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CONTENTS

Dear reader of our Maastricht Student Journal of Psychology and Neuroscience,

We present to you: volume 9 of our Maastricht Student Journal of Psychology and Neuroscience.

The journal's main aim is to provide students with the valuable experience of both publishing and reviewing scientific articles. Publishing your own work makes you go through the process of receiving feedback, rethinking your work, adjusting it, and resubmitting it, all using a format that is also used by peer reviewed scientific journals. Reviewing papers provides the opportunity critique someone else's writing, which possibly makes you susceptible to spotting room for improvement in your own writing. Therefore, both aspects prepare you for being a better scientific writer.

Since the previous edition of this journal we have set a path that should lead to increased visibility of our journal and publishing a larger variety of paper types. Our efforts included appearances in newsletters, spreading hardcopies of the journal, and notifying students during teaching activities that they could publish their work in our journal. This has resulted in an increase in papers that have been submitted and we were planning to publish two editions per year instead of one.

However, then the Covid-19 crisis took hold of our lives. The crisis demanded focus on curricular work of both students and staff. The absence of routines, the amount of time that was spent on

preparing online and hybrid teaching, additional rules and arrangements to consider when performing studies, and simply studying or working in a non-work environment (i.e. home) all pressed hard on the time we could spend efficiently on our beloved journal. The increased workload resulted in the absence of a 2020 edition of the Maastricht Student Journal of Psychology and Neuroscience despite the increase in submissions resulting from our previous actions.

There does appear to be light at the end of the tunnel. The number of corona virus infections is decreasing and restrictions are being lifted, providing space for regular work routines again. The time we have spent on learning new ways of teaching, studying and working should also pay off by having a larger toolbox available, which could actually free up time and space. Finally, Maastricht University (with the Faculty of Psychology and Neuroscience at the forefront) is working on reducing workload for everyone. In my opinion, all these measures and events will lead to more time spend on creative processes that are associated with writing. It allows more room for our enthusiasm, which sparks thoughts about the topics we wish to write about; those bright ideas that you typically have in the shower or when doing the dishes; the moments when the outside world is not in demand.

Let's hope that the increased bandwidth for creative work will be the foundation of future papers published in our journal. Maastricht Journal of Psychology and Neuroscience is often the first

contact students have with publishing their work, which reflects an important part of a potential future career in science. Next to the formal routines that it provides students for publishing their work, it should provide the seed for creativity and enthusiasm. The journal should be a place for ideas that ultimately should enrich the knowledgebase of our community. With increased space and time for both authors, reviewers and editors to develop these ideas, and our platform to mediate spreading of the ideas, we hope to contribute to the development of students as scientists with fruitful and enjoyable careers.

Back to reality, the current edition is the result of hard work of some very resilient authors, reviewers and editors, who have pushed hard to make this 9th edition a reality despite the less-than-ideal situation. This edition contains a review of the literature on psychedelic drugs by *Philipp Mateo Härter*. The review is timely given the increased interest in psychedelic drugs to be used in clinical settings. The author discussed the evidence and neurobiology of ego-dissolution caused by psychedelic drug administration. The second paper in this edition is a report of an experiment that assesses the effects of 'Bulletproof coffee' written by *Anna Bergauer, Elisabeth Achteresch, Leonard Niekerken, Tom Fernandes Visser, Noa Kök, Alyssa Meng, Antonia Varsamis, & Jannis Wolff*. So called 'Bulletproof coffee' is a mixture of coffee with medium chain triglycerides (e.g. coconut oil) and butter. The authors put the popular claim to the test that the mixture has cognition enhancing properties. Next, *Anna Katharina Selter* reviews the evidence for emotion (dis)regulation to

mediate the occurrence of nightmares in traumatised children. She presents a comprehensive view by discussing both psychological and biological mechanisms. Finally, *Lianne Wolsink* presents an overview of experimental studies that assessed the effects of initial denial of crime-related events on later recall of those events. The topic is highly relevant for societal issues in crime solving.

We hope that this edition provides inspiring ideas and motivation to continue studying scientific topics. In addition, we hope that we can provide the platform to spread at least some of these ideas.

Peter van Ruitenbeek, PhD

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This paper is the product of students from the Faculty of Psychology and Neuroscience, Maastricht University and is meant for student educational purposes only.

PHILIPP MATTEO HÄRTER

The Influence of Psychedelic Drugs on the 'Sense of Self'

Literature Review

Potent psychedelic drugs such as LSD and psilocybin are implicated in a multitude of cognitive and perceptual alterations. Through the mediation of serotonergic 5-HT2 receptors, distortion of the self is a common effect of these drugs. The self is usually referred to as an entity comprising physical and psychological attributes that are coherent with our self-concept. This so-called binding process can be affected through psychedelic experiences and can oftentimes lead to the phenomenon of ego-dissolution. Given this, ego-dissolving experiences lead to the elimination of a bound self-model which eventually results in the perception of distorted self-boundaries. There have been improvements conceptualizing and assessing ego-dissolution. The Ego-Dissolution Inventory is the first questionnaire dealing with the assessment of this phenomenon. In addition, a variety of neural studies associate this common psychedelia-induced phenomenon with different brain regions. The Default Mode Network and the Salience Network, two large-scale networks associated with distinct components of the self, show

altered activity and disintegration, leading to an enhanced global connectivity within the brain. Additionally, decoupling of the Medial Temporal Lobe, alterations in parahippocampal activity and reduced interhemispheric communication show the same correlations. These findings are first attempts to conceptualize the self and the concept of ego-dissolution in terms of large-scale networks within the brain.

Keywords: sense of self, psychedelics, self-binding, ego-dissolution, neural correlates, brain networks

INTRODUCTION

Despite their different chemical structure, these psychedelic effects are mediated by serotonin 2A receptor (5-HT2) agonism (Nichols, 2016). People under the influence of psychedelics experience visual alterations, such as color enhancement and color shifting. Common physical responses comprise increased heart rate, pupil dilation and tactile enhancement. Commonly referred to as hallucinogens, they increase a vivid perception of the outer world and generate strong visual, auditory and tactile hallucinations.

Arguably, a unique side effect of psychedelic drugs is the capacity to generate distorted thoughts, delusions and spiritual experiences. In this regard, being under the influence of psychedelics can lead to remarkably distorted ego perceptions, a phenomenon called ego-dissolution (ED) (Dittrich, 1998). This phenomenon can be explained by a disruption of the subjective experience of the 'sense of self' (James, 1882, Carhart-Harris et al., 2014), which oftentimes leads to the perception of blurred self-boundaries. For instance, during the activity of these mind-altering drugs the self-concept can experience a breakdown within its subjective and coherent psychological attributes. Subjects under psychedelic intoxication report a sense of alienation with the outer world, obtrusive internalized thought or an abolished feeling of connection with one's body and self (Milliere, 2017). Therefore, typical experiences under psychedelic drugs comprise an altered state of the sense of self, known as ego-dissolution as well as self-oriented mental activities (Palhano-Fontes et al., 2015).

It is necessary to precede with a short theoretical framework of the sense of self in order to understand the mechanisms of a self-disruptive phenomenon such as ego-dissolution. On the one hand, the self can be explained as a three-folded concept including a conjoint construct of experience, self-awareness and selfhood (Zahavi, 2008). On the other hand, the psychoanalytic approach defined by Freudian devotees, explains the ego as an intermediary unit between the unsocialized and selfish impulse-driven identity and the superego, which keeps internalized societal norms in check (Hogg & Vaughan, 1995).

To emphasize the importance of a cohesive or dissolved ego, clinical samples present major scientific insights. Disturbances of the self are one of the main symptoms in patients suffering from schizophrenia. Commonly, schizophrenic patients often show symptoms like hallucinations, delusions, avolition and affective/cognitive disruptions (Andreasen, 1995). Furthermore, people suffering from schizophrenia report in progressing stages of their illness a passivity phenomenon, which is characterized by a disturbance of the self which is no longer perceived as part of the human. In addition to the absence of a coherent self-image, patients become detached, show feelings of alienation and have a sense of estrangement concerning their own experiences (Northoff, 2014). It is therefore that in schizophrenia self-models are blurred, non-stationary and unpredictable by the perceiver.

Scientific investigations of the underlying mechanisms shaping the dynamic character of the self, remains a challenge. However, since psychedelics are known to induce a self-distortive ego-dissolution, it could be used as a tool to explore its underlying behavioral and neurobiological

mechanisms. So far, recent neuroscientific research shows little insight into the neurological grounds of ego-dissolution phenomena outside of the clinical context. Promoting a thorough neurobiological understanding of ego-disturbances under the influence of psychedelia would serve two purposes. First, the experimental use of psychedelic-induced ego-dissolution may provide us with new tools to investigate the neural correlates of the self, increasing the valence of research on self-models. Second, the understanding of neurological agents in ego-dissolution may emphasize beneficial effects of psychedelia-induced ego-dissolution which may inform novel treatment strategies.

In the latter scenario, recent studies have already given insight into positively experienced ego-dissolution and spiritual phenomena during the psychedelic experience. One work by Uthaug et al. (2018) studied the long-term impact of an Ayahuasca ceremony on affective and cognitive capacities as well as attitudes towards life. Moreover, this research attempted to shed light on positive ego-dissolution experience and according changes in life satisfaction, affective alterations and mindfulness. The results showed that all three aspects were significantly correlated with experienced ego-dissolution during Ayahuasca intake, therefore affirming useful long-lasting changes (Uthaug et al., 2018). This finding is consistent with other research on treatment-resistant depression, where an experimentally induced psilocybin dose predicted positive therapy outcome. In this context, an enhanced mystical experience termed oceanic boundlessness and low fear for ego-dissolution were main predictors of this effect (Roseman, Nutt & Carhart-Harris, 2018). In summary, ego-

dissolution plays an integral part in positive psychedelic experiences and can be associated with schizophrenic or depressive symptoms.

In spite of these promising findings, ego-dissolution remains a vague concept which requires further clarification. It remains unknown how to conceptualize distinct ego-dissolving experiences or which neurobiological processes take place. It is therefore crucial to shed light on to the psychological aspects of the self, the neurobiological structures of psychedelic ego-dissolution, and to quantify the construct through validated measures. This literature review seeks to elaborate on the question of how psychedelics influence the sense of self in terms of neurobiological and behavioral findings. First, binding as a mechanism of a coherent sense of self and the process of ego-dissolution will be investigated. Second, the novel Ego-Dissolution Inventory will be presented with its robust internal and construct validity. Lastly, the review concludes with findings on neural correlates underlying ego-dissolution. In order to study the mechanisms of ED, the online databases PubMed and Google Scholar were searched for publications containing the following key words: sense of self, psychedelics and self-binding, psychedelia induced ego-dissolution, ego-dissolution inventory, neural correlates of drug induced ego-dissolution, ego-dissolution and brain networks.

The self-concept and the process of binding

When trying to conceptualize the self or the self-concept, one comes across a wide range of defining features. Baumeister (1997) characterizes it as “The individual's belief about himself or herself, including the person's attributes on who and what the self is” (Baumeister, 1999). Another view stems from

Carhart-Harris and Friston (2010) who refer to the self as an umbrella term. Here they state that the concepts of self-awareness, self-monitoring, self-recognition and self-control/agency constitute the bases of the self. Furthermore, Letheby & Gerrans (2017) explain the self as an entity which is guided by higher-level processes in order to bind or integrate information and attributes which matter to oneself. Binding is therefore a process in which physical and psychological attributes are linked to one's self-model in a coherent and persistent way. Within this framework, a prediction processing model has been postulated, dating back to early developments of Helmholtz and Kant (Helmholtz, 1925; Swanson, 2016). Generally, the process of binding relies on an error signal detection mechanism, finding discrepancies between predicted and actual inputs. In other words, information bound to our self-model is facilitated via a probabilistic model weighing relevant in- and outputs (Letheby & Gerrans, 2017). Therefore, binding is phenomenally or cognitively described as the unity of self-experience and the coherence or integration of existing ideas of the self-model (Revonsuo, 1999). Given this, the self-model represents a persisting object in order to make sense of, to unify and predict patterns of egocentric and salient autobiographical experience.

Ego-dissolution

In some subjects, the intake of psychedelics can generate a powerfully altered perception of the self, named ego-dissolution. Distortions in subjective experiences of one's self or ego are central to the psychedelic experience (Carhart-Harris et al., 2014; James, 1882). From a psychoanalytical perspective, it is the disruption of ego-boundaries, which

results in a blurring of the distinction between self-representation and object-representation. This leads to an unstable synthesis of self-representations into a coherent whole (Federn, 1926; Fischman, 1983; Savage, 1955). Therefore, the dissolution of the self within the psychedelic experience leads to cognitions that are not bound to our self-models any longer. Moreover, the lack of reliable flow of information about the body and one's self-perception as well as the loss of the sense of ownership (Letheby & Gerrans, 2017) particularly contribute to this ego-loss.

As we continue our discussion it will become clear that converging, yet sparse evidence of underlying neurobiological evidence in ego-dissolution phenomena exist. So far, neuroscientific evidence of psychedelic ego-dissolution has been correlated to aspects of separate self-representations such as the embodied and narrative self. The embodied self relates to a persisting entity of the self and the narrative representation to that of goals and attributes important to the self-model (Letheby & Gerrans, 2017). Although this hypothesis remains a challenge to test, both self-modules seemingly seated in different brain networks (Craig & Craig, 2009; Davey & Harrison, 2018; Seth, 2013) compromise the integrity of a functioning binding process. Therefore, it is assumed that psychedelic ego-dissolution represents the collapse of a well-functioning self-binding process. However, evidence that psychedelic ego-dissolution directly abolishes a naturally functioning binding process is currently lacking. Nonetheless, research starts making assumptions about affected entities of self-models based on differential findings on psychedelia-influenced brain activation, as will be discussed in the course of this review.

All in all, diverse aspects and dimensions of the self-model during the psychedelic experience are disturbed, resulting in a decoupling of self-representations. On one hand, this stark feeling can evoke strong emotional reactivity (Soler et al., 2015), but, on the other hand, one can see own dysfunctional emotional and behavioral patterns and can act upon those (Shanon, 2002). Given different conditions, the disruption can lead to a wide range of reactions and can be linked to distinct neural networks related to the self. Therefore, fine-grained psychometric instruments are needed to distinguish between different aspects of the self.

The Ego-Dissolution Inventory

So far, the concept of the self has been discussed, as well as aberrations that can occur during the psychedelic experience. The understanding of this particular phenomenon should be further researched, because self-disturbances and disturbed ego-boundaries are a core phenomenological aspect of psychosis and schizophrenia (Northoff, 2014; Nour and Barrera, 2015). Additionally, experiencing the loss of ego-boundaries is a primary feature of the mystical experience (James, 1985) and leads commonly to cathartic maturity (Shannon, 2002). Conceptualizing the ego is a crucial step to understand pathologies in which the self is disrupted, in order to elaborate on therapeutic interventions. Accordingly, psychedelic drugs may bring a beneficial thoroughfare for upcoming research into neural correlates of normal and abnormal self-awareness (Nour et al. 2016). In order to clear this path, the construction of validated assessments for the concept of psychedelia-induced ego-dissolution is needed.

Currently, there is a considerable number of surveys assessing states of drug-induced self- distortion. The widely used standardized ASC (Altered States of Consciousness) and its revised 5D-ASC scale measure positively and negatively experienced ego dissolving phenomena. Indices of positive depersonalization experiences are captured by the dimension of 'oceanic boundlessness', whereas negative and unpleasant experiences are related to the 'dread of ego dissolution' dimension. In addition, the 'visionary restructuralization' and 'auditory alterations' captures hallucinations or illusions frequently reported by psychedelic drug users. A latter secondary index entails a general measure of consciousness alteration (Dittrich, 1998; Studerus, Gamma & Vollweider, 2010). Although this validated scale offers a comprehensive evaluation of drug-induced experiences, it is not satisfactory to efficiently gauge the single dimension of ego dissolution (Nour et al. (2016). However, if the field of ego-disturbances through psychedelic drugs is to prevail in future, it necessitates a simple and quick measure of the ego-dissolution dimension.

In this context, Nour et al. (2016) introduced the Ego-Dissolution Inventory (EDI) based on individuals' subjective experiences under psychedelic drugs (Nour et al., 2016). The EDI includes a total of 16 items which capture ego-dissolution and the opposing construct called ego-inflation also titled as self-assuredness. Within the factor of ego-dissolution, items pose statements such as 'I experienced a dissolution of my 'self' or 'ego'' in which individuals have to respond on a visual analog scale (VAS) with 0 defined as 'No, not more than usually' and 100 defined as 'Yes, entirely or completely'. Further, 8 items of ego-inflation such as 'My ego felt inflated' on the same VAS are included. The scores are calculated

as the sum across items for both factors separately and as a total sum across all items. The statistical analyses show internal consistency and construct validity of the EDI and convergent validity with the well-established Mystical Experiences Questionnaire (MEQ) (Barrett, Johnsons & Griffiths, 2015; MacLean, Leoutsakos, Johnson & Griffiths, 2012). Moreover, it exhibits a significant positive correlation with drug dose and experience intensity for the ego-dissolution and -inflation subscale for psychedelics and cocaine respectively (Nour et al. 2016). These findings argue for a dose-response and intensity-response relationship in psychedelic drug use. Arguably, a higher dose and higher subjective intensity correlates with stronger ego-dissolving experiences. On the contrary, larger cocaine drug doses are associated with increasing ego-inflation, whereas alcohol does not show such trends. Lastly, both cocaine and alcohol seem to be positively associated with subjective intensity and ego-inflation. All in all, ego-dissolution is significantly explained by experience intensity and drug class (Nour et al., 2016).

To sum up, the EDI captures the construct of ego-inflation as an antithetical concept of ego-dissolution. In contrast, the ASC refers to positively ego-dissolution experiences as oceanic boundlessness reported by drug users as pleasant mystical experience. Therefore, these two concepts are related to ego-dissolution in different ways: ego-inflation as an antagonistic factor and oceanic boundlessness diverging in the subjective level of valence.

Interestingly, the conceptual dissociation between positive (oceanic boundlessness) and negative ego-dissolution can be demonstrated by looking into recent neurophysiological findings. Neurobiological studies

using multimodal brain imaging after a dose of psilocybin revealed different anatomical and functional patterns depending on a positively or negatively experienced ego-dissolution. According to studies, glutamate release within 5-HT2 receptors plays a central role in stimulating and maintaining primary effects of psychedelics. It is hypothesized that prefrontal cortex cell activation depends on the level of glutamate release through 5-HT2 receptors, fostering the psychedelic experience (Aghajanian & Marek, 1997; Aghajanian & Marek 1999; Mason et al., 2020; Vollenweider & Kometer, 2010). To be more precise, a study by Mason et al. (2020), suggests that glutamate levels in brain regions such as the medial prefrontal cortex (mPFC) and the hippocampus play a key role in initiating subjective experiences of positive and negative ego-dissolution. Both of these regions overlap with the large-scale brain network called the Default Mode Network (DMN), which is implicated in self-referential mental tasks and thought to show heightened activation during psychedelic experiences. The findings demonstrate that negatively experienced ego-dissolution correlates strongly with an increase in glutamate within the mPFC, whereas a positively experienced ego-dissolution termed oceanic boundlessness suggests a diminution of glutamate in hippocampal regions. The questionnaires used to assess different aspects of the psychedelic experience were the ASC and EDI. Besides the fact that these preliminary findings show regional dissociation of subjective experiences of psychedelic drugs, they also emphasize the importance and need for standardized, validated scales for different types of ego-dissolution. Taken together, the ED-inventories serve as a primary tool to investigate the subjective,

phenomenological and behavioral effects that accompany neurobiological changes in the brain during psychedelic states.

Ego-dissolution and the brain

So far, the paper sought to examine the conceptual influence of psychedelia on disrupted self-binding and the relationship of distinct ego-dissolution phenomena such as oceanic boundlessness. In this context, the EDI as a standardized survey has been explored. However, it remains ambiguous which neural correlates are linked to these processes and how brain regions behave in reaction to psychedelics. Therefore, it is crucial to associate brain regions with the experience of a dissolved ego under psychedelics.

In regard to psychedelic induced ego-dissolution, two large-scale brain networks have been investigated: The Salience Network (SN) and the Default Mode Network (DMN). Identifying coherent and salient information fitting into the self-model is associated with the SN of the brain, which is anchored in the anterior insula and dorsal anterior cingulate cortex (Menon, 2015). Moreover, it is involved in directing and binding emotionally salient information and is often referred to as the seat of the embodied self (Letheby & Gerrans, 2017). In contrast, the DMN which is rooted in the posterior cingulate cortex and the medial prefrontal cortex, is suggested to be responsible for the narrative, egocentric self. Besides a heightened activation during self-referential tasks, it is also implicated in self- and other-judgements (Lou et al., 2004; van Buuren, Gladwin, Zandbelt, Kahn, & Vink, 2010). Because of their distinct association with self-perceptive processes, these brain networks are currently under investigation in relation to psychedelic ego-dissolving experiences.

First attempts have been made to study the self-concept via advanced brain imaging. A study by Tagliazucchi et al. (2016) investigated the communication between large scale networks after an LSD administration through fMRI. This neuroimaging measure captures the oxygenated blood of the brain (BOLD). The results show an enhancing effect of LSD on the global and between-module communication within the brain, while ceasing the integrity of individual modules. This means that single large-scale networks are more interconnected compared to a sober state of the brain. This is a coherent finding with earlier studies on global brain communication under psychedelics (Carhart-Harris et al., 2014; Muthukumaraswamy et al., 2013). This research utilized the overall functional connectivity density (FCD), a measure of average correlation between fluctuating BOLD signals of regions in relation to others. Herein, high values in a region coincide with a strong connectivity with the rest of the brain. Generally, under the influence of LSD the resting state networks such as the DMN and SN were identified as major hubs using this analysis. Also, the high FCD value corresponded and overlapped with an enlarged distribution of 5-HT2 receptors in these regions. The modulatory role of the 5-HT2 receptors leads back to the effect psychedelics have on the self-perception and to the findings of positively and negatively experienced ego-dissolution (Mason et al., 2020). This study revealed that the bilateral temporo-parietal junction (angular gyrus), which is a part of the DMN and the bilateral insular cortex corresponding to a subpart of the SN, correlated with intensity of subjective ego-dissolution (Tagliazucchi et al., 2016). It has been further suggested that the angular gyrus is typically involved in out-of-body experiences which occur during ego-dissolution (Blanke et al.,

2002). In addition, the insular cortex is commonly referred to as the seat of self-awareness and the main processor of emotional information. Both of these findings are significantly associated with ego-dissolution (Tagliazucchi et al., 2016). Lastly, the study hypothesized that the increase in global connectivity of higher-level regions such as the DMN and SN particularly involve sensory areas. This enhanced communication of association and sensory cortices might represent a collapse of the hierarchical organization compared to a normal state of the brain. Accordingly, such collapse leads to the experience of blurred ego-boundaries and ultimately to an ego-dissolution. Taken together, this study informs our understanding on brain mechanisms of psychedelic ego-dissolution.

Further, converging evidence from Carhart-Harris et al. (2016) support the relationship of the DMN integrity and profound ego-dissolution experiences. Results show a replication of a disintegrated DMN which correlated with acute ratings of LSD-induced ego-dissolution by means of FCD. However, the study also found a relationship between decoupled parahippcampal gyrus resting state activity (PH-RSC) and psychedelic ego-dissolution assessed by the ASC, revealing another possible neurobiological correlate of psychedelic-induced ego-dissolution (Carhart-Harris et al., 2016). Taken together, the preservation of the DMN and PH-RSC integrity may contribute significantly to the perception of the self and its abolishment shaping deep psychedelic experiences.

Another recent work by Lebedev et al. (2015) investigates the effect of psilocybin-induced ego-dissolution on the Salience Network and the Medial Temporal Lobe (MTL). The MTL with its key PHC area is implicated

in contextual processing, associative memory and is commonly referred to as the mediating link between the DMN and the SN (Aminoff et al., 2013). The study identifies a disruption of the MTL communication with neocortical areas such as the SN, associated with the drug-induced ego-dissolving phenomenon. In addition, Lebedev et al. (2015) observed a decreased SN integrity under psilocybin, which was significantly related to the ego-dissolution phenomenon. Furthermore, the study displayed a disconnected crosstalk between PHC regions and the DMN which was strongly associated with the experience of ego-dissolution and consistent with previous work (Carhart-Harris, 2016; Carhart-Harris & Friston, 2010). Above all, this research reports results about the aberrant role of interhemispheric communication found during ego-dissolution. Incoherent functioning between the two hemispheres contributes to the maintenance of an aberrant sense of self, especially in regions such as the medial temporal lobe under a psilocybin dose (Lebedev et al., 2015). These results highlight the role of the PHC, the DMN and SN in the neurobiological framework of psychedelic ego-dissolution. The PHC is thought to play a key role in the communication with the DMN and research in which stimulation of the MTL circuitry has been performed, lead to dreamy states and depersonalization-like experiences (Bancaud, Brunet-Bourgin, Chauvel & Halgren, 1994; Bartolomei et al., 2012; Lee & Axmacher, 2013) In addition, altered states of self-awareness as it is experienced in patients suffering from psychosis, has been linked to the aberrant function of the MTL (Lambert et al., 2002; Lemche et al., 2013). Further, the SN and related components represent neural correlates of self-awareness and are thought to be the seed of the embodied self (Craig &

Craig, 2009; Seth, 2013). In summary, this research affirms ceased SN integrity, decreased inter-hemispheric communication and MTL-neocortex disintegration under psilocybin promoting a state of ego-disturbance.

At last, a study by Carhart-Harris et al. (2013) analyzed the role of orthogonality (inverse coupling) between resting-state networks such as the DMN and the task-positive network (TPN). In this experiment, subjects under psilocybin exhibited an abolishment of orthogonal DMN-TPN functional connectivity which is thought to be characteristic of the psychedelic state (Carhart-Harris et al., 2013). Interestingly, along with a reduced DMN-TPN inverse coupling, results exhibited a preservation of thalamocortical connectivity. This is especially indicative of a confused state of consciousness, since this was not found in a study using a propofol sedative (Boveroux et al., 2010). The DMN as previously stated is implicated in introspection and exploratory thoughts, whereas the TPN is associated with focused attention. Usually, both DMN and TPN show a dissociated activity pattern especially during task performance (Fox et al., 2005). If, however, these fundamental processes are unified, a confusion of consciousness states may result. Such an increase in coupling is seen in experienced meditators and introduces a disturbed state of cognitions observed in high-risk schizophrenia patients (Brewer et al., 2011; Shim et al., 2010). Therefore, suppressed orthogonality by means of the DMN-TPN coupling and preserved thalamocortical connectivity are indicative of psychedelic induced consciousness alterations. This model is a neurobiological hallmark for psychedelics and ego-dissolution experiences.

All in all, the integrative role of the DMN/DMN-TPN connectivity and the SN as well as cortical decoupling of the MTL, enhanced interhemispheric and global network communication are crucial contributors in the experience of ego-dissolution. Further, ongoing brain research with regards to psychedelia-induced ego-dissolution shed light onto a novel stream of research, which will be helpful in treating self-distorted mental disorders. Under the assumption that positive ego-dissolution in mystical practices is closely associated with self-distortions in psychopathologies, treatments may target ego-dissolution. These first neurological correlation studies are a tentative draft to define the neurobiological basis of ego-dissolution exploratively. In the future, controlled studies should involve investigations of the specific functionality of large-scale brain networks. In the context of psychedelia these networks could be inspected closely, hereby defining regions implicated in positive and negative ego-dissolving experiences. In these cases, studies will be able to determine long-term effects of psychedelic treatments in depression or psychosis. This potential notwithstanding, the studies used are primarily correlational studies, therefore no causality should be drawn between the investigated regions and experiencing ego-dissolution.

DISCUSSION

In this literature review, the research question of how psychedelics influence the sense of self has been addressed. The paper sought to explore current scientific findings on brain correlates of ED and psychometric properties of a standardized ED questionnaire. The theoretical ideas of the

self with its psychedelic ego-dissolution, as well as the EDI and the neural correlates, revealed mixed results. From a theoretical standpoint, the self can be defined according to Letheby & Gerrans (2017) with the help of the binding process, whereas ego-dissolution is a course where the coherent self is distorted, and loss of ego-boundaries are experienced. People generally seek for information that is salient and coherent with their self-model. This binding process is presumably mediated via two large-scale networks: the DMN and the SN. FMRI research suggested this mediating role, showing that disintegration and alterations of the activity in these networks are a crucial contributor in ego-loss (Tagliazucchi et al., 2016). Furthermore, global communication and interhemispheric connection give additional proof that neural processes influence the experience of ego-dissolution. To conclude, the changes in diverse brain regions and processes lead to a breakdown of the normal hierarchical organization of the mind with regard to psychedelic experiences such as altered perception of the self (Lebedev et al., 2015).

This research is an essential step towards a more detailed comprehension of the self and its related concepts. The investigations are first attempts to conceptualize the self and its components. In future upcoming studies, subjects who suffer from self-concept disturbances, e.g. psychosis, should be included in order to compare such conditions and its related brain areas. Negatively experienced ego-distortions should be researched in near future, because ego-dissolutions are not necessarily unpleasant experiences but can lead to the suspension of dysfunctional self-patterns. Hereby, existing therapeutic interventions in psychosis and depression, where patients suffer from aberrant self-representations, will

profit from future lines of ED research. Also, the examination of large-scale networks such as the DMN could reveal more implicated features of these regions, which fosters the understanding of brain-self-concept interactions. Besides the already known functions of the DMN in self-referential activities, the network is correlated with the experience of a dispersed self, demonstrating additional roles of such networks. For instance, studies showed that an induction of dexamphetamine reduced connectivity of large resting state networks such as the DMN and SN, establishing convergent validity ongoing drug research (Schrantee et al., 2016). In the following years, neurobiological research should examine the specifics of psychedelia-induced ego-dissolution. Furthermore, studies should comprise scientific comparisons with other drugs, for instance cocaine, in order to disentangle the opposite of a dissolved ego, namely ego-inflation. This could help to yield a clearer and more concise overview of the diverse elements of the self. For this reason, the concept of ego-dissolution necessitates assessments through accurate inventories. The EDI is a comprehensive inventory addressing this concept and future investigations should implement and use this detailed questionnaire, therewith facilitating analyses.

Nonetheless, some limitations need to be mentioned. First, the self-model proved to be a complicated intertwined concept with different constituents connected to it. In this context, processes like binding and predictive processing are elaborated components of the model, but other additional concepts need to be mentioned. It also incorporates self-knowledge and identity, as well as cultural differences that can be found when comparing individuals. These points should be taken into

consideration; therefore, these studies give only limited information about the self as a whole. Second, current research showed only correlational results, so nothing can be stated about causal relations between ego-dissolution and the brain areas. To establish stronger correlations in the future and to obtain insights into brain areas, a variety of brain imaging technologies and questionnaires have to be used interchangeably. Third, several psychedelics should be used for the purpose of settling differences between the ego-effects. Knowing the action mechanisms of psychedelics in combination with a hazard-free ego-dissolution experience can lead to the solving of pathological self-models. This should be helpful for integrating psychedelics in future therapeutic contexts.

Conclusion

The research question '*How do psychedelic drugs influence the sense of self*' has been interpreted by looking conceptually at the various effect mainly psilocybin and LSD have on the bound self-concept, by means of indicative neural evidence underlying this process. All in all, this review dealt with the conceptualization of the self and elaborated on the process of ego-dissolution. By investigating large-scale networks via brain imaging studies essential neural underpinnings of self-distortions can be found, occurring under influence of psychedelic drugs. However, future research should include patients suffering from disorders related to the self, as well as comparisons to other drugs in order to yield a sophisticated view on the concept of the self.

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This paper is the product of students from the Faculty of Psychology and Neuroscience, Maastricht University and is meant for student educational purposes only.

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Bulletproof Coffee and Cognition: A Double-blind, Placebo-controlled Study on the Effects on Working Memory

Original Article

There is evidence that caffeine has positive effects on cognition. Studies have shown that caffeine is an indirect enhancer of cognitive functions such as memory, concentration, and mood. A new caffeine-based trend has emerged, claiming "massive impact on energy and cognitive function." This new beverage, known as Bulletproof coffee, is a combination of coffee, grass-fed butter, and medium-chain triglycerides (MCT) oil. Thus far, these claims have not been supported by any scientific evidence. This research aimed to determine whether these enhancing effects could be corroborated by empirical data, by using a double-blind within-subject design. We hypothesized that participants in the Bulletproof condition would perform better on memory-related

tasks and would score higher on subjective mood ratings. 21 participants performed two working memory-related tasks after consuming a coffee beverage (either decaffeinated coffee, regular coffee, or Bulletproof coffee). Subjective mood ratings were collected before and after coffee consumption as a secondary measure. Results did not show an effect of Bulletproof coffee on cognitive performance during working-memory related tasks. A significant effect was found on subjective measures of mood. Contrary to expectations, participants reported an increase in alertness after consumption of the placebo drink and a decrease in alertness after consumption of regular coffee. However, this finding likely represents a measurement artifact. Further research needs to be conducted to gain more conclusive results.

Keywords: bulletproof coffee, working memory, caffeine, cognitive enhancement, mood

INTRODUCTION

In modern society and working environments, people face enormous pressures to excel. Consequently, there is a growing interest in enhancing human performance (Reissig, Strain, & Griffiths, 2009). Therefore, an increasing number of healthy people are looking for ways to safely enhance their cognition as they seek to increase their learning capacities and abilities. One possibility that is considered safe for cognitive enhancement is the ingestion of caffeine. Caffeine is the most widely used and accepted psychoactive drug in the world (Lyvers, Brooks, & Matica, 2004). It is consumed in many forms (e.g. beverages, food, medication) and readily available on the market. Caffeine is considered a stimulant as it has arousing effects on the central nervous system (CNS), for instance increasing vigilance and alertness (Franke, Christmann, Bonertz, Fellgiebel, Huss, & Lieb, 2011). According to Nehlig (2010), caffeine should be regarded as an indirect cognition enhancer as its positive effects are mediated by mood, arousal levels, and concentration. Specifically, Nehlig (2010) identifies caffeine-facilitating effects on learning in tasks where information is presented passively rather than in tasks in which material is learned intentionally. Most studies however, found improvements on reaction time (Nehlig, 2010; Haskell-Ramsay et al., 2018). Furthermore, cognitive performance was found to be improved in a range of 37.5 to 450 mg of caffeine, which resembles the normal range of moderate coffee drinkers (Nehlig, 2010; Ruxton, 2008).

While there have been many studies focusing on the effects of caffeine on cognitive performance there is no research regarding the

stimulating effects of a beverage called 'Bulletproof coffee'. The creator, and main proponent of Bulletproof coffee, Dave Asprey, designed a rather uncommon mixture of regular coffee, "brain octane" oil, and grass-fed butter, claiming it to have numerous advantageous effects on cognition ("Official Bulletproof Coffee Recipe", 2019). "Brain octane" oil is a purified form of medium-chain triglycerides (MCT) oil, which is derived from coconut oil. The advertisement of this fashionable beverage promises a "massive impact on your energy and cognitive function" ("Official Bulletproof Coffee Recipe", 2019), yet there is no scientific evidence to support these claims. As previous studies have postulated that caffeine, as well as MCT oil, have been shown to improve cognition (Page, Williamson, Yu, McNay, Dzuira, McCrimmon, & Sherwin, 2009), the question is raised whether this combination has additional cognitive enhancing effects compared to regular coffee.

Since cognition covers a wide range of capabilities that are of increasing importance in modern society, its different domains are continuously being studied. Cognition involves memory, attention and perception among other domains which themselves can be further subdivided into complex aspects (Nehlig, 2010). For example, memory can be broken down into short-term, long-term and working memory (Nehlig, 2010; Haskell-Ramsay et al., 2018). To illustrate the effects of caffeine on cognition we decided to assess working memory since it is considered to be associated with information processing, executive function, problem solving, comprehension and learning (Cowan, 2013). Working memory is a limited capacity system that enables maintaining and manipulating information temporarily in order to guide and execute complex cognitive

tasks (Nehlig, 2010). Since many of the aforementioned studies found significant effects for coffee on multiple cognitive domains, we expect to also find an effect of coffee on working memory.

Since there is no scientific evidence to support the promised cognitive boost of Bulletproof coffee, as advertised on the website, we want to investigate this in our study by using working memory as our primary measurement. We hypothesized that participants in the Bulletproof coffee condition will perform better on cognitive tasks compared to the regular coffee condition, and placebo condition. Additionally, we hypothesized that the regular coffee group will perform better than the placebo condition. For the secondary measurement of mood, we hypothesized that the Bulletproof coffee condition will yield greater scores on positive mood dimensions (alertness, and contentedness).

METHODS

Participants

We recruited 22 second year bachelor students from Maastricht University via the online system SONA to participate in our study on Bulletproof coffee and memory-related performance. The SONA system is an online platform that allows researchers to advertise their studies and recruit participants. Participants can sign up anonymously for time slots and are rewarded in the form of SONA credits corresponding to the time spent participating in a certain study. In our study, the students were compensated with five SONA credits overall. Prior to the experiment, prospective participants were instructed to fill out a pre-screening form. In

order to be included in the study, participants had to be aged between 18 and 40 and moderate coffee consumers (1-4 cups per day). This criterion was necessary, to make sure that participants are approximately equally sensitive to the effects of caffeine. Participants were excluded if they were over the age of 40, pregnant, or had a Body Mass Index outside the range of 18.5-28.0 kg/m^2. Students following a vegan diet or who were lactose intolerant were also excluded, as all three beverages (Bulletproof coffee, regular coffee, decaffeinated coffee) were prepared with at least a hint of grass-fed butter. Further, individuals currently on medication or with a history of mental illness were restricted from participating in this study. In addition, students who took part in the study "Do you like coffee?" were prohibited from participating due to the similarity in research designs, which could bias participants, and therefore influence interpretation of the results. Furthermore, participants were requested to sleep at least six hours the night before testing. We asked participants to abstain from consuming caffeine 12 hours prior to testing and to abstain from alcohol and other drugs 24 hours before. In addition, participants were asked to have a so-called "light breakfast" (maximum 4 slices of bread) which had to be consumed at least two hours before testing. Due to violations of one of the aforementioned criteria, one participant had to be excluded from the study. Therefore, data of in total 21 participants were analyzed. Before participating in the study every student gave written informed consent. The study was approved by the Maastricht's University Ethics Review Committee (ERCPN; ERCPN-Nr.: RP2027_2019_30).

Design and treatment

In order to test whether Bulletproof coffee has an effect on working-memory related performance and mood, a double-blind within-subject design was used. The three treatment conditions were (1) decaffeinated coffee, (2) regular coffee and (3) Bulletproof coffee. Each participant was tested three times; receiving treatment in a counterbalanced order. To control for possible carry-over effects from the cognitive tests, testing-days were separated by a wash-out period of at least four to five days. This time window even exceeds the length of wash-out periods in previous studies (Childs & DeWit, 2006) and can therefore be regarded as a reliable way to prevent possible carry-over effects. All testing sessions were scheduled in the morning (08:30 am - 01:00 pm). To minimize random noise, all three testing sessions took place at the same time. If this was not possible, participants could deviate by one time slot which equated approximately 45 minutes deviation from the original time slot. The coffee conditions were prepared fresh on location just before administration. Bulletproof coffee and coffee were brewed using Senseo "Dark roast" pads (caffeine concentration 70-90mg/100ml) and coffee placebo was brewed using "Decaffeinated" pads (caffeine concentration 3mg/100ml). By taking the average of 70 and 90 mg caffeine (80+/-10mg/100ml) we calculated a caffeine range of 190+/-10 mg /237ml as caffeine concentration used in the experimental conditions. This caffeine concentration was selected based on previous research on the effects of caffeine and cognitive performance (Ruxton, 2008).

Since Bulletproof coffee is prepared by adding one tablespoon of grass-fed butter and one tablespoon of MCT oil to the coffee (Official

Bulletproof Coffee Recipe, 2019) it differs in taste and appearance from the other two conditions. Therefore, beverages were administered orally in an opaque container. Moreover, ½ teaspoon of grass-fed butter was added to the regular coffee and decaffeinated coffee condition to mask the taste as well as the appearance. The addition of butter to the placebo and regular coffee condition is believed to be small enough to not have a significant impact on cognitive performance. Further, there is no evidence to suggest that the combination of butter and coffee is responsible for enhanced cognitive effects but more so the joint combination of MCT oil, coffee, and butter.

For the first test day, participants performed a practice version of the Spatial Memory Task (SMT) in order to familiarize themselves with this task. This practice version was administered during the 45 minutes waiting period (Figure 1). Subjective mood was assessed with the Bond & Lader (B&L) questionnaire which was administered before receiving the beverage and after testing had been completed (Bond & Lader, 1974). Spatial- and working- memory were assessed 45 minutes after coffee administration as coffee shows cognitively enhancing effects approximately 45-90 minutes after consumption (Nehling, 2010). The participants started with the immediate version of the SMT directly followed by the N-back task. A 30 minutes waiting period between the immediate and the delayed STM task was needed, therefore participants had a second waiting time of about seven minutes before completing the testing with the delayed SMT task (Figure 1). The total testing time was therefore approximately 96 minutes per session.

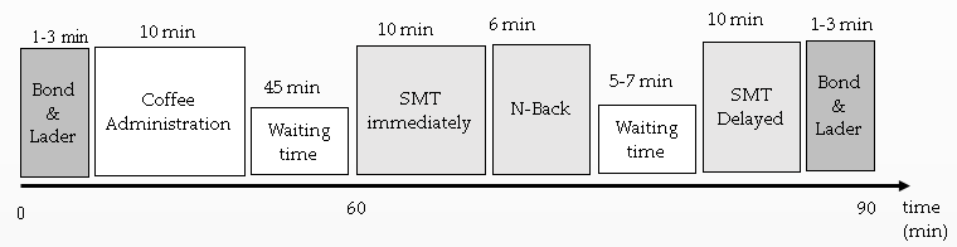

Figure 1. Schematic Overview of the Testing Procedure.

Neurocognitive assessment

The primary aim of this study was to test whether participants in the Bulletproof coffee condition perform better on spatial and working memory tasks than people in the regular and decaffeinated coffee condition. Spatial memory was assessed using the spatial memory task (SMT) which is derived from an object relocation test and consists of an immediate and delayed relocation phase. This task was chosen based on previous research demonstrating significant effects of psychostimulants on spatial memory (de Sousa Fernandes Perna et al., 2016). The immediate SMT consists of six trials, in which ten black and white pictures are presented on different locations on a computer screen (Figure 2). The participants had to remember these locations. After every trial, the pictures reappeared one by one in the middle of the screen followed by the presentation of a '1' and a '2' in different locations. If they opted for number 1, they had to press the z-key, and if they chose number 2 they had to press the m-key on the "QWERTY" computer keyboard. For the delayed relocation performance, the same pictures reappeared in a random order in the middle of the screen, and participants again had to indicate the

correct location by deciding between two given alternatives. The dependent variables of the SMT are quantitative scores: The Immediate Relocation Score (IRS), mean Immediate Reaction Time (mIRT), Delayed Relocation Score (DRS) and mean Delayed Reaction Time (mDRT) (de Sousa Fernandes Perna et al. 2016).

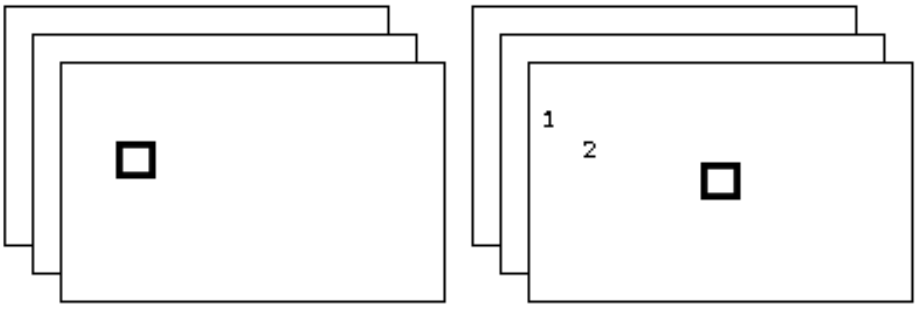

Figure 2. Spatial Memory Task. Participants have to remember the location of black and white pictures appearing at different locations of the screen (left). Subsequently, participants indicate the previous location of the pictures by deciding between two given alternatives (right).

Working memory performance was assessed with the N -back task. Earlier research gave sufficient evidence that this task is sensitive to stimulant drugs (Mattay et al., 2000) and reliably activates the dorsolateral prefrontal cortex (dlPFC), which is believed to be the brain area most implicated in working memory (Van Ruitenbeek, Hernaus, Dennis, Mehta, & Mitul, 2018). Participants were presented with blocks composed of 14 letters. The letters were presented successively on a computer screen each for a duration of 2 seconds. Target stimuli had to be identified by pressing the 3-key, and non-target stimuli by pressing the "z"-key. A target was defined as either the letter X in the 0-back condition or if the presented

letter was identical to the one 2 letters before (i.e., A-B-A) in the 2-back condition (Figure 3). The two conditions 0-back and 2-back appeared in a random order. The participants were required to respond as quickly and accurately as possible. The dependent variables of the N-back task are quantitative scores: average reaction time and number of correct responses (Van Ruitenbeek, 2018).

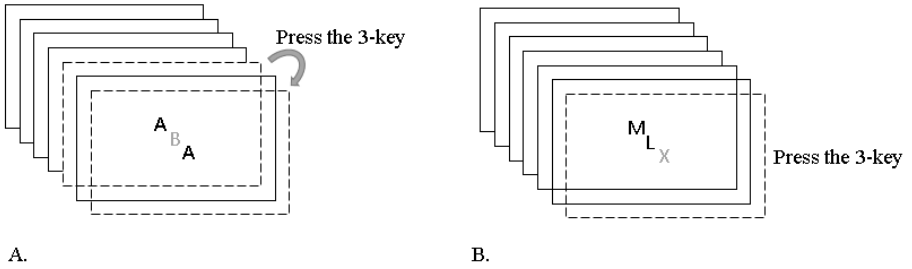

Figure 3. N-Back Task. A. 2-back Task: Participants have to indicate whether the present letter is the same as the letter that appeared two times before it by pressing the 3-key. B. 0-back Task: The participants have to indicate whether an X appears.

Subjective assessment

The secondary interest of the current study was to assess whether the participants' subjective mood was dependent on the different conditions. This was carried out by means of the B&L questionnaire consisting of 16 visual analogue scales (VAS) for subjective feelings. As opposed to normal Likert Scales which includes numbers, participants had to specify their current mood state on a scale between two given mood dimensions (e.g. calm vs. excited). The B&L VAS has been proven to be effective in evaluating alertness, contentedness, and calmness and can therefore be used as a reliable measurement for the dependent variable subjective mood

(Bond & Lader, 1974; Van Ruitenbeek et al., 2018). To increase efficiency, we transferred the original questionnaire to the QualtricsXM software, an online questionnaire platform, which facilitated the participant's access to the questionnaire and stored the data anonymously.

Statistics

Data were analyzed using a general linear model (GLM) repeated measures analysis of variance (ANOVA). Overall, seven analyses were conducted. For the SMT and the N-Back task, Accuracy and Reaction Time was analyzed separately. SMT scores were analyzed with a 3x2 factorial design with main factors Coffee condition (Bulletproof Coffee, Regular Coffee, Placebo) and Delay (Immediate Recall and Delayed Recall). N-back scores were analyzed with a GLM repeated measures ANOVA with main factor Coffee condition. B&L mood questionnaire ratings were analyzed on three different dimensions (Alertness, Contentedness, Calmness) separately using a 3x2 factorial design with main factors Coffee condition and Time point (prior and after coffee consumption). In cases where sphericity was violated, the Greenhouse Geisser epsilon correction was used. Given the small sample size of n=21, possible violations of the normality assumptions must be considered. The data was examined carefully and the distribution of scores was found to be approximately normal, thus a repeated-measures ANOVA could be validly applied. The alpha criterion significance level was set at α=0.05. All statistical analyses were conducted with SPSS version 24.0.

RESULTS

Spatial Memory Task (SMT)

GLM analyses revealed no significant difference in SMT performance- neither Accuracy nor Reaction Time between the experimental conditions (Bulletproof Coffee, Regular Coffee, Placebo). There was a significant difference in performance- for both Accuracy and Reaction Time between Immediate recall and Delayed recall task (Acc: p=0,024; Cohen´s d=1,05; RT: p=0,000; Cohen´s d= 0,12). As expected, performance in the Delayed recall task decreased compared to Immediate recall performance. Follow-up analyses comparing each of the experimental conditions separately (paired sample t-test, Bonferroni correction applied) revealed no significant difference in SMT performance (Accuracy and Reaction Time).

N-Back task

GLM analyses revealed no significant difference in performance in the N-back task between experimental conditions. No significant effect of bulletproof coffee on Accuracy and Reaction Time was found. Follow-up analyses comparing each of the experimental conditions separately revealed no significant difference in N-back performance (Accuracy and Reaction Time).

Bond & Lader Mood Questionnaire

Effects on three mood dimensions were assessed: Alertness, Contentedness and Calmness. No significant main effect of the Coffee condition on any of

the three mood dimensions was found. However, GLM analyses revealed a significant interaction effect between Coffee condition and Time point (pre and post-test) with p=0,038 (Figure 4). Subjective mood ratings indicate that Alertness increased significantly after consumption of the Placebo drink, whereas a significant decrease in Alertness could be observed after consumption of Regular Coffee. Within-Subjects contrast analysis revealed a significant interaction effect for the Placebo and Regular coffee condition, but not for the Bulletproof Coffee condition.

DISCUSSION

This study investigated the effect of Bulletproof coffee on working-memory related performance using the immediate and delayed spatial memory task (SMT) and the N-back task. Additionally, it was explored whether the consumption of Bulletproof coffee was associated with changes in mood by means of the B&L Mood Rating Scale (BL-VAS - Bond & Lader VAS). The present study is one of the first randomized controlled trials that assessed the effect of Bulletproof coffee on cognitive performance. We hypothesized that adding MCT oil and butter to coffee (Bulletproof coffee) will have additional enhancing effects on memory compared to regular coffee. However, the findings of the current study, indicate that working memory performance was not affected by Bulletproof coffee. We hypothesized that adding MCT oil and butter to coffee (Bulletproof coffee) will have additional enhancing effects on memory compared to regular coffee. Surprisingly, regular coffee also did not have an effect on working memory performance.

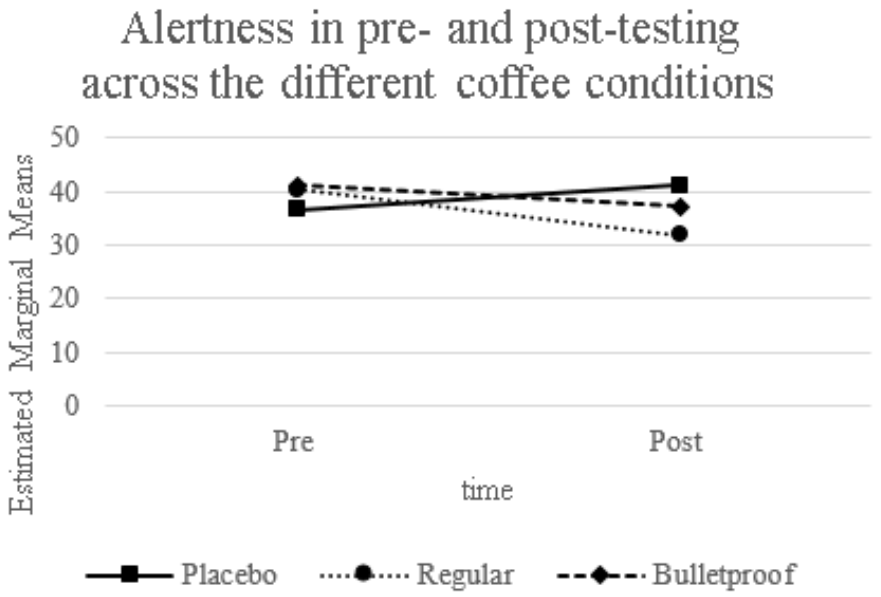

Figure 3. Interaction Effect. Graph displaying the coffee conditions on the Alertness dimension of the B&L Questionnaire. The x-axis is representing timepoint of measurement (pre-testing and post-testing). The y-axis is displaying the estimated marginal means for the different conditions. The solid line is representing the estimated marginal means in the placebo condition across pre- and post-testing, the dotted line is representing the means in the regular coffee condition and the dashed line representing the Bulletproof coffee condition.

Since regular coffee was not found to have a significant effect, it is not surprising that also Bulletproof coffee failed to significantly enhance memory-related performance. Participants that consumed Bulletproof coffee did not perform better in any of the cognitive tasks or subjective

assessments compared to the participants in the regular or decaffeinated coffee condition.

Nevertheless, we found a significant interaction between coffee type with time of measurement on feelings of alertness. Interestingly, participants in the regular coffee condition reported being more alert at the pre-measurement (before coffee intake) compared to the post-measurement (after coffee intake). Furthermore, the opposite was observed for participants in the decaffeinated coffee condition who reported being more alert at the B&L post-measurement compared to the pre-measurement. This could be explained by the peak time of the effects of caffeine which appears at 45 minutes after consumption. The B&L post-measurement was taken 95 minutes after beverage consumption. The decrease in alertness in the regular coffee condition could be explained by the decreasing arousing effects of coffee at 50 minutes past peak time. Another potential explanation for the decrease in alertness may be a result of the duration of the testing procedure, instead of the coffee beverage itself. The post-measurement for subjective mood ratings was applied after the immediate and delayed SMT, and N-back task. Mood may have been influenced by these cognitive tasks as well. Interestingly, there was no significant main effect of Bulletproof coffee on alertness. This may be due to the possibility that the addition of MCT oil and butter may have a potential influence on alertness and that Bulletproof coffee may evoke a prolonged peak of caffeine. However, this possible effect needs to be further investigated in future research.

The non-significant effect of coffee on working memory performance is in line with the controversy around the performance-

enhancing effects of coffee. Although research suggests an effect of coffee on cognitive performance, findings were inconsistent regarding the effect size and the domain of cognitive performance. Concerning, the cognitive domain, performance-enhancing effects of coffee have most frequently been reported for vigilance and alertness and less for memory (Sherman, Buckley, Baena, & Ryan, 2016).

When Nehlig (2010) investigated the effect of coffee on memory for materials presented passively and material studied intentionally he found that coffee only improved performance on passively studied material. The spatial memory task in the present study focuses on intentionally studied material and therefore the non-significant result on this task in the present study is consistent with the results found by Nehlig (2010). Also, most studies supporting the effect of coffee on memory have focused on coffee as an enhancer under suboptimal conditions, for example during a non-optimal time of the day (Nehlig, 2010). Hogervorst, Riedel, Schmitt, & Jolles (1998) found that coffee improved memory performance during distraction in a sample of middle-aged individuals. The unique enhancing effects of coffee on memory in a student population under suboptimal conditions was also demonstrated by Sherman et al. (2016). More specifically, students performed better during their non-optimal time of the day (6am-7am) when consuming normal coffee compared to decaffeinated coffee (Sherman et al., 2016). The present study investigated the effects of coffee under normal conditions- participants were well rested and no distraction or stress was induced. The lack of significant results therefore is in line with previously reported findings and the hypothesis that the cognitive-enhancing effects of coffee only emerge under suboptimal conditions.

The absence of a significant effect of coffee can also be explained by age as previous research suggests that the effect of coffee on memory performance is age dependent. Since caffeine supposedly has greater effects on elderly than the young, a possible explanation for these insignificant findings could be the limited age range of our sample (Swift & Tiplady, 1988), which only included University students. Also, it is likely that university students are, on average, more used to regular coffee consumption and thus less sensitive to the effects of caffeine than the ageing population. Also, the study by Hogervorst et al. (1998) demonstrated that coffee improves memory during distraction in middle-aged participants but not in young or old participants. This finding is also supported by Jarvis (1993) who found an effect for coffee in old but not young participants. The results of the present study are in line with the findings that old or middle-aged people are more susceptible to the memory-enhancing effect of coffee as our sample was limited to second-year bachelor students. A more heterogenous sample with regard to age would have been optimal but since the majority of young people are coffee consumers it is relevant to also focus research on this age group (Brazier, 2016).

The current study is limited by the narrow age range and size of the sample. Future studies should include larger and more varied samples to increase the power of the statistical analysis and the generalizability of the results. A varied sample should be used in order to determine if the effects of caffeine beverages are uniform across subpopulations. Moreover, there are several factors that could have confounded the results of our studies. For example, the current study did not control for gender. However,

previous studies were able to demonstrate that women might be more sensible to the effect of caffeine than men, as there seems to be an interaction between caffeine and the level of estrogen found in the female body (Arnold, Petros, Beckwith, Coons & Gorman, 1987). Consequently, we advise future research to also control for the intake of contraceptives in females. In addition, there also seems to be an interaction between smoking and caffeine on the effects of arousal (Rose & Behm, 1991). Hence, future studies should control for smoking in order to obtain more accurate results on the B&L questionnaire.

Conclusion

To conclude, the present findings fit into the controversy surrounding the enhancing effects of caffeine. No significant effects of Bulletproof coffee on cognition were found. However, this study proposes important implications for future research. More research is needed to investigate potential cognitive-enhancing effects of Bulletproof coffee, especially since this is one of the first studies on this matter so far. Future research should continue to compare Bulletproof coffee with regular and placebo coffee, to discover whether this beverage has any health and cognitive advantages over the former.

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This paper is the product of students from the Faculty of Psychology and Neuroscience, Maastricht University and is meant for student educational purposes only.

ANNA KATHARINA SELTER

Nightmares in Traumatized Children: Does Emotion Dysregulation Play a Mediating Role?

Literature Review

This review paper discusses to what extent emotion dysregulation acts as a mediator in the relationship between trauma in children and the experience of nightmares. There is evidence for an association between trauma and the experience of nightmares. The role of emotion dysregulation is discussed both from a neurological and from a psychosocial perspective. The neurological perspective concerns the key role played by the amygdala, which is hyperactive during nightmares and chronically hyperactive after trauma. The psychosocial perspective concerns the role of attachment style for the development of emotion regulation and presents theories of dream function in order to portray the role of an emotion dysregulation for post-traumatic nightmares in children. A critical review of the literature suggests that - from both the neurological and the psychosocial perspective - emotion dysregulation plays the role of a mediator between child trauma and nightmares. A model showing this interplay is hypothesized and explained. Empirical

research is needed to validate the hypothesized model and to further examine emotion regulation therapy as part of the treatment for child trauma patients who frequently experiencing post-traumatic nightmares.

Keywords: nightmares, childhood trauma, emotion dysregulation, triangular model

INTRODUCTION

Feelings of being chased, falling, being paralyzed and the death of a loved one are some of the most common contents of nightmares in humans (Schredl, 2010). It can be assumed that nearly everyone can remember at least one nightmare in their life that contained one of these situations. But what if one nightmare – which may always be the same horror scenario – disrupts one's sleep one night after another? This describes the burden frequently faced by many traumatised children (Secrist, Dalenberg & Gevirtz, 2019). This review paper hypothesizes a model for the relationship between child trauma, which might lead to the experience of nightmares, and emotion dysregulation as a mediator in this relationship.

Nightmares are frightening dreams that include terrifying, vivid phases and often lead to the awakening of people (Tanskanen, Tuomilehto, Viinamäki, Vartiainen, Lehtonen, Puska, 2001; Zadra, Pilon & Donderi, 2006). The awakening mostly happens during rapid eye movement (REM) sleep and people can clearly recall the content of the nightmare. This content mainly involves a high load of emotions of fear, but disgust and anger as well (Nielsen & Levin, 2007). Nightmares are the most prevalent form of sleep disturbances (Zadra, Pilon & Donderi, 2006). Often, nightmares and bad dreams are differentiated by the awakening criterion. Nightmares cause an awakening of the sleeper due to very frightening dreams, whereas bad dreams describe frightening dreams as well, but these do not cause awakening of the sleeper (Zadra & Donderi, 2000).

Experiencing nightmares significantly disturbs one's sleep quality (Zadra, Pilon & Donderi, 2006). The huge impact of the negative

consequences of having a disturbed sleep quality can be estimated when considering the evidence that was found for sleep quality as a major determinant of human health (Mander, Winer, Jagust & Walker, 2016). Sleep disturbances may have consequences including physical ones such as an increasing risk of developing Alzheimer's disease or cardiovascular diseases (Mander, Winer, Jagust & Walker, 2016). Furthermore, sleep disturbances heighten the likelihood of developing depression, anxiety or other psychiatric pathologies. Sleep accounts for one third of life, therefore the consequences of sleep disturbances are dramatic (Wong et al., 2013).

Most people experience bad dreams from time to time, but nightmares occur more rarely. In a sample of 10-year-old children, 3.5% stated that they experienced nightmares 'often' (Schredl, Fricke-Oerkermann, Mitschke, Wiater & Lehmkuhl, 2009). A distinction is made between idiopathic nightmares and post-traumatic nightmares. Idiopathic nightmares describe nightmares without a specific cause, whereas post-traumatic nightmares are a symptom of people suffering from post-traumatic stress disorder (PTSD) (Langston, Davis & Swopes, 2010) following a traumatic event, which is the type of nightmare focused on in this review.

The regular experience of nightmares is a symptom found especially in the population of individuals with a traumatic history (Secrist, Dalenberg & Gevirtz, 2019). A trauma arises after the exposure to a traumatic event that has a huge emotional impact and that has such power that the person is unable to handle it (Copping, Warling, Benner & Woodside, 2001). Traumatic events leading to chronic trauma with persisting symptoms may include abuse - either physical or sexual -,

(domestic) violence, death of a loved one, threats of suicide, neglect or other traumatic events (Copping, Warling, Benner & Woodside, 2001). Someone who has suffered a traumatic event may develop a PTSD, which is a pathology listed in the DSM-5 (PTSD; DSM V, American Psychiatric Association, 2013).

Trauma is especially drastic and triggers massive, possibly lifelong consequences when it happens during childhood, since the central nervous system, especially the brain, has not completed maturation yet (Copping et. al, 2001). The trauma may have adverse effects on cognition and attention as well as on impulse control and self-esteem, particularly because these functions are still in development in the early years of life (Pfefferbaum, 1997). Moreover, childhood trauma is known to cause anxieties and sleep disturbances, such as nightmares (Winje & Ulvik, 1998).

Experiencing a traumatic event overwhelmingly triggers negative emotions in the child. This consequently may disturb the children's emotion regulation. Emotion regulation describes the ability to identify all kinds of emotions and understand them, accept emotions, control one's own emotions and show appropriate emotions in all kinds of situations (Weinberg & Klonsky, 2009). An emotion dysregulation, on the other hand, describes a person's disability in these regards. It is defined as a poor understanding of emotions as well as having difficulties regulating negative emotions (McLaughlin, Hatzenbuehler, Mennin & Nolen-Hoeksema, 2011). Research found out that emotions play a major role in the experience of nightmares, since the 'dreamer' experiences a high load of negative emotions, which they are temporarily unable to downregulate (Nielsen & Lara-Carrasco, 2007).

This review paper takes the previously described three components into account and arranges them in an explanatory model: Childhood trauma, experience of nightmares and emotion dysregulation. An association between child trauma and the experience of nightmares has been found. Since research suggests that the key cause for nightmares is a failure of processing emotions (Levin & Nielsen, 2009; Schredl et al., 2008; Van der Helm et al., 2011), and since it has been established that especially children with trauma may develop emotion dysregulation (Aideuis, 2007), this review paper discusses to what extent emotion dysregulation plays a mediating role in the association between child trauma and the experience of nightmares. It is hypothesized that an emotion dysregulation is a mediating dysfunction and trigger for the symptom of nightmares, which lower the life quality of child trauma patients due to the described major impact of sleep on health. The question whether emotion dysregulation acts as a mediator in the relationship between children with trauma and the experience of nightmares, is answered from two perspectives: firstly, from the viewpoint of neurology and secondly, from the viewpoint of psychosocial mechanisms. Both the neurological and the psychosocial perspective are related to the experience of nightmares, the presence of child trauma and how emotion dysregulation factors in.

Neurological perspective on nightmares, childhood trauma and emotion dysregulation

Nightmares explained neurologically

When an individual experiences nightmares, a unique interplay between different brain areas creates a specific condition. Brain regions involved in nightmares are the medial prefrontal cortex (mPFC), the hippocampus, the

amygdala and the anterior cingulate cortex (ACC) (Nielsen & Lara-Carrasco, 2007). The amygdala responds to fear-related memory elements in the dream in an exaggerated, hyperactive manner. MPFC, hippocampal complex and ACC are substrates that have the function of reacting to the activity of the amygdala by downregulating it, but these mechanisms are disrupted during the experience of nightmares (Nielsen & Lara-Carrasco, 2007).

Levin and Nielsen (2009) assume a multilevel model for the function of dreams and the production of nightmares. It includes both neural processes and cognitive processes that take place during sleep and waking phases. These two branches – neural and cognitive – are represented as two networks: the AMPHAC for the neurophysiological processes and the AND for cognitive processes. The AMPHAC network includes the amygdala (A), the medial prefrontal cortex (MP), the hippocampus (H), and the anterior cingulate cortex (AC) and it illustrates the interconnected network of forebrain and limbic structures included in the expression and representation of emotions. The affective network dysfunction (AND) describes the system of dream-production that converts memories of fears into dream or nightmare fantasy and imagery, as for example the replaying of traumatic memories (Levin & Nielsen, 2009). Levin and Nielsen (2009) suggest that the intentional function of dreaming is the extinction of fear. In the networks mentioned above, emotions are reduced by re-processing memories. This function is exactly what is disrupted during nightmares: the intention of decreasing fear is failing which is suggested to be due to a failure of emotion regulation (Levin & Nielsen, 2009).

On the neurotransmitter level, an equivalent theory is proposed. Van der Helm, Yao, Dutt, Rao, Saletin and Walker (2011) conducted research in the depotentiation of emotional experiences during REM sleep. Dreams mainly occur during REM sleep, where a suppression of central adrenergic neurotransmitters takes place. These adrenergic neurotransmitters are incorporated in stress and arousal. In addition to that, amygdala-hippocampal networks are activated during REM sleep in order to encode salient events, and to depotentiate these. This means that previously affective events or memories are decreased in their intensity of emotional load. The adrenal neurotransmitter suppression and the amygdala-activity for depotentiation are two processes during REM sleep that have the intention to decrease the emotional load of events; therefore, if these processes do not work and if an individual experiences high next-day emotional load from dreaming (persisting feelings caused by the dream content), this may be due to an exaggerated amygdala reactivity (Van der Helm et al., 2011).

Discussing the neurophysiology of nightmares, one element seems to play a major role: the inclusion of emotions, especially via the emotion substrate amygdala. It is suggested that dreaming is a process including high emotions, often coming from emotional memories, salient events or extreme feelings. The amygdala is supposed to over-react during nightmares (Nielsen & Lara-Carrasco, 2007). Levin and Nielsen (2009) suggest the multilevel model of dreams and nightmares that includes the amygdala - which represents emotions during sleep – and discuss how the affective network dysfunction converts fears into nightmare imagery. Van der Helm et al. (2011) claim that the mechanisms normally taking place

during REM sleep involve a special role of the amygdala: to reduce the emotional load of salient events in order to reduce the emotional intensity of previously affective events (that may cause nightmares). The amygdala and the emotional load and regulation seem to play a major role in the production and experience of nightmares, supporting the hypothesis emotion dysregulation may be an indirect cause of the nightmare experience in traumatized children.

Childhood trauma explained neurologically

A trauma may cause huge consequences on and for the brain. Evidence was found that individuals suffering from PTSD have an increased amygdala responsivity during the recall of traumatic states, but during the processing of affective information and events that are unrelated to the trauma content, as well (Shin, Rauch & Pitman, 2006). In addition to the amygdala over-reacting in this way, the mPFC, which is supposed to downregulate affective arousal of the amygdala via critical thinking, has a smaller volume in PTSD patients. This leads to a hypo-responsivity of the mPFC during emotional cognitive events and tasks (Shin, Rauch & Pitman, 2006).

The amygdala is a mediator of the acquisition and expression of fear conditioning and it further increases the emotional memory (Koenigs & Grafman, 2009). The ventromedial prefrontal cortex (vmPFC), on the other hand, is intended to extinct this conditioned fear. This task of the vmPFC – to decrease emotional memory by inhibiting the amygdala– is the stage of defect in people suffering from symptoms related to trauma, which often includes nightmares (Koenigs & Grafman, 2009). Functional imaging studies found decreased activity of the mPFC and an increased and

exaggerated activity of the amygdala in patients with PTSD (Shin et al., 2004).

The neural disruptions occurring in people suffering from the post-traumatic symptom of nightmares include emotion dysregulation of negative events, as well. The vmPFC and the amygdala play a major role in this negative emotion regulation: the vmPFC is intended to suppress negative emotions triggered by the amygdala. This process is an inverse relationship: during events that trigger negative emotions, the vmPFC increases its activity, which is coupled with a decrease in the amygdala's activity (Koenigs & Grafman, 2009). This inverse relationship is comparable and somehow the same in the mechanisms of emotion regulation, as well as in the extinction of fear (lowering the extremity of emotional load) (Delgado, Nearing, Ledoux & Phelps, 2008). And this inverse relationship between vmPFC and amygdala seems to be disrupted in pathologies characterized by an increased level of negative affect, including PTSD and trauma patients in general (Koenigs & Grafman, 2009).

All in all, neural consequences of trauma mostly refer to disruptions of emotional substrates combined with disruptions of evaluative substrates (Milad, Rauch, Pitman & Quirk, 2006). On a more detailed level, researchers claim that the pathogenesis of PTSD is based on two main disruptions including the hyperactivity of the amygdala that causes high emotional distress on the one hand, and a disinhibition of the vmPFC, which should normally act in an inhibitory fashion on the amygdala (Milad, Rauch, Pitman & Quirk, 2006). This leads to the assumption that since PTSD patients intensively suffer from traumatic nightmares, the imbalance between the activity of the amygdala as the emotion regulation substrate

and the prefrontal cortex as the critical thinking substrate may as well play a major role in the general population with trauma history suffering from nightmares.

A lesion study, conducted by Koenigs and others (2008), further confirms the hypothesis that a hyperactivity of the amygdala is the major neural mechanism characteristic for symptoms of post-trauma patients. The so-called Vietnam Head Injury Study found out that Vietnam veterans that suffer from a lesion in their amygdala have a lower-than-normal likelihood of the development of PTSD. In fact, the prevalence of developing PTSD among the control conditions – consisting of a group of veterans with no brain lesion and a group of veterans with a non-amygdala lesion – was the same prevalence as found among all Vietnam veterans. But the prevalence found in this VHIS study for amygdala-lesioned veterans was zero percent. This study therefore supports the amygdala-hyperactivity hypothesis regarding the genesis of nightmares and the other PTSD symptomatologies of people who experienced major trauma (Koenigs et al., 2008).

Experiencing a traumatic event during childhood may be an amplifying factor in the extremity of consequences on the psyche and on the brain (Perry, Pollard, Baker & Vigilante, 1995). Traumatic experiences or salient events happening during the maturation of the brain determine the organization and functional status of the fully matured brain. Consequently, the unmatured brain may incorporate extreme emotional affect in its normal reactions towards stimuli, which quickly becomes pathological (Perry, Pollard, Baker & Vigilante, 1995).

The findings give arise to the assumption that trauma significantly leads to an imbalance between emotion regulatory mechanisms, which again seems to be the cause of the experience of nightmares (Milad, Rauch, Pitman & Quirk, 2006). The role of the prefrontal cortex - to downregulate emotional affection, which is happening in traumatized patients when re-living memories - is disrupted, which leads to frequent nightmare imagery. This disrupted mechanism is found to be especially present in children who have less prefrontal cortex ability to downregulate amygdala activity, due to the incomplete maturation (Perry, Pollard, Baker & Vigilante, 1995).

Emotion dysregulation explained neurologically

The amygdala is the core substrate responsible for generating negative emotional stimuli, and therefore abnormalities in the amygdala are taken as the cause for an emotional dysregulation (Donegan, et al., 2003). The hypothesis raised by the present research question, i.e., whether emotion dysregulation may play a mediating role in the relationship between child trauma and nightmares, may be confirmed based on the neuronal phenomena of nightmares, trauma and emotion dysregulation. Traumatised individuals often suffer from a decreased volume of the mPFC and experience hypersensitivity and hyperactivity of the amygdala (Shin, Rauch & Pitman, 2006). Most healthy people occasionally experience nightmares, and in these cases, the mPFC is not downregulating the emotional load of the dream content as usual. In people who experienced trauma, and especially dramatic in children who experienced trauma, this hypoactivity of the mPFC and the hyperactivity of the amygdala become chronic (Koenigs & Grafman, 2009). This is due to an overload of negative

affect stemming from the traumatic event that leads to an emotional lability and to an emotion dysregulation (Milad, Rauch, Pitman & Quirk, 2006). A child's amygdala, which is still in maturation, suffers even more from the trauma since the prefrontal cortex has not matured to a sufficient degree to downregulate emotional stimuli (Hartley & Lee, 2014). A traumatic event is such an invasive experience that it causes unhealthy reactions in the neurophysiology of the patient. It may be concluded that many children who experienced trauma suffer from an increased frequency of nightmares due to a neurologically caused emotion dysregulation occasioned by a hyperactivity of the amygdala and a hypoactivity of the mPFC.

Psychosocial perspective on nightmares, childhood trauma and emotion dysregulation

Nightmares explained psychosocially

Nightmares are most common at the age between five and ten and children in that age group experience nightmares more often than parents assume, which was found out by comparing children's and parents' questionnaires (Schredl, Fricke-Oerkermann, Mitschke, Wiater & Lehmkuhl, 2008). The production of nightmares seems to be associated with a personality that is characterized by great reactive emotional distress (Levin & Nielsen, 2009).

Factors influencing the frequency of nightmares have been found to be the experience of stressors, such as problems at school, parental divorce or traumatic events that may be sexual abuse or even the experience of wars (Schredl et al., 2008). Furthermore, children frequently

watching violent TV shows are found to experience negatively toned dreams more often (Viemeroe & Paajanen, 1992). For a child, the procedure of going to sleep already involves several stressors, including the end of social contact and darkness (Schredl, Blomeyer & Görlinger, 2000). In addition to the process of falling asleep, the awakening after sleep – in case of having experienced a nightmare – causes stress again, which further increases the frequency of nightmares, like a vicious cycle (Schredl, Blomeyer & Görlinger, 2000). The association found by Schredl et al. (2008) is in line with the other influencing factors for nightmares that have been discussed so far: the highest correlation of nightmares was found with emotional symptoms and problems which include worrying, experiencing anxieties and being nervous. Partly due to the fact that girls have higher values of trait anxiety and emotionality, it was found that girls experience nightmares more often than boys (Schredl et al., 2008). It can be concluded that events with a high emotional load lead to an increased probability of experiencing nightmares (Schredl et al., 2008).

A theory developed by Revonsuo (2000) further shows the incorporation of emotions in nightmares via describing the function of dreaming: to simulate threatening events. This Threat Simulation Theory (TST) of dreaming was inspired by the evolutionary theory: it is suggested that the original function of dreaming was an adaptive solution in order to survive life threats. Repeatedly facing these life-threatening situations via dreams may provide protection in the long run by practising and enhancing threat perception and threat avoidance (Revonsuo, 2000; Valli et al., 2006).

Theories about the function of dreams suggest that dreaming plays a facilitating role in the process and regulation of emotional distress,

triggered by emotional events in daily life (Vandekerckhove & Cluydts, 2010). A restful sleep provides the ability for an individual to cope with emotionally triggering events during the day, therefore sleep has a de-arousing and restorative function. If sleep is disturbed by an over-interpretation and over-imagery of emotional stimuli leading to nightmares, this restorative role gets lost, leading to even more distress. Sleep deprivation causes an even higher sensitivity to stressful and emotional events during the day, which again may lead to nightmares which triggers the downward spiral of sleep disturbances (Vandekerckhove & Cluydts, 2010).

Additionally, sleep disturbances in children and youth, leading to a low sleep quality, have been found to impair the development of cognitive and physical functions (Fernandez et al., 2013). This causes problems at school, a disturbed affect regulation and frustration processing and this again causes huge distress for the child. Consequently, a vicious cycle has been started including poor sleep quality, distress in the child, stress that affects the family and performance, leading to poor sleep again (Fernandez et al., 2013). This emergence of a vicious cycle again shows the major influence of emotions on sleep and dream content and underlines why children are especially prone to experience nightmares after highly emotional-enhancing events such as a traumatic event.

Childhood trauma explained psychosocially

Evidence was found that stimuli which are emotionally arousing are better remembered by individuals than stimuli that are emotionally neutral (Koenigs & Grafman, 2009). This enhancement of the emotional memory

is carried out by the hippocampus-amygdala interaction, where the amygdala's hyperactivity - due to the high emotional load - leads to an enhanced memory of the event in the hippocampus. Individuals suffering from the disorder PTSD have an extreme emotional memory enhancement and their memory of the traumatic event becomes consolidated in an excessive manner (Koenigs & Grafman, 2009). This emotional memory is easily transferred into nightmare imagery (Brewin, 2001). People suffering from trauma-related nightmares often experience a 're-living' of the traumatic event, also known as flashbacks (Brewin, 2001). The traumatic event is not only remembered as one memory as usual, but the content of the situation is accompanied by high sensory details including sounds, vivid visual imagery, smells and other sensory perceptions. The traumatic memory is emotionally charged and thus gets triggered involuntarily very easily via external or internal stimuli, which leads to flashbacks (Brewin, 2001). Valli et al. (2006) found out, that children reported a higher number of dreams containing threatening events and a higher number of threats per dream compared to non-traumatized children. These children activate the threat-simulation response and dream about the traumatic event frequently and intensively, other than children who did not experience real-live threat.

Furthermore, the extent to which a child can cope with high emotions and emotional events is assumed to be influenced by the attachment the child experiences in the setting it is brought up in (Aideuis, 2007) and evidence was found that the type of attachment determines the extremity of symptomatology following a trauma (Waldinger, Schulz, Barsky & Ahern, 2006). This finding further supports the assumption

regarding the influence of emotions on symptoms following trauma. The psychologist Bowlby hypothesized an ethological theory of attachment: He assumes that attachment derives from a biological preparation of parents for the child's future and relates to the extent to which the parents provide the child with care and protection (Leman & Bremner, 2012). The development of attachment is closely connected to and highly responsible for the emotional development of the infant (Leman & Bremner, 2012). A secure attachment – characterized by interaction, security and proximity by the parents – is responsible for the development of a healthy emotion regulation (Aideuis, 2007). This self-regulation of the child develops as a result of parents or caregivers providing a secure environment This encourages a feeling of safety and receiving help, i.e. in situations where the child is overwhelmed by its feelings, the parent or caregiver provides assistance in coping with the situation. This provides the child with the ability to regulate affects and emotional events on its own (Aideuis, 2007). If this safe environment and secure attachment style is not given, the child's development may go in the exact opposite direction. Neglect and disinterest of the parents lead to the development of a fearful and insecure attachment of the child (Leman & Bremner, 2012). Waldinger, Schulz, Barsky and Ahern (2006) found out that the attachment style the child is surrounded with, has adapted to and develops later, has a significant impact on the somatization and the extremity of symptoms in adulthood. Since the attachment style of the parents determines the emotion regulation of the child, this result further supports the hypothesis that emotion dysregulation is responsible for the high frequency of symptoms including nightmares in children after a trauma.

Emotion dysregulation explained psychosocially

Several psychosocial phenomena give rise to the assumption that emotions play a mediating role in the experience of nightmares in traumatized children. First, nightmares are in general more prevalent in children than in adults and especially emotional symptoms, such as anxiety, are significant predictors of a higher number of nightmares (Schredl et al., 2008). This higher number of nightmares in children can be attributed to the unfinished brain maturation leading to high emotional sensitivity (Perry, Pollard, Baker & Vigilante, 1995). Second, the Threat Simulation Theory of dreaming by Revonsuo (2000) suggests that dreams are a re-experience of life-threatening situations in order to heighten the perception of threats and to self-regulate the exposure to threat. Consequently, since traumatized children are exposed to real-life traumatic events, these children activate the threat-simulation response – dreaming of the event to acquire a better regulation of emotion and perception – more frequently. Others, including Vandekerckhove and Cluydts (2010) assume that dreams play the role of emotion regulators that fails during nightmares. This happens even more extremely in children suffering from PTSD, since they store their traumatic memory in an excessive manner (Koenigs & Grafman, 2009). This extreme memory is not only an exact imagery of the event, but also of other sensory perceptions which in turn increases its emotional load (Brewin, 2001). The emotional load thus plays a determining role in the frequency of nightmares in traumatized children.

The role of emotions becomes even more significant in case of a traumatic event happening in the family environment. A healthy emotion regulation develops via a secure and enhancing environment, leading to a

secure attachment of the child (Aideuis, 2007). If this is not given, but instead neglect, abuse or traumatic events take placeemotion dysregulation is the consequence, leading to the experience of nightmares in order to try to decrease the emotional load (Cook et al., 2005; Revonsuo, 2000). Furthermore, if the traumatized child grows up with an insecure attachment style, the child cannot build a healthy emotion regulation, which may be the mediating reason why children with an insecure attachment style at home experience more frequent and extreme PTSD symptoms in adulthood (Waldinger, Schulz, Barsky & Ahern, 2006).

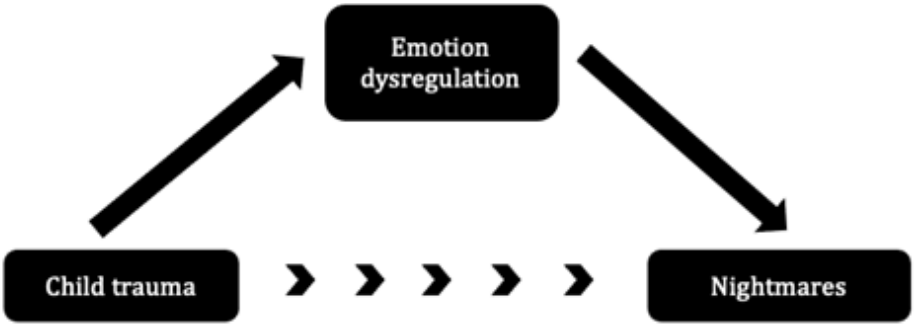

Figure 1. Hypothesized model for the causative pathway of nightmares in children with trauma history.

Modelling the relationship between nightmares, childhood trauma and emotion dysregulation

Based on the discussion about the occurrence, the mechanisms and the circumstances in which children with trauma experience nightmares, a model for the construct and origin of nightmare experience is hypothesized. Both from a neurological perspective, as well as from a

psychosocial perspective, emotion dysregulation seems to play the role of a mediator for the experience of nightmares in children with a trauma history.

The relation between childhood trauma and nightmares

Evidence was found that nightmares occur more often in children who experienced a trauma (Secrist, Dalenberg & Gevirtz, 2019). The experience of post-traumatic nightmares is currently even seen as the 'hallmark' of PTSD (Langston, 2007). From another perspective, Agargun et al. (2003) found out via interviews with undergraduates at a university that the rate of experienced childhood trauma was larger in people who suffered from nightmares.

For many children, nightmares are one of the most prominent symptoms after the experience of a traumatic event (Fernandez, DeMarni Cromer, Borntrager, Swopes, Hanson & Davis, 2013). Traumatic events cause short-term consequences, but nightmares are a symptom that has long-term and severe effects. As children who experience trauma and suffer from PTSD often have nightmares, a search for a specific term was conducted, which shows the high prevalence and common occurrence of nightmares in traumatized children (Fernandez et al., 2013). Often, they are referred to as trauma nightmares, trauma-related dreams or post-traumatic nightmares, but no agreement has been reached in this regard. The occurrence of nightmares in traumatized children is presented with a dotted arrow in Figure 1. It is to be assumed that there is some kind of link since evidence was found that nightmares are frequently occurring in children with trauma. This research paper, however, hypothesizes that this

connection might often be an indirect one (Fig. 1, dotted line), since emotion dysregulation seems to play a mediating role and act as an indirect cause for the nightmare experience.

The relation between childhood trauma and emotion dysregulation

An emotion dysregulation describes a loss of control of emotions, extremely high emotional reactions and extremely rapid changes in emotions (Bradley, Defife, Guarnaccia, Phifer, Fani, Ressler & Westen, 2011) and emotion dysregulation is suggested to have different possible causative factors. Biological factors as well as temperamental factors, that are intrinsic, may be the reason for emotion dysregulation, but exposure to stressful or chaotic situations in early life – extrinsic factors – as well (Bradley et al., 2011). These may include childhood abuse or the failure of an appropriate attachment of parents or caregivers to the child. An association was found between negative childhood experiences and adult mental health problems including depression and substance abuse. And this association is suggested to originate from the child's vulnerability to emotion regulation because of the exposure to adverse and traumatic events in their childhood (Bradley et al., 2011). Dvir et al. (2014) found an association between affect/emotion dysregulation and PTSD, where an affect dysregulation is defined as the disturbed ability to regulate negative emotional states (Dvir et al., 2014). Furthermore, children that were exposed to traumatic events have a high chance of developing developmental, psychosocial and medical impairments in the long run, and an emotion dysregulation is the core trigger that heightens the risk of

developing pathologies (Dvir et al., 2014). Due to this associations, there is a direct link between child trauma and emotion dysregulation (Figure 1).

The relation between nightmares and emotion dysregulation

Secrist, Dalenberg and Gevirtz (2019) conducted research with the aim to find factors influencing the frequency of nightmares. One of the predicting factors was a low level of emotion regulation. Emotion regulation can be measured on the basis of the individuals' heart rate variability (HRV). HRV is a psychosomatic measurement which determines how capable the individual is of rapidly adjusting to internal and external circumstances such as physiological arousal. HRV reflects oscillation amplitudes. A high complexity and a high variability determine a healthy adjustment to stressors. A low variability, on the other hand, is characteristic for lower levels of recovery of the body and psyche after the exposure to stressors. Thus, HRV is an important measure of emotion regulation capacity (Secrist, Dalenberg & Gevirtz, 2019). With the help of HRV as a measure for emotion regulation, Secrist, Dalenberg and Gevirtz found an association between low HRV, meaning low emotion regulation, and low quality of sleep in children. Furthermore, HRV was even directly associated with the experience of nightmares (in a small sample). Due to significant research that has established the connection of emotion dysregulation and experiencing nightmares, Figure 1 shows a direct arrow between the two factors.

The triangular model

An association was found for the experience of nightmares in children with a trauma history. When investigating this association more closely, emotion dysregulation seems to play the role of a mediator between these two factors, since separate connections were found between child trauma and emotion dysregulation, as well as between child trauma and emotion dysregulation. Thus, emotion dysregulation seems to be explanatory for the experience of nightmares. This assumption was supported by the neurological and psychosocial underpinnings of nightmares, trauma in children and emotion regulation – in each case both individually and in their interplay, as discussed above. Emotion dysregulation seems to play the role of a mediator since nightmares trigger huge hyperactive waves of emotions that neurologically are unable to get downregulated by prefrontal substrates and that psychosocially are unable to be coped with due to learned emotional distress since the traumatic event has happened to an unmatured brain of a child. Consequently, emotion dysregulation can be suggested to constitute a step in between the two factors of child trauma and nightmares (Figure 1).

DISCUSSION

This review discusses whether emotion dysregulation might play the role of a mediator in the relationship between child trauma and nightmares. Based both on the neurological background of nightmares and trauma and on proven psychosocial mechanisms, it may be concluded that emotion dysregulation indeed acts as a mediator. In neurological terms, the

amygdala's activation seems to represent the connecting component between trauma consequences and nightmare aetiology. This substrate for emotion processing acts as an arousal and emotional centre, which exaggerates its activity during nightmares. The emotions of fear or anxiety are represented in an excessive manner leading to a hyperarousal of the amygdala. During this process, the prefrontal cortex should normally downregulate the amygdala's activity, but this is failing during nightmares and a hypoactivity is the result. Indeed, a smaller prefrontal cortex volume was found in children with PTSD (Schredl et al., 2008). In children experiencing trauma, this amygdala hyperactivity and mPFC hypoactivity becomes chronic due to the excessively intrusive event that conditioned the activity of the substrates mentioned above and the unmatured brain which already has an imbalance between amygdala and prefrontal cortex (Nielsen & Lara-Carrasco, 2007; Levin & Nielsen, 2009; Van der Helm et al., 2011; Shin, Rauch & Pitman, 2006; Perry, Pollard, Baker & Vigilante, 1995).

In psychosocial terms, emotion regulation is learned via a secure attachment style from the parents. If this is not given, for example due to neglect or disinterest, the child cannot develop a secure emotion regulation leading to huge emotion processing problems at night. Children growing up with an insecure attachment style from the parents experience a higher somatization of the PTSD symptoms in adulthood. This includes the frequent experience of nightmares, where the brain does not process the emotionality of the dream content correctly but in an excessive fashion. Furthermore, nightmares are associated with emotional symptoms in children, and a theory hypothesizes that nightmares simulate threat perception that took place during the day, which is happening in children

exposed to trauma. Emotion regulation seems to play a major role in the aetiology of traumatized people who experience nightmares, and this major role is even more important in children experiencing trauma. When a child experiences a trauma, it is still in the development of emotion regulation and cognitive functions such as critical thinking, which further means that an extreme emotional event may disrupt the healthy development of the prefrontal cortex and amygdala. (Schredl et al., 2008; Revonsuo, 2000; Leman & Bremner, 2012; Aideuis, 2007).

On both the neurological and the psychosocial level, emotions and emotion dysregulation seem to act as a mediator in the relationship between trauma in children and their frequent experience of nightmares. Consequently, therapies for children with nightmares due to a trauma experience may be enhanced via focusing on the improvement and acquisition of a healthy emotion regulation. If a traumatized child learns how to cope with waves of different extremities of emotions, how to process emotions and how to reflect emotions, the frequency of nightmares may decrease.

Limitations and Implications

The theoretical framework and model approached in this review paper is hypothetical and hypothesized via discussing current literature. All conclusions and findings should be tested scientifically via empirical research. Empirical research is needed to test whether an emotion regulation therapy can significantly decrease the frequency and intensity of nightmares in child trauma patients. In case of a significant decrease of nightmare frequency and intensity, clinics with a population of trauma

patients may include an emotion regulation therapy, such as mindfulness-based therapy, in the treatment plan of traumatized children suffering from nightmares.

In terms of research, the hypothesized model might be strengthened via further empirical research, in order to obtain a more reliable arrow construction. In this review, there is a dotted line between child trauma and nightmares, which implies the need for further evidence as regards the possible direct connection between the two factors of child trauma and nightmares, since the literature review cannot explain the whole interplay, or confirm that emotion dysregulation is the mediator in every case, for every patient. In addition, the connection of emotion dysregulation leading to nightmares needs more empirical evidence as well, since the HRV approach has been criticised. HRV alone may not be enough to explain the occurrence of emotional distress, which emphasized the need for more evidence for this connection.

Furthermore, especially in the section about the neurological perspective, a significant part of literature was taken from adult population. The adult population studies were used for the explanation of the three components of nightmares, trauma and emotion dysregulation. In addition, literature based on studies carried out in children was used for explaining the age factor as an amplifying factor for mechanisms occurring in adulthood. Due to the focus on child population in this review, reliability for the neurological underpinnings in children may be enhanced. More research is needed regarding the neurological underpinnings of nightmares in a child population to get more reliable and direct results. In the future, functional imaging methods may be used to study amygdala and prefrontal

cortex activity in children and adolescents, maybe even in comparison to adults, to verify and further support the hypothesis of childhood as an especially susceptible age to nightmare imagery after a traumatic event due to emotion regulatory disturbances.

Additionally, literature used for this review regarding the explanation and symptoms of traumatized patients was a mixture of studies conducted in people diagnosed with PTSD and patients suffering from post-traumatic nightmares, but it is not clearly stated that they are diagnosed with PTSD. Since research in nightmares, and especially its neurological underpinnings is only a relatively new field of research, this review could not focus on only one of the two populations, but future studies may separate the two groups and may even find interesting results when comparing the two.

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This paper is the product of students from the Faculty of Psychology and Neuroscience, Maastricht University and is meant for student educational purposes only.

LIANNE WOLSINK

The Memory Effects of Simulating Crime-related Amnesia: A Review of Experimental Studies

Literature Review

Crime-related amnesia refers to the amnesia for an offense that is sometimes reported by offenders of a crime. Although some amnesia claims may be genuine, others are likely to be simulated. Simulating amnesia can have advantages for the offender, but some offenders will discontinue claiming amnesia during the police investigation. The current paper reviews experimental studies on the effects of simulating amnesia on subsequent honest memory recall. In general, simulating amnesia has a memory-undermining effect, but exact effects depend on the simulation strategy used. In line with the Memory and Deception framework, false denial is likely to induce omission errors, whereas fabrication is likely to induce commission errors. These errors are suggested to result from a lack of rehearsal and retrieval-induced forgetting, and source monitoring errors, respectively. In contrast to free recall, cued recall appears to be unaffected. Experimental characteristics, such as mock crime presentation, and legal implications are discussed.

Keywords: crime-related amnesia, simulation, memory, deception

INTRODUCTION

Crime-related amnesia refers to the amnesia for an offense that is sometimes reported by offenders of (violent) crimes. Approximately 20 to 30% of offenders of violent crimes report to have crime-related amnesia (Cima et al., 2002; Jelicic & Merckelbach, 2007). Although some amnesia claims may be genuine, others are likely to be simulated. Genuine crime-related amnesia can have organic or psychogenic causes (Cima et al., 2002). Organic amnesia results from a permanent or temporary brain dysfunction, such as traumatic brain injury or drug/alcohol intoxication, that interferes with memory encoding and causes retrograde amnesia (Cima et al., 2002; Merckelbach & Christianson, 2007). The reported amnesia needs to be proportional to the brain damage and should cause a deranged hippocampus, a brain structure crucial for memory encoding (Jelicic, 2018). Psychogenic amnesia results from an extreme level of arousal during memory encoding that is not in line with the arousal level during retrieval, the so-called state-dependent memory theory (Cima et al., 2002; Merckelbach & Christianson, 2007). However, the authenticity of this type of amnesia is often questioned (Merckelbach & Christianson, 2007). Simulation of the amnesia may be a more plausible explanation in such cases.

Simulating (or malingering, feigning) amnesia has several possible advantages for the offender, such as avoiding responsibility, hindering the police investigation, and avoiding the recall of the, often traumatic, crime (van Oorsouw & Cima, 2007). However, not all offenders will continue simulating amnesia during the police investigation. Consider for example

the case of Rudolf Hess, a Nazi politician who claimed to have amnesia for his Nazi period before and during the Second World War. When he realised, however, that he could not defend himself against the accusations, he admitted to have simulated his amnesia (Picknett et al., 2001). This raises the question whether and how simulating crime-related amnesia affects subsequent genuine memory recall of the crime. The current paper will review experimental studies on the memory effects of simulating crime-related amnesia and the suggested underlying mechanisms involved.

Simulating amnesia can be considered a form of deception. According to the Memory and Deception (MAD) framework (Otgaar & Baker, 2018), forms of deception can be placed on a continuum of required cognitive resources. From one side to the other, false denial requires the lowest level of cognitive resources, simulated amnesia somewhat more, and fabrication of an alternative story requires the highest level of cognitive resources. Of course, the latter differs between fabrication of a detail and fabrication of an entire story. The MAD framework proposes that the different forms of deception, and their levels of required cognitive resources, result in distinct memory errors (Otgaar & Baker, 2018). False denial is likely to lead to omission errors (i.e. failure to report information), whereas fabrication is likely to lead to commission errors (i.e. introduction of new information) and distortions of details. The suggested underlying mechanisms of these errors will be discussed in a later section. Simulated amnesia assumably results in a combination of omission and commission errors, depending on the used strategy. When individuals simulate amnesia mainly by denying, omission errors are more probable, whereas

commission errors are expected when individuals use fabrication as strategy.

Experimental studies on crime-related amnesia

The effects of simulating amnesia on actual memory recall have been examined in several experimental studies (Bylin, 2002; Bylin & Christianson, 2002; Christianson & Bylin, 1999; Mangiulli et al., 2018b; Mangiulli et al., 2019a; Sun et al., 2009; van Oorsouw & Merckelbach, 2004; van Oorsouw & Merckelbach, 2006). For that purpose, participants, often college students, are asked to imagine being the offender in a written or filmed mock crime or to perform a mock crime themselves, for example stealing a wallet. Afterwards, participants perform several memory tests about the mock crime on which they have to respond honestly (control condition) or as if they have amnesia (simulation condition). Usually, after about one week, participants return to the lab to perform follow-up memory tests, but now all participants are asked to respond honestly. The memory tests often consist of free recall and cued recall. Outcome measures are correctly recalled information, omission errors, and commission errors. The memory effects of simulating amnesia are the differences in memory performance between the simulation condition and the control condition during the follow-up session, when all participants respond honestly. The results of these experiments will be discussed in the following section.

The memory-undermining effect of simulating amnesia

In general, experimental studies using the previously discussed design find a memory-undermining effect of simulating amnesia on the follow-up memory tests (Bylin, 2002; Bylin & Christianson, 2002; Christianson & Bylin, 1999; Mangiulli et al., 2018b; Mangiulli et al., 2019a; Sun et al., 2009; van Oorsouw & Merckelbach, 2004; van Oorsouw & Merckelbach, 2006). Simulating amnesia specifically leads to omission errors during follow-up honest memory recall. That is, simulators recall less crime-related details than honest controls (Bylin, 2002; Bylin & Christianson, 2002; Christianson & Bylin, 1999; van Oorsouw & Merckelbach, 2004). However, simulators do not differ in terms of omission errors from participants who were only tested during the follow-up (delayed-testing only condition; Bylin & Christianson, 2002; Sun et al., 2009; van Oorsouw & Merckelbach, 2004). This finding suggests a lack of rehearsal as underlying mechanism for the memory-undermining effect in simulators (Bylin & Christianson, 2002; Sun et al., 2009; van Oorsouw & Merckelbach, 2004).

Lack of rehearsal as explanation has directly been examined in a recent experiment in which participants received reminders of the crime between the first and second memory tests (Mangiulli et al., 2019a). The memory-undermining effect of simulating amnesia diminished in terms of correct responses when simulators had to chronologically order frames of the mock crime video as reminder of the crime, compared to simulators that did not receive this reminder. In contrast to earlier studies, simulators performed better than delayed-testing only participants (Mangiulli et al., 2019a). A potential explanation for this finding is a more profound initial processing of the crime-related information by simulators, as they had to

imagine being the offender (Mangiulli et al., 2019a; Mangiulli et al., 2018b). Overall, lack of rehearsal appears to be involved in the memory-undermining effect of simulating amnesia, and reminders could help to preserve the memory for the crime, but it is probably not the only mechanism involved.

Another possible explanation for the memory-undermining effect of simulated amnesia is retrieval-induced forgetting (RIF). RIF is a process in which retrieval of a memory item leads to forgetting of another closely related memory item (Anderson et al., 1994). For example, when the word combination "fruit-banana" is practiced, the recollection of the closely related word combination "fruit-apple" deteriorates. In an experimental study on the memory effects of simulating amnesia, it was indeed found that RIF plays a role in the memory-undermining effect of simulating amnesia (Mangiulli et al., 2019b). Crime-related details that were not included in a retrieval practice were reported less than details that were included. RIF may be restricted to a simulation strategy in which simulators retrieve certain crime details, while leaving out others. Selective retrieval could thereby lead to the forgetting of other details and, thus, to omission errors during memory recall.

Simulating amnesia also leads to commission errors during the follow-up honest memory recall (Bylin, 2002; van Oorsouw & Merckelbach, 2006), but this depends on the used strategy. In particular when simulators fabricate an alternative scenario, commission errors occur (Bylin, 2002; van Oorsouw & Giesbrecht, 2008; Mangiulli et al., 2020). The longer the fabricated scenario, the more commission errors occur (van Oorsouw & Giesbrecht, 2008). Moreover, when participants are explicitly instructed to

simulate amnesia by withholding information, more omission errors occur, whereas more commission errors occur when they are instructed to simulate amnesia by distorting information (Bylin & Christianson, 2002).

According to the MAD framework, commission errors occur as a result of source monitoring errors (Otgaar & Baker, 2018). Source monitoring is the process of deciding whether a memory has an internal (e.g. thoughts, imaginations, including lies) or external (information from others, including misinformation) source. The Source Monitoring Framework (SMF; Johnson et al., 1993) states that memories of actual experiences are more rich in perceptual, contextual, and affective information than memories of imagined experiences. Individuals use this information to distinguish between memories of actual experiences and memories of imagined experiences. When, however, memories of imagined experiences are rich in information, source monitoring errors could occur, that contribute to the formation of a false memory (Otgaar & Baker, 2018). Fabrication as strategy during simulating amnesia probably leads to confusion about the source of the memory: whether it was part of the crime, or part of the fabricated story. Thereby, this strategy could lead to commission errors. In addition, external misinformation may also contribute to source monitoring errors and commission errors (both for honest controls and simulators; Mangiulli et al., 2020).

Source monitoring errors may, however, play a weaker role in commission errors after simulating amnesia than previously assumed. When fabricating an alternative story, both the actual crime and the fabricated crime should be kept in mind to ensure a consistent story over repeated interrogations. These elaborate processes may prevent confusion

over the source of memories (Mangiulli et al., 2018a). Indeed, simulators appear to be able to correctly distinguish between the actual crime and their fabricated story (Mangiulli et al., 2018a).

In contrast to earlier findings, Mangiulli and colleagues (2018b) found no memory-undermining effect on cued recall. Simulators showed an equal number of commission errors and correctly recalled information during cued recall than honest controls. Although this result is in contrast with the earlier found impairing effects on cued recall (e.g. Bylin & Christianson, 2002; van Oorsouw & Merckelbach, 2004), it is in line with retroactive interference (Bylin, 2002) that could occur when no cues are present during free recall. During the first memory tests, when participants simulate amnesia, the incomplete retrieval may interfere with the actual memory of the crime. During the follow-up memory tests, when participants have to respond honestly, they may incorrectly perceive their former memory recall as indicative of all information they can remember (Bylin, 2002). Therefore, they probably recall less information on free recall. Cues during memory recall could help to activate crime-related memories and counteract the effects of retroactive interference. To conclude, the memory-undermining effect seems to be weaker than previously shown and restricted to free recall (Mangiulli et al., 2018b).

DISCUSSION

The aim of the current paper was to examine the memory effects of simulating amnesia on the basis of experimental studies. In general, simulation of crime-related amnesia has a memory-undermining effect

(Bylin, 2002; Bylin & Christianson, 2002; Christianson & Bylin, 1999; Mangiulli et al., 2018b; Mangiulli et al., 2019a; Sun et al., 2009; van Oorsouw & Merckelbach, 2004; van Oorsouw & Merckelbach, 2006). Both omission and commission errors are reported after simulating amnesia, but this depends on the used strategy. In line with the MAD framework, false denial is more likely to induce omission errors, whereas fabrication is likely to induce commission errors (Otgaar & Baker, 2018). Suggested explanations for these errors are a lack of rehearsal and retrieval-induced forgetting for omission errors, and source monitoring errors for commission errors. However, these explanations are far from conclusive.

One-third of the participants instructed to simulate amnesia fabricate an alternative scenario (van Oorsouw & Merckelbach, 2006). Therefore, it is crucial to know whether and to what extent participants are fabricating an alternative story as strategy for simulating amnesia, because that may explain the occurrence of commission errors reported in some studies (Bylin, 2002; van Oorsouw & Merckelbach, 2006). It would be even better to instruct participants to use one specific strategy. Besides the strategies discussed, Mangiulli and colleagues (2018b) suggest to change the simulation instruction to a retrieval suppression instruction. Participants are then asked to consciously suppress memory retrieval because it causes high levels of distress. This strategy would better resemble the used strategy of actual offenders (Mangiulli et al., 2018b). Memory suppression can induce forgetting of the encoded material (e.g. Anderson et al., 2001; Stramaccia et al., 2020). This so-called suppression-induced forgetting has, however, not yet been examined in a mock crime

scenario in which participants use suppression as strategy for simulating amnesia.

The memory effects of simulating amnesia appear to be restricted to free recall, and do not occur during cued recall (Mangiulli et al., 2018b). An important difference with previous studies is the use of a mock crime video instead of a written story. Earlier studies often used written stories (e.g. Bylin, 2002; Christianson & Bylin, 1999), whereas more recent studies use mock crime videos (e.g. Mangiulli et al., 2018b). Although a mock crime video is likely to have facilitated memory encoding (Mangiulli et al., 2018b), these passive forms of mock crime presentation could limit the generalisability to real life simulated amnesia. Actually acting out (enactment) enhances memory for the act (e.g. Engelkamp, 1995), and fits better with a real life experience. Therefore, in some studies, participants performed a mock crime themselves (e.g. van Oorsouw & Merckelbach, 2004). Then, however, the mock crime paradigm may be experienced as artificial due to ethical constraints in experimental research. An interesting development in this regard is the use of virtual reality (VR) in experimental research. VR is for example used in an experimental study concerning eyewitness memory (Romeo et al., 2019). By using VR, the mock crime can be made more immersive and realistic, possibly resembling real life memory processes to a larger extent.

Characteristics of the experimental samples could also limit the generalisability of the findings. Experimental samples often consist of college students, mostly female. These participants arguably differ from actual offenders, for example in educational level and gender (e.g. Schacter, 1986). Therefore, further research should use more variable or realistic

sample, such as participants from the general population or a forensic sample. A related point is the potential mismatch between the participant's gender and the offender's gender in the mock crime story or video. This mismatch may limit the emotional involvement of the participant and affect the effectiveness of the manipulation. For example, a female participant could have more difficulty to imagine being a male offender than being a female offender in a mock crime scenario. Further research could be improved by adjusting the offender in mock crime story or video to fit the participant's gender, and perhaps also other characteristics.

Claiming amnesia is more common for violent than nonviolent crimes (Jelicic & Merckelbach, 2007). Examining the memory effects of simulating amnesia for a violent mock crime would, therefore, be more useful for forensic practice than examining these effects for a nonviolent mock crime. Still, some studies examined a nonviolent mock crime, such as stealing exam answers (Romeo et al., 2018) or drinking-related death (Sun et al., 2009), because these scenario's may be more realistic for college students. Sun and colleagues (2009) argued, for example, that a mock crime paradigm in which participants had to act as if they robbed a bar and killed someone (van Oorsouw & Merckelbach, 2004), is not realistic for college students. Yet, the participant's self-ratings of emotional impact and subjective guilt did not indicate an impaired emotional involvement (van Oorsouw & Merckelbach, 2004). Moreover, asking participants to simulate amnesia after a nonviolent mock crime, such as stealing exam answers, is not realistic either. Deciding upon an appropriate mock crime scenario is thus an ongoing point of debate.

The discussed research findings have implications for the legal field. After the first studies on the memory-undermining effect of simulating amnesia, it was argued that preventing offenders from simulating amnesia is of foremost importance because of the memory-undermining effects (e.g. van Oorsouw & Merckelbach, 2006). However, the recent study by Mangiulli and colleagues (2018b) showed a weaker memory-undermining effect, and restricted to free recall. This suggests that cues during police interrogations could be helpful to facilitate memory retrieval and to increase information gathering. Offenders appear to be able to remember many crime-related details despite having simulated amnesia. Legal practitioners could therefore consider their reports as more complete and accurate than previously assumed.

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