnaire with better psychometric properties may have been more suitable, (e.g., the Ruminative Response Scale-short form; Treynor et al., 2003).

Conclusions

To our knowledge, this was the first study to directly examine the association between AUD and by assessing the effects of alcohol on rumination and metacognitions in a sample of clinically depressed individuals. We did not find that alcohol reduced rumination, metacognitions about rumination, or depressive symptoms. Thus, our results suggest that previous models of alcohol effects from the domain of anxiety disorders (e.g., Sayette, 1993) may not be easily transferable to the domain of depressive disorders.

Consistent with the findings of previous studies (see, e.g., Nolen-Hoeksema et al., 2008; Ottaviani et al., 2016), we were able to show that rumination negatively affects disorder-specific processes in MDD. Surprisingly, rumination did not elicit negative metacognitions about the uncontrollability and danger of rumination, although this would have been expected in terms of the MCM.

However, due to the novelty of this research approach, further studies are needed to further test existing models / theories linking depression and alcohol. For example, this could include studies with individuals who drink more and use alcohol more regularly for coping, with a modified paradigm, i.e.,

with other forms of rumination induction, with manipulated arousal feedback, or with a lower dose of administered alcohol, and / or with other (physiological) measurement methods.

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Appendix

Table A1

Descriptive Statistics for all Questionnaires With Multivariate Analysis

Questionnaires	AC *n* = 22 *M* (*SD*)	PC *n* = 22 *M* (*SD*)	OC *n* = 21 *M* (*SD*)	F	η^2_{part}
BDI-S	52.8 (16.8)	54.4 (12.2)	60.5 (14.7)	1.64	.05
PSWQ	59.6 (10.7)	60.4 (9.2)	62.9 (11.4)	.56	.02
RSQ	76.1 (8.5)	78.3 (8.0)	76.00 (8.6)	.54	.02
MCQ-30	69.4 (13.0)	69.1 (10.4)	71.3 (12.1)	.23	.01
AUDIT	6.2 (3.9)	7.7 (4.4)	7.7 (4.8)	.79	.03
WBSI[a]	58.5 (12.4)	58.0 (7.4)	59.0 (10.7)	.06	.00
TCQ	65.2 (7.5)	66.1 (8.2)	67.4 (8.1)	.44	.01
CAS-I	6.00 (1.5)	5.7 (1.2)	5.6 (1.3)	.56	.02

Note. AC = alcohol condition, PC = placebo condition, OC = control / orange juice condition. BDI-S = Beck Depression Inventory Simplified, German version. PSWQ = Penn State Worry Questionnaire, German version. RSQ = Response Styles Questionnaire, German version. MCQ-30 = Metacognitions Questionnaire, German version. AUDIT = Alcohol Use Disorder Identification Test, German version. WBSI = White Bear Suppression Inventory, German version. TCQ = Thought Control Questionnaire, German version. CAS-I = Cognitive Attentional Syndrome Questionnaire, German version. *df* = 2, 62. The groups did not differ significantly.

[a] For WBSI, TCQ and CAS-I, the number of participants per condition changed to PC (*n* = 20) and OC (*n* = 23).

Table A2

Bonferroni-Corrected Post-Hoc Tests for Repeated Measures ANOVAs

Time point	Measures	*MD*	*SE*	*p*	η^2_{part}
T1 vs. T2	RS	-12.32	2.30	< .001	.32
	MCQ-state	-.49	.68	*ns*	.01
	HR	-1.58	.45	.003	.17
	HRV	1.27	3.31	*ns*	.00
	EMG	-60.77	23.01	.032	.11
T2 vs. T3	RS	12.08	1.89	< .001	.40
	MCQ-state	.29	.50	*ns*	.01
	HR	-.76	.42	*ns*	.05
	HRV	3.61	3.17	*ns*	.02
	EMG	-3.43	20.79	*ns*	.00

Note. MD = Mean difference. T1 = time point 1: before rumination / second baseline, T2 = time point 2: after rumination, T3 = time point 3: after relaxation. RS = rumination score. MCQ-state = two subscales of the Metacognitions Questionnaire, German version. HR = heart rate, beats per minute (bpm). HRV = heart rate variability, RMSSD. EMG = facial electromyography, absolute EMG values (uV). *ns* = nonsignificant.

Table A3

Rumination Score, Metacognitions Ratings, Heart Rate, Heart Rate Variability and Facial Electromyography Over Time

	Measures	T1 *M* (*SD*)	T2 *M* (*SD*)	T3 *M* (*SD*)
AC (*n* = 22)	RS	38.3 (25.2)	53.7 (24.4)	39.9 (27.4)
(*n* = 15)[a]	MCQ-state	36.2 (12.2)	36.7 (10.7)	36.9 (12.8)
	HR	81.2 (11.7)	82.9 (12.5)	82.8 (12.7)
	HRV	26.4 (14.0)	26.6 (13.1)	27.6 (14.3)
	EMG	72.7 (38.6)	116.0 (70.2)	136.7 (135.6)
PC (*n* = 20)	RS	47.7 (23.3)	56.1 (20.6)	47.2 (18.3)
(*n* = 15)	MCQ-state	34.4 (6.4)	34.6 (9.2)	34.3 (9.3)
(*n* = 19)	HR	79.2 (14.0)	80.8 (13.6)	81.8 (12.5)
(*n* = 19)	HRV	38.3 (26.6)	29.8 (17.7)	29.8 (28.7)
(*n* = 16)	EMG	89.0 (60.9)	181.0 (330.1)	179.4 (290.9)
OC (*n* = 23)	RS	41.8 (20.9)	55.0 (18.4)	41.5 (22.3)
(*n* = 15)	MCQ-state	36.8 (8.9)	37.5 (9.8)	36.8 (10.1)
	HR	78.2 (10.2)	79.6 (9.8)	80.9 (9.8)
	HRV	45.5 (56.4)	50.1 (87.6)	38.3 (53.1)
	EMG	80.4 (71.9)	127.5 (87.5)	118.3 (108.9)

Note. AC = alcohol condition, PC = placebo condition, OC = control / orange juice condition. T1 = time point 1: before rumination / second baseline, T2 = time point 2: after rumination, T3 = time point 3: after relaxation. RS = rumination score. MCQ-state = two subscales of the Metacognitions Questionnaire, German version. HR = heart rate, beats per minute (bpm). HRV = heart rate variability, RMSSD. EMG = facial electromyography, absolute EMG values (uV).

[a] A deviation from the original number of participants (AC: *n* = 22, PC: *n* = 20, OC: *n* = 23) is indicated, e.g. (*n* = 15).

Linking Depression With Negative Alcohol-Related Consequences:

A Structural Equation Model Among a Clinically Depressed Population

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This article is the manuscript version.

The article was revised and resubmitted to *Substance Use & Misuse* for a re-review on May 19, 2022.

Abstract

Background and Objectives: Previous research suggests that rumination acts as a mediating mechanism in the association between depression and drinking motives, particularly drinking to cope, as well as negative alcohol-related consequences. In this study, we tested the connections between depressive symptoms, rumination, drinking motives, alcohol consumption, and alcohol-related problems in a clinically depressed population ($N = 209$).

Methods: Structural equation modeling was used to test the models. Specifications were based on the results of a previously evaluated model in a sample of college students.

Results: The complex model showed a significant positive association between depressive symptoms and rumination. Drinking motives (enhancement and coping) were linked to more alcohol-related negative consequences. In a simplified model, pronounced depressive symptoms were associated with both increased ruminative thinking and more negative alcohol-related problems. Rumination was connected with stronger drinking motives (combined in one general factor), which were again associated with alcohol consumption and alcohol-related problems.

Limitations: The use of self-report measures to determine diagnostic validity.

Conclusions: In a clinically depressed sample, depressive symptoms were linked to increased negative alcohol-related consequences. This association was partially explained by rumination and drinking motives. However, rumination was less relevant than previous studies suggested.

Keywords: depression, rumination, drinking motives, alcohol use, alcohol-related problems

Linking Depression With Negative Alcohol-Related Consequences:

A Structural Equation Model Among a Clinically Depressed Population

Affective and substance use disorders, particularly alcohol use disorder, exhibit a high comorbidity (e.g., Boschloo et al., 2011; Brière et al., 2014; Grosshans & Mann, 2012; Lynskey, 1998). For example, 30.5% of individuals with an affective disorder also met criteria for alcohol dependence (in a Canadian survey; Spaner et al., 1994) and 32.5% of individuals with major depression had a lifetime diagnosis of alcohol dependence (U.S. National Longitudinal Alcohol Epidemiology Study; Grant & Harford, 1995). Furthermore, the presence of depression increases the risk of comorbid alcohol dependence (Kessler et al., 1997; Boschloo et al., 2011). However, the underlying mechanisms responsible for this high comorbidity have received little empirical investigation.

Motivational models of alcohol consumption (Cooper, 1994; Cox & Klinger, 1988) assume that drinking motives have a relevant influence on the decision to drink alcohol: among these are (1) social motives (external reward × positive reinforcement), i.e., drinking represents a way to socialize with others, (2) conformity motives (external reward × negative reinforcement), i.e., drinking serves to avoid negative social consequences, such as criticism, (3) enhancement motives (internal reward × positive reinforcement), i.e., drinking serves to increase physical or psychological well-being, and (4) coping motives (internal reward × negative reinforcement), i.e., drinking serves to avoid an unpleasant state. All these drinking motives are associated with more frequent use, larger amounts of drinking (Cho et al., 2019; Ham & Hope, 2003; Piasecki et al., 2014) as well as problematic use and alcohol-related problems (Cooper et al., 1995; Cho et al., 2019; Jester et al., 2015; Simons et al., 2017). Moreover, previous studies suggest that coping motives may also be linked to depression and may be a possible explanation for the relationship between depressive symptoms and negative alcohol-related consequences. Most importantly, students with coping motives tend to have more depressed mood and try to improve their mood with the help of alcohol (Stewart & Devine, 2000). Among students who drink alcohol, depressive symptoms have both a direct effect on alcohol-related problems and an indirect effect via drinking to cope motives (Gonzalez et al., 2011). In another student study, which examined coping-anxiety and coping-depression motives as well as daily alcohol consumption, higher coping scores were shown to predict daily mood-related alcohol consumption (Grant et al., 2009). Women who

exhibited depressive symptoms prior to starting college tend to drink to cope more and to experience more alcohol-related problems during college (Kenney et al., 2015). Moreover, individuals with depression who began drinking to cope at the beginning of a 10-year study period showed a stronger association between depressive symptoms and both alcohol use and drinking problems at the end of the survey (Holahan et al., 2003). Similarly, in individuals with high enhancement motives monthly negative affect and drinking frequency were correlated more strongly than in individuals with low enhancement motives (Armeli et al., 2010). Regarding conformity motives, Villarosa et al. (2018) found that students may drink both to improve their social status (i.e., conformity motives) and to reduce their depressiveness (i.e., coping motives). Finally, social motives have been mainly associated with generally increased alcohol consumption and not specifically with depression-related drinking (e.g., Cho et al. 2019). Nevertheless, these motives may be relevant for drinking behaviors of individuals with depression who often avoid social gatherings. This avoidance, in turn, may be a protective factor for alcohol abuse (cf. Peterson et al., 2021). In summary, whereas coping motives have most closely been linked to depression, other drinking motives may also be relevant when trying to better understand the connection between depression and alcohol use (disorder).

Rumination, repetitive negative thinking about past events and possible causes and consequences of negative emotions (Nolen-Hoeksema & Morrow, 1993), is a hallmark symptom of major depression and has been proposed as a central mechanism linking depressive symptoms to negative alcohol-related consequences (e.g., Clancy et al., 2016; Nolen-Hoeksema & Harrell, 2002). In fact, it even has been suggested that alcohol is rather used to break through repetitive negative thoughts than to directly change aversive emotional states (Ciesla et al., 2011). In recent studies employing structural equation modelling (SEM), depressive symptoms were indeed related to an increase in rumination, which in turn was associated with increased drinking (to cope) motives as well as more alcohol use and alcohol-related problems (Bravo et al., 2017; Bravo et al., 2018). However, these postulated associations between depression, rumination, drinking motives, and alcohol effects have only been examined in a nonclinical population (college students).

With this in mind, this study has three main goals: 1) to understand the role of depression as a predictor of negative alcohol-related consequences in a clinical context, 2) to replicate the findings of

Bravo and colleagues (Bravo et al., 2017; Bravo et al., 2018) in a sample of likely clinically depressed individuals, and 3) to examine the function of rumination in relation to negative alcohol outcomes[1].

Specifically, we aim to test the following hypotheses: (H1) depressive symptoms are associated with increased rumination, drinking motives, and more alcohol consumption as well as alcohol-related problems; (H2) rumination is linked to increased drinking motives and negative alcohol outcomes; (H3) drinking motives are associated with more negative alcohol outcomes.

Method

2.1 Participants and Procedure

Initial exploratory analyses of the total sample (N = 214) revealed five outliers, which were excluded from further analyses. The study thus comprised 209 participants (n = 156, 74.6% women) who were currently undergoing treatment (n = 197, 94.3%) or had received treatment for depression in the past, and the majority of them reported drinking alcohol at least occasionally in the last year, i.e., once a month or less frequently (n = 155, 74.2%).

Although not all of our participants reported currently drinking alcohol, nevertheless, the entire population of depressed individuals was included in the study and model calculations based on the following considerations: from an epidemiological perspective, and as mentioned in our introduction, depression is considered a risk factor for the development of an alcohol use disorder (see, e.g., (Abraham & Fava, 1999). We hypothesize that depressive symptoms promote negative alcohol-related outcomes (knowing that other pathways may also be relevant to understanding this comorbidity, e.g., that alcohol use disorder may be a risk factor for the development of depression or that common risk factors underlie the comorbidity). Specifically, we would like to understand the extent to which depression acts as a predictor of alcohol use and problems. However, previous studies examining depression as a risk factor for pathological alcohol use did not distinguish between depressed individuals with and without alcohol use, and therefore the entire sample should be examined. In a further step, we are interested in other

[1] "Negative alcohol-related consequences" and "negative alcohol outcomes" are used to describe the same term and include both alcohol use and alcohol-related problems.

mechanisms / predictors, such as rumination and drinking motives, which may also be relevant for understanding comorbidity. In summary, our focus is on the entire population of depressed individuals rather than, for example, exclusively on those individuals that already consume alcohol and may have specific drinking motives that further promote consumption. It should be noted, however, that the general effects did not change when we compared the results of the total population with those of the population including only participants who reported to drink alcohol. This finding also argues in favor of including the entire population of depressed individuals in our calculations.

Initially, participants were recruited exclusively from outpatient practices and in psychiatric clinics. Practices and clinics that offered treatment for individuals with depression were contacted and informed about the study. If the institutional boards allowed advertising, flyers with information about the study and access to the anonymized survey were handed to suitable participants by the professional staff. However, due to the COVID-19 pandemic, contacts with the relevant facilities were restricted, making further visits to the clinics impossible or difficult. For this reason, participants were additionally recruited online, for example through posts on depression-specific websites. See Table 1 for sociodemographic data.

Individuals that participated were included in a lottery of five 20-euro Amazon vouchers. Participation in the study was voluntary and anonymous. The procedure was in accordance with the ethical guidelines for human research and was approved by the ethics committee of the Faculty of Human Sciences of the University of Cologne (LGHF0037).

Table 1

Demographic Data of all Participants

Variables	
Mean Age (*SD*)	32.1 (11.5)
Sex, *n (%)*	
Women	156 (74.6)
Men	50 (23.9)
Diverse	3 (1.4)
Education, *n (%)*	
O level / Secondary	59 (28.2)
Specialized A level	39 (18.7)

A level	106 (50.7)
Still attending school	5 (2.4)
Family status, *n (%)*	
Unmarried	145 (69.4)
Married – living together	38 (18.2)
Divorced	22 (10.5)
Registered civil partners	3 (1.4)
Widowed	1 (0.5)
Treatment, *n (%)*	
Current outpatient treatment	146 (69.9)
Current psychiatric inpatient treatment	14 (6.7)
Current antidepressant medication	145 (69.4)
Past outpatient treatment	168 (80.4)
Past psychiatric inpatient treatment	123 (58.9)
Past antidepressant medication	153 (73.2)

Note. O level = ordinary level high school certificate, A level = advanced level high school certificate.

2.2 Measures

2.2.1 Depressive Symptoms

Center of Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The German version of the CES-D (Hautzinger & Bailer, 1993) is a 20-item self-report measure assessing depressive symptoms during the previous week on a 4-point Likert-type scale (0 = "rarely or not at all (less than 1 day)", 3 = "mostly, all the time (for 5 to 7 days)"). A cut-off point of 16 has been recommended for the CES-D (Radloff, 1977). Most of the 209 participants (n = 197, 94.3%) had a CES-D score above the cut-off. Although a few individuals scored below this point, we decided to include them in further analyses because they were currently undergoing treatment for major depression. For the overall scale, Cronbach's $\alpha = .85$.

2.2.2 Rumination

Ruminative Thought Style Questionnaire (RTSQ; Brinker & Dozois, 2009). The German version of the RTSQ (Helmig et al., 2016) consists of 20 items measuring rumination on a 7-point Likert-type scale (1 = "not at all", 7 = "very well"). Both a single rumination factor (Brinker & Dozois, 2009)

and four subcomponents (problem-focused thoughts, counterfactual thinking, repetitive thoughts, and anticipatory thoughts; Tanner et al., 2013) were verified empirically (for the single factor, Cronbach's $\alpha = .93$).

2.2.3 Drinking Motives

Drinking Motives Questionnaire Revised Short Form (DMQ-R SF; Kuntsche & Kuntsche, 2009). The German version of the DMQ-R SF assesses four drinking motives (conformity, coping, enhancement, social) with 12 items on a 5-point Likert-type scale (1 = "almost never / never", 5 = "almost always / always"). For the overall scale, Cronbach's $\alpha = .92$. The subscales / motives, each consisting of three items, had similarly high reliabilities: conformity motives ($\alpha = .83$), coping motives ($\alpha = .91$), enhancement motives ($\alpha = .87$), and social motives ($\alpha = .93$).

2.2.4 Alcohol-Related Problems and Alcohol Consumption

Alcohol Use Disorder Identification Test (AUDIT; World Health Organization, 2001). The AUDIT is a 10-item screening scale to assess alcohol consumption, drinking behaviors, and alcohol-related problems. In both the original and the German version (Dybek et al., 2006), one of five statements about alcohol consumption in the past year can be selected for each question on a 5-point Likert scale from 0 ("never") to 4 (e.g., "daily or almost daily"). A cut-off value of five is recommended for the German version (cf. Dybek et al., 2006; Rumpf et al., 2002). In the current sample, Cronbach's $\alpha = .88$.

In addition, the German AUDIT asks about the number of drinking days in the last month (item 11: "On how many days did you drink alcohol during the last 30 days? __ days (please fill in: 0-30)"), and the number of consumed drinks on a typical drinking day in the last month (item 12: "If you drank alcohol during the last 30 days: How many glasses of what beverages did you drink on a typical day? __Beer (0.33 l), __Beer (0.5 l), __Wine/champagne (0.2 l), __Spirits (0.02 l)"). Using the number of drinks, we first calculated the total amount of alcohol in percent by volume (e.g., 5% for a 0.33 beer) and then converted that into grams of alcohol. We multiplied the grams of alcohol by the number of

drinks consumed in the past 30 days, as measured with item 11, and then divided the result by 30 to calculate the average amount of alcohol consumed per day.

2.3 Statistical Analyses

All statistical analyses were conducted using R 4.0.2 (R Core Team, 2020) and RStudio 1.3.959 (RStudio Team, 2020). To test the proposed relations (H1-H3), SEM using lavaan 0.6-7 (Rosseel, 2012) was conducted.

We tested two models in total. The first, more complex model closely followed the specifications of Bravo and colleagues (2018). In the following, it will be referred to as the *rumination factor model*. The second model, referred to as the *simplified model*, differs somewhat from the models of Bravo et al. (2018) and from the rumination factor model. The rationale for the specification of this simplified model is explained in detail in section 3.2.2. The analysis steps of both models are described in the following sections.

2.3.1 Model Structure

The latent variable "depression" was operationalized using the CES-D, "rumination" using the RTSQ, the four “drinking motives” using the DMQ, and “alcohol consumption” as well as “alcohol-related problems” using the AUDIT.

In the rumination factor model, depressive symptoms were associated with rumination and alcohol outcomes. Rumination, in turn, was related to drinking motives (conformity, coping, enhancement, social) and alcohol outcomes. In addition, drinking motives were related to alcohol outcomes. Consequently, the model was designed to have the following structure: depressive symptoms → rumination → drinking motives → alcohol outcomes.

The RTSQ allows to assess four rumination factors (problem-focused thoughts, counterfactual thinking, repetitive thoughts, and anticipatory thoughts). In the rumination factor model, these factors served as indicators of a second-order latent rumination factor. Since Tanner et al. (2013) concluded that 15 of 20 items adequately represent the four rumination factors based on a factor analysis of the RTSQ, Bravo and colleagues (2018) opted for this reduced version of the questionnaire. Following

Bravo et al. (2018), we also employed these 15 items as manifest variables for estimating the four factors.

The simplified model assumes the same interrelationships of factors as the rumination factor model (see above), but the latent variables "drinking motives" and "rumination" were operationalized in a somewhat leaner manner. Instead of distinguishing between four drinking motives and four rumination subscales, only one general drinking and one rumination factor were specified (compare results section 3.2.2). The structure of the rumination factor model is shown in Figure 1, that of the simplified model in Figure 2.

Following Bravo and colleagues (2018), we used the item-to-construct balance procedure (Little et al., 2002) to reduce model complexity. An exploratory factor analysis (EFA) was conducted that identified a single factor for the two constructs "depressive symptoms" and "alcohol-related problems". For each latent factor, three parcels were created, each containing between three and seven items depending on the questionnaire used. Then the items with the highest and lowest factor loadings were assigned to the first parcel, the items with the second highest and second lowest factor loadings were assigned to the second parcel, and so on. This procedure was continued until all items were distributed across three balanced parcels (for a detailed description, see Little et al., 2002).

In the simplified model, parceling was additionally applied to the constructs "rumination" and "drinking motives". We again decided to use this approach because, on the one hand, we aimed at further reducing model complexity and, on the other hand, because building latent variables with the help of item parcels can be beneficial for the model. For example, parcels are more likely to be related to the latent variable, more likely to be normally distributed, and less likely to be contaminated by methodological effects or item formulations (Marsh et al., 1998). After EFA was conducted, three balanced parcels were built for the factors "rumination" and "drinking motives", each containing four to seven items.

2.3.2 Structural Model Differences

Our models differ from the model specifications of Bravo and colleagues (2018) in two respects. First, we used the AUDIT as indicator of negative alcohol-related consequences (consumption and

problems). We decided against using the Daily Drinking Questionnaire (DDQ; Collins et al., 1985) and the Young Adult Alcohol Consequences Questionnaire (YAACQ; Read et al., 2006) because the AUDIT is the most frequently used diagnostic instrument in Germany for problematic alcohol consumption, but also for the assessment of alcohol-related problems (e.g., it is asked whether one can no longer meet obligations or has injured oneself or others while intoxicated). Moreover, no German versions of the other two questionnaires were available.

Second, we focused exclusively on the direct effects of each predictor variable on alcohol outcomes and on the direct effects of one variable on the subsequent variable along the model structure (e.g., depression → rumination). However, we decided against regression analyses and in favor of SEM a) because we wanted to replicate the results of Bravo et al. (2018) and therefore followed their lead and b) because of the many advantages associated with the SEM framework, such as the ability to interpret or estimate latent variables or to obtain information about the goodness of model fit (cf. Gunzler et al., 2013). Readers interested in the indirect and total effects can find them as supplementary data in the appendix (Table A2 and Table A3).

2.3.3 Data Preparation and Further Analysis

First, univariate and multivariate normality and outliers were screened. Three out of four questionnaires were not normally distributed. In addition, five outliers were identified and excluded from the analyses. There was no missing data. Due to the presence of ordinal outcome measures (indicators) and non-normally distributed data, the standard maximum likelihood (ML) was used to estimate the parameters of the rumination factor model with robust standard errors and the Satorra-Bentler scaled test statistic (Satorra & Bentler, 1994). This method was chosen because its correction is effective in medium sized samples with non-normally distributed data (Chou et al., 1991; Curran et al., 1996).

For the simplified model, the Diagonally Weighted Least Squares (DWLS) method with robust standard errors and the Satorra-Bentler scaled test statistic (Satorra & Bentler, 1994) was used. DWLS is a suitable estimation method for medium-sized samples of ordinal data that (as in this case) deviate strongly from the normal distribution (Jöreskog & Sörbom, 1989). In a model with more than 20

variables, DWLS provides less accurate estimates and requires a particularly large sample (Gana & Broc, 2019), which is why its use in the rumination factor model did not seem appropriate.

2.3.4 Model Fit Indices

To verify the overall model fit in both of our models, we used criteria for fit indexes suggested by Hu and Bentler (1999), such as Comparative Fit Index (CFI) > 0.95, Tucker-Lewis Index (TLI) > 0.95, Root Mean Square Error of Approximation (RMSEA) < .06, and Standardized Root Mean Square Residual (SRMR) < .08. To test the statistical significance of the model, the chi-square (χ^2) test was used. In addition, we tested the local fit of the solution by examining the factor loadings of the measurement model (which is equated to a confirmatory factor analysis). Factor loadings that are positive and > .40 indicate a relevant link between indicator and corresponding factor (cf. Gana & Broc, 2019).

Results

3.1 Descriptive Statistics

The mean value of the CES-D was in the clinically significant range (M = 34.0, SD = 10.2). The mean value of the AUDIT was below the cut-off indicating problematic alcohol use (M = 4.2, SD = 5.4). A relatively low mean value was also found for the DMQ (M = 10.4, SD = 10.8), and a relatively high value for the RTSQ (M = 99.4, SD = 22.7). The bivariate correlations of the variables can be found in Table 2.

3.2 Structural Equation Modeling

3.2.1 Rumination Factor Model (following Bravo et al., 2018)

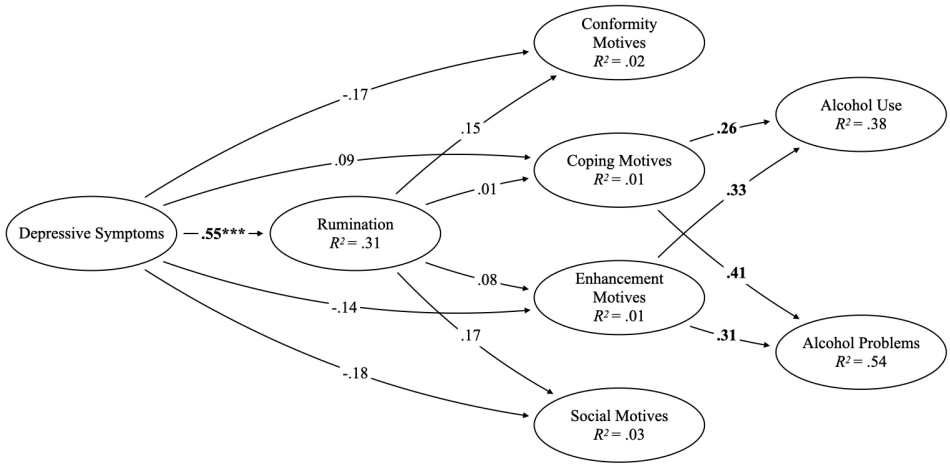
Each factor loading (λ) of the (standardized) measurement model was positive and > .40. For example, the loadings of the depression parcels were all significant and similarly high (parcel one: λ = .84). The initial model provided an acceptable fit (robust indices): CFI= 0.92, TLI = 0.91, RMSEA = .06 (90% CI [0.05, 0.07]), SRMR = .06. The significant model χ^2 [χ^2(496, N = 209) = 785.05, p < .001]

would suggest poor model fit, however, the model χ^2 is highly sensitive to sample size (Gana & Broc, 2019).

According to the (standardized) structural model, depressive symptoms are positively associated with rumination ($\beta = 0.55$, $p < .001$). Both coping and enhancement motives are positively related to alcohol-related problems ($\beta_{CO} = 0.41$, $p = .001$; $\beta_{EN} = 0.31$, $p = .047$) and the amount of alcohol consumption ($\beta_{CO} = 0.26$, $p = .049$; $\beta_{EN} = 0.55$, $p < .001$). Compare Figure 1 for all direct effects.

Figure 1

Rumination Factor Model



Note. Depicts all standardized direct effects of the rumination factor model ($N = 209$). Significant associations are in bold typeface for emphasis. The nonsignificant associations between the predictor variables (depressive symptoms, rumination, drinking motives) and both outcome variables (alcohol consumption and alcohol-related problems), as well as the factor loadings are not shown in the figure for reasons of parsimony. An overview of all direct effects on both outcome variables, the corresponding confidence intervals and p-values can be found in the appendix, Table A1. Factor loadings are available from the authors upon request.

Table 2

Bivariate Correlations Between Study Variables in the Total Sample

	1	2	3	4	5	6	7	8	9	10	11	12
1. Depressive Symptoms	–											
2. Rumination	**.46**	–										
3. Problem-focused thoughts	**.45**	**.86**	–									
4. Anticipatory thoughts	**.36**	**.78**	**.61**	–								
5. Counterfactual thinking	**.35**	**.81**	**.59**	**.51**	–							
6. Repetitive thoughts	**.42**	**.79**	**.62**	**.53**	**.55**	–						
7. Drinking motives	-.04	.10	-.00	.04	**.14***	.06	–					
8. Coping	.09	.08	.01	.02	.07	.10	**.79**	–				
9. Conformity	-.07	.09	.03	.11	.10	-.03	**.73**	**.48**	–			
10. Social	-.07	.12	-.01	.04	**.19**	.08	**.86**	**.48**	**.60**	–		
11. Enhancement	-.09	.05	-.03	-.01	.08	.03	**.86**	**.59**	**.45**	**.70**	–	
12. Alcohol consumption (in grams)	-.06	-.07	-.13	-.12	-.01	-.06	**.57**	**.51**	**.32**	**.45**	**.53**	–
13. Alcohol-related problems	.03	.00	-.07	-.03	.05	.03	**.64**	**.62**	**.42**	**.47**	**.56**	**.47**

Note. For rumination and drinking motives, the correlations of the total scales as well as those of the subscales are shown. Significant correlations ($p < .001$) are bolded for emphasis.

An exception to the significance level is marked accordingly: * $p < .05$

3.2.2 Simplified Model

Based on the original model, in addition, a second (less complex) model was specified in order to deal with potential power problems. In particular, the exceedingly small association of depression and drinking motives / alcohol outcomes as well as of rumination and drinking motives prompted a re-specification. Note that the sample size was smaller than that of Bravo and colleagues (2018), but the complexity of the model was the same.

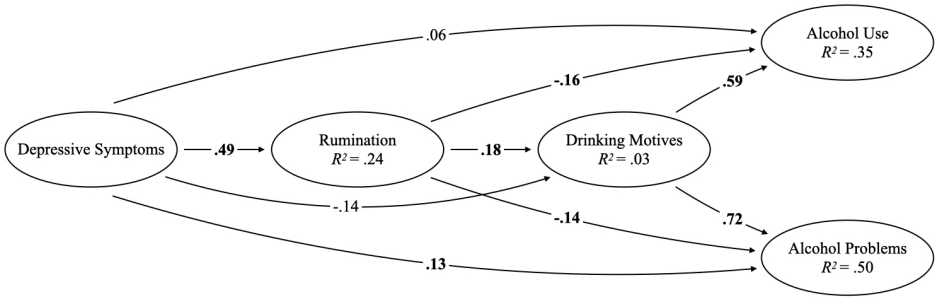
The simplified model assumes a single depression factor, a single factor for rumination, a general factor for drinking motives, as well as two factors for the consequences of alcohol use, i.e., consumption and alcohol-related problems, and has 35 free parameters. For the application of ML or DWLS, there should be five times as many participants as free parameters, i.e., at least 175 (which the present sample size allows; cf. Bentler, 1995).

In the simplified (standardized) measurement model, the factor loadings were again all positive and > .40. The simplified model provided an excellent fit (robust indices): CFI= 0.99, TLI = 0.99, RMSEA = .04 (90% CI [0.03, 0.05]), SRMR = .05. The nonsignificant model χ^2 [$\chi^2(56, N = 209) = 113.46, p = < .001$] would suggest poor model fit, however, the model χ^2 is highly sensitive to sample size (Gana & Broc, 2019).

As seen in Figure 2, depressive symptoms are positively associated with rumination ($\beta = 0.49$, $p < .001$) and alcohol-related problems ($\beta = 0.13$, $p = .036$). Rumination is positively linked to drinking motives ($\beta = 0.18$, $p = .022$), and negatively linked to alcohol-related problems ($\beta = –0.14$, $p = .011$) as well as alcohol use ($\beta = –0.16$, $p = .017$). Drinking motives are positively connected with alcohol consumption ($\beta = 0.59$, $p < .001$) and negative alcohol-related problems ($\beta = 0.72$, $p < .001$). For both models, an overview of all direct effects on both outcome variables, the corresponding confidence intervals and p-values can be found in the appendix, Table A1.

Figure 2

Simplified Model



Note. Depicts all standardized direct effects of the simplified model (N = 209). Significant associations are in bold typeface for emphasis. An overview of all direct effects on both outcome variables, the corresponding confidence intervals and p-values can be found in the appendix, Table A1. Factor loadings are not shown in the figure for reasons of parsimony, but are available from the authors upon request.

Discussion

The aim of this study was to examine the association between depressive symptoms and negative alcohol-related consequences using structural equation modeling. To this purpose, we transferred a previously evaluated model explaining the association via rumination and drinking motives (cf. Bravo et al., 2018) to a clinically depressed population.

In the course of the study, we succeeded in partially confirming our hypotheses: in the more complex model, which assumes a second-order rumination factor and differentiates between four drinking motives, depressive symptoms were associated with rumination, and coping and enhancement motives related to both alcohol-related problems and alcohol consumption. Somewhat surprisingly, no association was found between rumination and drinking motives or between rumination and negative alcohol outcomes. However, in a simplified model (with more statistical power), the associations between depressive symptoms, rumination and alcohol-related problems were significant. Specifically, rumination was positively associated with drinking motives and negatively associated with negative alcohol-related consequences. In addition, both drinking motives were significantly linked to both alcohol use and alcohol-related problems.

Given the results of the simplified model, it is reasonable to assume that the assumptions of Bravo and colleagues (2018) are also valid, at least in part, in a sample of individuals suffering from major depression. Nevertheless, it should be noted that only some associations (e.g., between depression and rumination) from the Bravo model could be replicated, and that new significant associations also emerged. For example, unlike in the model based on a student population, enhancement motives (e.g., because I like the good "feeling") were significantly associated with alcohol use and alcohol-related problems in addition to coping motives. These findings are consistent with those of previous research and highlight a relatively common association between drinking motives and alcohol outcomes: self-related motives with positive (reinforcement) and negative (coping) reinforcement promote alcohol use (Piasecki et al., 2014) and alcohol-related problems (Comasco et al., 2010; Webb et al., 2020) in both clinical (alcohol use disorder; Cho et al., 2019) and nonclinical samples (Simons et al., 2017). To retest the more complex model in future studies, a larger sample would be required to achieve sufficient statistical power. Therefore, it remains possible that a significant relationship between rumination and, for example, specific drinking motives might then emerge. However, the effect size of such a relationship is likely to be small.

Considering the connections within the simplified model also seems significant in this context. Although we were able to show that rumination is associated with drinking motives and that these, in turn, are associated with increased negative alcohol-related consequences, no differentiated conclusions can be drawn about the association of specific drinking motives with rumination and alcohol outcomes, since the model was calculated with a general motive factor. Therefore, it can only be assumed that rumination is associated with a general motivation to drink alcohol and that this motivation, in turn, is related to alcohol consumption and alcohol-related problems. Moreover, the majority of studies on drinking motives advocate the four-factor structure (for a review, see Kuntsche et al., 2005) because, on the one hand, it is consistent with the assumptions of the motivational model of alcohol use (Cox & Klinger, 1988) and, on the other hand, it has already been confirmed as the best solution (e.g., Crutzen & Kuntsche, 2013). Nonetheless, there are some studies that support a single-factor solution for drinking motives (Lac & Donaldson, 2017a; Lac & Donaldson, 2017b; Urbán et al., 2008) and also provide evidence for a general drinking motivation that appears to exist beyond the four drinking motives (Lac

& Donaldson, 2017a). In light of the common four-factor solution, our single-factor structure may represent a limitation in the validity / interpretation of our results, but at the same time may also prompt future studies to examine both factor solutions in relation to clinical depression, rumination, and negative alcohol-related consequences.

The negative association between rumination and alcohol outcomes is also of interest. This association was significant in the simplified model and also observable in the model of Bravo et al. (2018). Such a suppression effect is difficult to interpret. Yet, assuming that there is a negative effect of rumination on alcohol use and alcohol-related problems, the finding might suggest that individuals with depression whose ruminative thinking is more pronounced are less likely to use alcohol as a strategy for dealing with their repetitive thoughts. Bravo et al. (2018) did not directly interpret the direction of this association, but only highlighted the link. Nonetheless, one might speculate that depression motivates individuals to deal with their negative feelings either through rumination (for a functional account of rumination within a metacognitive explanation of depression; cf. Papageorgiou & Wells, 2003) or, alternatively, through the consumption of alcohol. The negative association would then be the result of individuals either using one or the other coping mechanism. Another, alternative explanation is that this finding might be the result of *net* or *cross-over suppression*: sometimes the weight of the stronger predictor can change the sign of the weaker predictor after calculating the regression weights (Paulhus et al., 2004). In the present data set, with regard to the criterion alcohol-related problems, adding the predictor drinking motives changed the sign of the predictor rumination to negative, although the null correlation between rumination and alcohol-related problems was positive.

The positive association between drinking motives and alcohol outcomes suggests that alcohol is experienced as helpful when consumption is actively used as a coping strategy. Arguably, rather than to cope with recurrent negative thoughts (i.e., rumination), alcohol may be used with the intention to regulate negative affect directly. At least, the positive association between depressive symptoms and alcohol-related problems combined with the negative association between rumination and both negative alcohol outcomes could be interpreted that way. This positive relation between aversive affect and alcohol outcomes is consistent with the assumptions of various affect regulation models (Cooper et al., 1995; Quitkin et al., 1972). However, empirical evidence justifying this postulated association in

clinically depressed individuals is scarce (Anonymous, submitted for publication); most studies either assessed nonclinical samples (Gonzalez et al., 2011; Kenney et al., 2015; Simons et al., 2016; Stewart & Devine, 2000) or focused on individuals with anxiety (Sorid et al., 2021) / anxiety disorders (e.g., Battista et al., 2010) rather than affective disorders.

Similarly, the assumption that rumination acts as a link between depressive symptoms and alcohol consumption or alcohol-related problems is empirically confirmed by only a small number of studies limited to nonclinical samples (Bravo et al., 2017; Bravo et al., 2018). Thus, the validity and generalizability of these results are limited. Also, the connection between rumination and alcohol outcomes was found exclusively in correlative studies (e.g., Ciesla et al., 2011; Nolen-Hoeksema & Harrell, 2002).

Limitations

Some limitations of this study should be noted. First, the study design was cross-sectional and examined only direct effects. Longitudinal data would have been necessary to test causal (indirect / mediated) relations. Second, our methodological approach may not in all cases have ensured a valid diagnosis since we had to adjust our recruiting process due to the COVID-19 pandemic. Although we recruited most participants through professional staff, we still obtained self-report information and applied cut-off criteria from the depression questionnaire to determine caseness. However, the use of a clinical interview would have significantly increased the validity of the diagnostic procedure. Third, the use of the RTSQ to assess rumination can be criticized, as it may not be a depression-specific instrument. Bravo et. al. (2018) also pointed out that there are alternatives with potential benefits, such as the Ruminative Responses subscale of the Response Styles Questionnaire (cf. Nolen-Hoeksema & Morrow, 1991). But since we wanted to deviate as little as possible from the originally used dimensions, we opted for the RTSQ. Fourth, the relatively low proportion of variance explained for alcohol outcomes may indicate the influence of other variables, such as neuroticism or alexithymia (Chinneck et al., 2018; Lyvers et al., 2019; Ruiz et al., 2003). Finally, some characteristics of the sample can be criticized: the aforementioned small sample size, which limits statistical power, the small proportion of male participants, who tend to differ from women in terms of mood-related drinking motives (Foster et al.,

2014; Young-Wolff et al., 2009), and the different recruitment methods (online vs. practice / clinic), which may have favored demographic differences in the sample and biased the results (see, e.g., Benedict et al., 2019; Topolovec-Vranic & Natarajan, 2016).

Conclusions

In summary, in a clinically depressed sample, we found that depressive symptoms are associated with rumination, and rumination in turn is related to drinking motives. Furthermore, drinking motives are both associated with subsequent alcohol use as well as alcohol-related problems. Our results confirm that rumination may be important when understanding the association between depressive symptoms and negative alcohol-related consequences. Nevertheless, further research is needed on the specific direct and indirect effects of rumination, both in terms of specific drinking motives and alcohol-related consequences. In the search for treatment targets in comorbid cases of alcohol use disorders and affective disorders, both rumination and negative affect are suggested as valid candidates by our findings.

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Appendix

Table A1

Summary of all Direct Effects of Depressive Symptoms, Rumination, and Drinking Motives on Alcohol Consumption and Negative Alcohol-Related Problems in Both Structural Equation Models

Rumination Factor Model							
Outcome variables		Alcohol consumption			Negative alcohol-related problems		
		β	CI	*p*	β	CI	*p*
Predictor variables	Depression	0.03	-0.18, 0.31	*ns*	0.09	-0.05, 0.22	*ns*
	Rumination	-0.14	-0.28, 0.02	*ns*	-0.10	-0.10, 0.02	*ns*
	Conformity motives	-0.03	-0.29, 0.23	*ns*	0.12	-0.10, 0.21	*ns*
	Coping motives	0.26	0.05, 0.39	*.011*	0.41	0.07, 0.25	*.001*
	Enhancement motives	0.33	0.05, 0.52	*.019*	0.31	0.00, 0.25	*.047*
	Social motives	0.17	-0.13, 0.34	*ns*	0.01	-0.12, 0.13	*ns*
Simplified Model							
Outcome variables		Alcohol consumption			Negative alcohol-related problems		
		β	CI	*p*	β	CI	*p*
Predictor variables	Depression	0.06	-0.07, 0.23	*ns*	0.13	0.01, 0.36	*.036*
	Rumination	-0.16	-0.32, -0.03	*.017*	-0.14	-0.31, -0.04	*.011*
	Drinking motives	0.59	0.53, 0.92	*< .001*	0.72	0.70, 1.30	*< .001*

Note. CI = 95% confidence interval.

Table A2

Summary of all Indirect and Total Effects of Depressive Symptoms, Rumination, and Drinking Motives on Alcohol Consumption and Negative Alcohol-Related Problems in the Rumination Factor Model

Rumination Factor Model												
Outcome variables	Alcohol consumption		Negative alcohol-related problems		Drinking motives: CN		CO		EH		SO	
Predictor: *depression*	β	CI	β	CI	β	CI	β	CI	β	CI	β	CI
Total	-0.01	-0.55, 0.25	0.12	-0.04, 0.26	-0.08	-0.51, 0.19	0.09	-0.15, 0.60	-0.09	-0.56, 0.13	-0.08	-0.53, 0.16
Indirect												
Rumination	-0.08	-0.35, 0.02	-0.06	-0.12, 0.02	0.08	-0.05, 0.37	0.92	-0.22, 0.25	0.36	-0.12, 0.34	0.09	-0.02. 0.48
CN	0.01	-0.08, 0.10	-0.02	-0.08, 0.04								
CO	0.02	-0.06, 0.15	0.04	-0.04, 0.11								
EH	-0.05	-0.23, 0.04	-0.04	-0.11, 0.03								
SO	-0.02	-0.14, 0.06	(-)0.00	-0.05, 0.05								
Rumination – CN	(-)0.00	-0.05, 0.04	0.01	-0.02, 0.04								
Rumination – CO	0.00	-0.05, 0.06	0.00	-0.04, 0.04								
Rumination – EH	0.02	-0.04, 0.10	0.01	-0.02, 0.04								
Rumination – SO	0.01	-0.03, 0.08	0.00	-0.03, 0.03								

Predictor: *rumination*	β	CI	β	CI
Total	-0.10	-0.27, 0.09	-0.05	-0.09, 0.05
Indirect				
CN	(-)0.00	-0.04, 0.03	0.02	-0.02, 0.03
CO	0.00	-0.04, 0.04	0.00	-0.03, 0.03
EH	0.03	-0.03, 0.08	0.03	-0.01, 0.03
SO	0.02	-0.02, 0.06	0.00	-0.02, 0.02

Note. No significant associations were found. The effects were calculated using the delta method (cf. Rosseel, 2012). CN = Conformity motives, CO = Coping motives, EH = Enhancement motives, SO = Social motives. CI = 95% confidence interval

Further comment by the authors: In contrast to Bravo and colleagues (2018), no significant total or indirect effects were found for the rumination factor model.

Table A3

Summary of all Indirect and Total Effects of Depressive Symptoms, Rumination, and Drinking Motives on Alcohol Consumption and Negative Alcohol-Related Problems in the Simplified Model

Simplified Model						
Outcome variables	Alcohol consumption		Negative alcohol-related problems		Drinking motives	
Predictor: *depression*	β	CI	β	CI	β	CI
Total	0.11	-0.01, 0.30	**0.19**	0.09, 0.46	-0.05	-0.21, 0.11
Indirect						
Rumination	**-0.08**	-0.19, -0.01	**-0.07**	-0.19, -0.01	**0.09**	0.01, 0.17
Drinking motives	-0.08	-0.22, 0.02	-0.10	-0.31, 0.03		
Rumination – drinking motives	**0.05**	0.00, 0.13	**0.06**	0.01, 0.17		
Predictor: *rumination*						
Total	-0.06	-0.25, 0.13	-0.02	-0.21, 0.17		
Indirect						
Drinking motives	**0.11**	0.01, 0.22	**0.13**	0.02, 0.30		

Note. Significant associations are in bold typeface for emphasis and were computed using the delta method (cf. Rosseel, 2012). CI = 95% confidence interval