The creative brain
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CHAPTER 7

General Discussion
The main aim of this dissertation was to study the neural mechanisms underlying creative processes. In four empirical chapters and one theoretical chapter, I approached some of the unresolved issues regarding the creative brain from several angles. My collaborators and I focused on the potential role of the neurotransmitter dopamine in different types of creative processes and measured creativity in ADHD, a neurodevelopmental disorder that is characterized by dopaminergic imbalance. We also investigated whether and how creative processes are affected by administration of methylphenidate, a stimulant that increases levels of dopamine (and noradrenaline) in the brain. In addition, we explored the EEG dynamics that underlie different creative processes. In this final chapter, I present the main conclusions of the preceding chapters and their theoretical and practical implications. Moreover, I discuss some limitations of the current studies and propose directions for future research.

**Overview of the Findings**

In Chapter 2, I described an EEG study that my collaborators and I conducted to investigate the oscillatory dynamics that underlie rule divergent and rule convergent processes (sub-processes of the broader constructs of divergent and convergent thinking) in creative idea generation. Previous EEG studies have investigated the neural oscillations that underlie divergent and convergent processes in creativity and found that divergent (vs. convergent) thinking was associated with increased alpha activity (e.g., Fink & Benedek, 2014; Jauk et al., 2012), but several methodological issues complicate the interpretation of these findings. For example, divergent and convergent thinking were measured in separate blocks of trials in these studies, so that block-related differences in motivation and attention may have influenced findings. To increase the comparability of divergent and convergent processes during idea generation, we developed a new event-related paradigm in which participants generated new names within a certain category (e.g., martial arts), given three examples of possible new names, all ending with the same letter(s) as an implicit rule. During idea generation, participants could either follow this rule by generating a name with the same ending (i.e., *rule convergent thinking*) or deviate from the rule (i.e., *rule divergent thinking*). Moreover, participants could repeat a
certain name ending or switch between name endings from one idea generation trial to another. This new paradigm allowed us to directly compare the EEG power associated with rule divergent and rule convergent processes, as well as spontaneous, trial-to-trial switching between these thinking modes. We found that power differences in the delta band in a widespread network dissociated rule divergent from rule convergent thinking, as well as switching vs. repeating a name ending. More specifically, both rule divergence and switching between name endings were characterized by reduced delta power compared to rule convergence and repeating a name ending. These delta-band power differences correlated with behavioral indices of creative idea generation. For both rule divergence (vs. convergence) and switching (vs. repeating a name ending), the delta power difference was smaller for participants who generated more ideas during the task. Moreover, these effects were larger for participants who generated new names with more unique name endings and who switched between name endings more often. Contrary to previous studies (Chermahini & Hommel, 2010, 2012), we did not find a relationship between participants' spontaneous eye blink rate (an indirect marker of striatal dopamine functioning; Groman et al., 2014) and either rule divergent or rule convergent thinking in our study.

In Chapter 3, I reviewed converging evidence indicating that dopamine in fronto-striatal brain areas modulates flexible and persistent processes in creativity. Based on this evidence, I presented a model of dopaminergic modulation of creativity that may guide future research in this area. As proposed in the Dual Pathway to Creativity Model (DPCM; De Dreu et al., 2008; Nijstad et al., 2010), creative ideas may result from flexible processes (e.g., having a broad attentional scope, seeing remote associations between concepts, divergent thinking), but also from more analytical, persistent processes (e.g., the thorough exploration of options along a certain line, convergent thinking). Our new model proposes that these flexible and persistent processes in creativity are modulated by the nigrostriatal and mesocortical dopamine pathway, respectively. More specifically, striatal dopamine may facilitate flexibility and control the flow of new information into working memory, so that people can easily switch between different task approaches and consider more remotely associated concepts (e.g.,
General Discussion

Chermahini & Hommel, 2010, 2012; Zhang et al., 2014a). Dopamine in the prefrontal cortex, on the other hand, supports the maintenance and manipulation of representations in working memory, allowing for persistence (e.g., Colzato et al., 2014; Mayseless et al., 2013; Zhang et al., 2014b). Moreover, the relationship between dopamine and these creative processes may follow an inverted-U-shaped function (Chermahini & Hommel, 2012; Durstewitz & Seamans, 2008; Kellendonk et al., 2006). Moderate levels of striatal dopamine benefit creative cognition by facilitating flexible processes, while both low and high levels of striatal dopamine impair these processes. Similarly, moderate (but not low or high) levels of prefrontal dopamine support persistent processes in creativity. As creative ideas likely result from a combination of flexible and persistent processes rather than one or the other, we propose that creativity also requires a delicate balance of dopaminergic functioning in fronto–striatal brain areas.

In both Chapter 4 and 5, we investigated several potential explanations for the inconsistent evidence regarding creativity in ADHD. This neurodevelopmental disorder is characterized by symptoms of distractibility, hyperactivity, and impulsivity, presumably as a result of dopaminergic hypoactivity in several brain areas (Sagvolden et al., 2005; Sonuga-Barke, 2003). Chapter 4 included three empirical studies in which we addressed the relationship between subclinical ADHD symptoms and different types of creative processes in a large sample of healthy students. We hypothesized that the relationship between ADHD and creativity would depend on the type of symptoms that people with ADHD experience (i.e., inattention, hyperactivity–impulsivity, or both) and on the specific (flexible or persistent) creative processes under investigation. In line with our expectations, we found that ADHD symptoms in general were associated with more publically recognized creative achievements in daily life and enhanced self-reported creative behavior (although effect sizes were small). Moreover, these symptoms were associated with enhanced divergent thinking and with more original, but less practical, reconstruction of complex problems. Our results indicate that these relationships were mainly driven by hyperactive–impulsive rather than inattention symptoms of ADHD. When controlling for hyperactive–impulsive symptoms, inattention
symptoms were associated with reduced self-reported creative behavior and enhanced divergent thinking during one of the ideation tasks, but not with any of the other creativity indicators. We also expected that the relationship between symptoms of ADHD and creative performance would be curvilinear rather than linear, similar to the relationship between other mental disorders and creativity (Acar & Sen, 2013; Furnham et al., 2008), but this hypothesis was not supported by the data across the three studies.

In the two studies presented in Chapter 5, we extended these findings regarding creativity in ADHD in healthy samples by investigating creativity in people with clinical ADHD (vs. healthy controls). In Study 5.1, we assessed the role of intrinsic motivation, performance during early stages of the creative process (i.e., the reconstruction of ill-defined creative problems in terms of goals and constraints), and medication use as potential explanations for the inconsistent findings in previous studies on creativity in ADHD. First of all, we replicated findings showing that people with (symptoms of) ADHD report more real-world creative achievements (Chapter 4; White & Shah, 2011). However, divergent thinking and intrinsic motivation during creative idea generation did not differ between groups. Although participants with ADHD (vs. controls) selected fewer useful problem reconstructions, they were not more original in reconstructing problems. Also, we did not find any differences in creative task performance between medicated and unmedicated participants with ADHD in this study. In Study 5.2, we assessed whether people with ADHD can be motivated to generate more original ideas by introducing competition between participants during an idea generation task. We found that competition for a bonus increased the originality of ideas that people with ADHD generated, whereas the creative performance of participants in the control group was not affected by competition. Although competition increased participants’ motivation during idea generation, motivation did not differ between groups. In addition, we found that ADHD participants (vs. controls) rated themselves as being more creative in specific domains of creativity. More specifically, people with ADHD reported higher creative abilities in the performance (e.g., playing music in public, shooting a YouTube video) and mechanical/scientific domain
General Discussion

(e.g., constructing something out of metal, programming), but not in the everyday, artistic, or scholarly domain.

In Chapter 6, I described a study in which we tested whether methylphenidate administration would affect creative performance in a group of healthy participants and, if so, whether such effects depended on individual differences in hyperactive–impulsive ADHD symptoms and working memory capacity. An increasing number of healthy people use methylphenidate, a stimulant that elevates dopamine and noradrenaline levels in the brain (Arnsten & Dudley, 2005; Kuczenski & Segal, 1997), to help them focus over extended periods of time. Methylphenidate may indeed facilitate cognitive functions related to cognitive stability, including persistent creative processes such as convergent thinking, but it may simultaneously impair people's cognitive flexibility, including flexible creative processes and divergent thinking (Gvirts et al., 2016; Linssen et al., 2014). Contrary to our expectations, we did not find methylphenidate effects on participants' convergent or divergent thinking performance on any of the three creativity tasks that we included in this study. Also, our findings did not indicate that methylphenidate effects depended on individual differences in hyperactivity–impulsivity or baseline working memory capacity.

Implications of the Findings

Across studies, our findings have a number of important implications. First of all, we argued that both flexible and persistent processes in creativity are modulated by (striatal and prefrontal) dopamine. Our proposed model implies that flexibility and persistence can be enhanced or impaired by administration of drugs that influence prefrontal and/or striatal dopamine levels in the brain, such as bromocriptine, sulpiride, and methylphenidate (cf. Clatworthy et al., 2009; Cools et al., 2007; Dodds et al., 2009; Gvirts et al., 2016; Mohamed, 2016). Also, this model predicts that situational factors associated with an increase or decrease of dopaminergic activity, such as stress (Arnsten, 2009), reward anticipation (Müller et al., 2007), and positive affect (Ashby et al., 1999; Dreisbach & Goschke, 2004) may affect flexible and persistent creativity in predictable ways. Importantly, the direction of such effects will likely depend on people’s baseline dopamine levels (cf. Cools & D’Esposito, 2011; Durstewitz &
Seamans, 2008). Increasing striatal dopamine in people with high baseline dopamine levels in this area may lead to distractibility and decrease creative performance, whereas dopamine administration may improve creative cognition in people with low baseline striatal dopamine. Likewise, increasing prefrontal dopamine in individuals with high baseline dopamine levels may lead to cognitive rigidity and impair creative performance, whereas it may increase the ability to focus in people with low prefrontal dopamine.

Because these claims are based on indirect evidence from several lines of research, we aimed to obtain direct evidence for our proposed model by manipulating dopamine (and noradrenaline) levels in healthy participants using methylphenidate. A recent study by Gvirts and colleagues (2016) showed that methylphenidate administration affected divergent thinking depending on individual differences in novelty seeking, but we did not find evidence for such methylphenidate effects on either convergent or divergent thinking in our study. Also, in our study, methylphenidate effects did not depend on individual differences in participants’ baseline dopamine levels, as indicated by participants’ working memory capacity and hyperactivity–impulsivity symptoms. Although these null findings may be due to methodological factors, such as low power to detect methylphenidate effects, the most straightforward interpretation of our findings is that manipulation of dopamine (and noradrenaline) levels using methylphenidate does not affect either flexible or persistent creative processes to an observable degree. This interpretation is in line with our observation that the use of ADHD medication (including methylphenidate) did not affect creative performance in adults with ADHD (see also White & Shah, 2011). We also did not find a relationship between participants’ spontaneous eye blink rates and either rule divergent or rule convergent thinking, further indicating that dopamine may not play a key role in modulating these processes. Thus, the findings presented here do not provide convincing evidence for the dopaminergic model that we proposed. Furthermore, although the use of methylphenidate by healthy people may still be undesirable for a number of reasons (De Jongh et al., 2008; Greely et al., 2008), our results do not imply that using methylphenidate as a cognitive enhancer is detrimental to people’s ability to be creative.
Our studies on creativity in ADHD provide some new insights that may resolve some of the inconsistencies observed in previous studies focusing on this relationship. First, we showed that it is important to take the specific ADHD symptoms that people experience into account when investigating creativity in ADHD. Also, the relationship between (symptoms of) ADHD and creativity seems to depend on the extent to which a creative task relies more on flexible or persistent creative processes. Our findings suggest that the positive association between ADHD and (flexible) creative processes observed in previous studies (Abraham et al., 2006; White & Shah, 2011, 2016) is mainly driven by hyperactivity–impulsivity symptoms of ADHD, including high energy, disinhibition, and enhanced risk taking (although we did not observe a relationship between these symptoms and creative performance in our methylphenidate study). Indeed, such traits have previously been associated with enhanced creativity through flexibility (Baas et al., 2016; Barron & Harrington, 1981; Feist, 1998; Radel et al., 2015). Contrary to studies showing that enhanced processing of task–irrelevant information contributes to divergent thinking (Baird et al., 2012; Carson et al., 2003), our findings do not indicate that the inattention symptoms of ADHD, such as distractibility and having difficulty organizing one’s activities, facilitate divergent thinking. Rather, our findings support the idea that divergent thinking requires some degree of cognitive control and sustained attention (Benedek et al., 2012; Zabelina et al., 2015; Zabelina et al., 2016b). As both hyperactive–impulsive and inattention symptoms of ADHD are associated with dopaminergic hypofunctioning in several pathways (Sagvolden et al., 2005; Sonuga-Barke, 2003), these findings provide additional indirect support for the involvement of dopamine in creative processes. However, the complexity of the dopamine system and its role in the development of ADHD symptoms makes it difficult to interpret these findings in terms of the two pathways proposed in our dopaminergic model. We did not observe any curvilinear relationships between ADHD symptoms and the creativity indicators, indicating that creative performance is not impaired as ADHD symptoms get more severe. Possibly, our highly educated participants had additional cognitive resources at their disposal to compensate for the cognitive deficits associated with their (strong) ADHD symptoms (Carson et al., 2003; Nijstad et al., 2010).
Our findings on creativity in ADHD also showed that increased goal-directed motivation may drive the enhanced real-world creative achievements of people with ADHD. Our results suggest that the prospect of rewards, either monetary or in the form of the chance to beat an opponent, increases the originality of ideas generated by people with ADHD. Possibly, this prospect can motivate people with ADHD to exert more effort during the idea generation processes, resulting in more original ideas (cf. Geurts et al., 2008; Kohls et al., 2009). Third, our results suggest that people with ADHD may be more creative in specific situations that fit their abilities and preferences (Verheul et al., 2015, 2016). By selectively engaging in such situations, people may develop a high level of expertise in specific creative domains over time, allowing them to generate more original ideas (Baer, 2015). Possibly, people with ADHD like to engage in those creative activities for which a specific framework (e.g., a script, a song to sing or play, a programming language) is available to provide structure and guide performance in case of executive dysfunctions (Barkley, 1997; Ramsay, 2010).

However, when interpreting these findings, it is important to keep in mind that they are based on self-reported creativity rather than actual creative performance and that scientific creativity may be overestimated in our sample of mainly university students. Together, our findings imply that directing people with ADHD (particularly those diagnosed with the combined and hyperactive–impulsive subtype) to creative tasks or jobs could help them take advantage from the disorder, especially when creative performance is rewarded in such situations.

Finally, we showed that both divergent (vs. convergent) creative processes and switching between (vs. repeating) thinking modes were associated with reduced delta-band activity. These findings are hard to interpret in terms of the underlying neural mechanisms, although we are not the first to associate delta-band oscillations with creative processes (Bhattacharya & Petsche, 2005; Foster et al., 2005). Oscillations in general seem to be involved in coordinating neural activity in large-scale networks (Buzsáki & Draughn, 2004; Fries, 2005), but much is unclear about the role that delta-band activity plays in such interactions (Nácher et al., 2013). Although speculative, oscillations in the delta band may be involved in functional interactions between areas in the large-scale neural...
network involved in creative processes (e.g., Beaty et al., 2016). Reduced delta-band activity over fronto-central areas has been associated with increased activity in the default mode network (Jann et al., 2010) – a network of brain areas that has recently been associated with enhanced divergent thinking (Beaty et al., 2014; Jauk et al., 2015; Kühn et al., 2014; Takeuchi et al., 2012). Also, delta-band activity has been associated with the inhibition of potentially interfering processes during cognitive tasks (Harmony, 2013; Prada et al., 2014) and differences in delta-band activity may thus reflect differences in top-down control and inhibition of task-irrelevant information that convergent and divergent processes require (Baird et al., 2012; Carson et al., 2003; Hommel, 2012). Contrary to a number of previous EEG studies (e.g., Fink & Benedek, 2014; Jauk et al., 2012), we did not observe differences in alpha power between divergent and convergent processes, although we focused on a more narrow subset of divergent and convergent processes in the present study, making it difficult to directly compare our results to those obtained in other studies. Thus, more research is necessary to establish whether oscillations in the delta band also dissociate the broader constructs of divergent vs. convergent thinking, and flexible vs. persistent creative processes in general. More importantly, however, the present study advances our understanding of the neural dynamics of creativity by resolving some of the methodological challenges that characterize neuroscientific creativity research.

**Limitations and Future Directions**

Although the studies presented in this dissertation have several interesting implications for the way in which we view and study the creative brain, several theoretical and methodological limitations of the present work can be identified. Also, some directly relevant topics were beyond the scope of our studies, leaving open a number of interesting questions that may be addressed in future research.

First of all, we focused on dopamine as the main neurotransmitter involved in creativity throughout this dissertation. Several chapters were directly or indirectly based on the idea that dopamine in nigrostriatal and mesocortical pathways plays a key role in flexible and persistent creative processes. However, direct evidence for dopaminergic modulation of creative processes in these areas is lacking at present. Although one recent study showed that methylphenidate
administration affects divergent thinking in healthy subjects (Gvirts et al., 2016), the possibility that these effects were driven by increases in noradrenaline levels cannot be excluded. Moreover, we did not manage to replicate these findings in the study described in Chapter 6. Thus, it is crucial that future research focuses on obtaining direct evidence for the proposed dopaminergic modulation of creativity. This could be done by manipulating dopamine levels in healthy individuals using dopaminergic (anti)agonists, such as bromocriptine and sulpiride, ideally by combining them in pretreatment studies that allow for neurochemical specificity (Dodds et al., 2008; Piray et al., 2015; Van Holstein et al., 2011). Alternatively, pharmacological studies could track dopaminergic activity using PET imaging to relate changes in dopamine availability in different brain areas to creative performance (Clatworthy et al., 2009; De Manzano et al., 2010). Importantly, such studies need to test the hypothesis that the relationship between dopamine and creativity follow an inverted-U-shaped function, so that effects of dopamine administration depend on participants’ baseline dopamine levels (e.g., Chermahini & Hommel, 2012; Cools & D’Esposito, 2011; Durstewitz & Seamans, 2008). At the same time, our methylphenidate study, as well as previous work by others (Farah et al., 2009; Gvirts et al., 2016; Müller et al., 2013), suggests that effects sizes in such pharmacological studies are likely to be small, making it difficult to obtain convincing support for one’s hypotheses using the sample sizes than are common in such studies. Possibly, future meta-analyses may clarify whether methylphenidate and similar substances affected creative processes across studies.

Moreover, our dopaminergic model ignores the potential role of a third main dopaminergic pathway – the mesolimbic pathway – in creativity. This pathway, projecting to the ventral striatum and parts of the frontal cortex, may influence creativity through its role in appetitive motivation and reward processing (Ikemoto, 2007; Schultz, 2002). For example, approach motivation, a strong sensitivity to appetitive stimuli and positive reinforcement, is associated with greater flexibility and originality during creative tasks (Baas et al., 2011, 2013; De Dreu et al., 2011). Involvement of the mesolimbic dopamine pathway is also suggested by our findings regarding the effects of competition on creativity in ADHD. Dopaminergic hypoactivity in the mesolimbic reward system, resulting
in motivational deficits and an increased sensitivity to (immediate) rewards, may underlie some of the core cognitive deficits observed in ADHD (Sagvolden et al., 2005; Sonuga-Barke, 2003; Ströhle et al., 2008; Toplak et al., 2005; Volkow et al., 2011b). In fact, evidence indicates that treatment of ADHD symptoms using methylphenidate may improve cognitive functioning by normalizing these motivation-related abnormalities in brain activity, thereby increasing task saliency (although we did not find differences in creativity as a result of medication use in people with ADHD; see Chapter 5) (Liddle et al., 2011; Volkow et al., 2004). Future studies could investigate the role of mesolimbic dopamine in creativity in both healthy people and people with ADHD to establish whether this pathway needs to be added to the dopaminergic model that I described in Chapter 3.

Furthermore, several factors limit the interpretation and generalizability of our findings on creativity in ADHD. First of all, it is important to note that we did not measure impulsive responding or attentional processes in participants with (symptoms of) ADHD, but only relied on self-reported ADHD symptoms. In our studies on subclinical symptoms of ADHD, a much larger percentage (17%) of presumably healthy participants reported a degree of symptoms that would qualify for a clinical diagnoses than would be expected based on prevalence of ADHD in the general population (2.5%; Simon et al., 2009). Thus, the self-reported symptoms may reflect common feelings of restlessness or boredom among students, rather than actual ADHD symptoms that would be reflected in decreased performance during cognitive (and creative) tasks. Second, because most of our studies on ADHD were conducted in samples of university students, the results may not generalize to more heterogeneous samples. Although this is the case in many scientific studies, it may be particularly problematic in case of research on ADHD, given that people with ADHD are generally less likely to do well in academic settings (e.g., Wilmshurst et al., 2011). Possibly, the high-functioning participants in our student samples may have had additional cognitive resources at their disposal to compensate for any cognitive deficits associated with their ADHD symptoms (Carson et al., 2003; Nijstad et al., 2010). Thus, future studies should look at relationship between symptoms of ADHD in a more representative sample.
Our studies on creativity in ADHD highlight the importance of distinguishing between real-world creative achievements and creative performance measured in the laboratory in future studies. In Chapter 5, we showed that people with ADHD reported more creative achievements than people without the disorder, but were not more or less creative than controls during divergent thinking tasks. These findings are in line with the idea that creative achievements and divergent thinking benefit from different types of attentional processes (Zabelina et al., 2016b). In this study, people who reported a high number of real-world creative achievements were more strongly influenced by task-irrelevant information during a cognitive task than low creative achievers. Good divergent thinkers, on the other hand, showed flexible but selective attention and were less affected by task-irrelevant information than people with lower divergent thinking abilities (see also Benedek et al., 2012; Zabelina et al., 2015). As real-world creativity is a complex construct that relies on several stages of creative problem solving, such as problem identification and the selection of the most promising ideas, it also seems important look beyond the idea generation stage when trying to relate real-world creativity to creativity in the lab. Our findings in subclinical ADHD (Chapter 5) show that certain characteristics or cognitive processes may simultaneously be associated with enhanced performance during one stage of the creative process and decreased performance during another. Future studies could focus on multiple stages of the creative problem solving process to obtain a more complete picture of the neural and cognitive correlates of creativity.

Finally, future research on the creative brain may further investigate dynamic interactions between large-scale networks in the brain in creativity. Several of our findings (indirectly) support accumulating evidence that associates creative processes with activation of the default mode network, a large network of functionally connected brain areas that is active during passive cognitive states (Beaty et al., 2014, 2016; Jauk et al., 2015; Kühn et al., 2014; Takeuchi et al., 2012). During performance of externally presented cognitive tasks, areas of the default mode network deactivate, while activity in the executive control network, another large-scale network, increases. Activation of the default mode network has been associated with self-generated thought and
mind-wandering – a process that may contribute to divergent thinking by expanding the associative network so that uncommon associations are activated (Baird et al., 2012; Buckner et al., 2008; Christoff et al., 2009). A recently proposed model states that the default mode and executive control networks cooperate during the creative process (Beaty et al., 2016). The authors suggest that the default mode network may be involved in the generation of ideas, whereas the executive control network contributes to idea evaluation and the modification of ideas to meet task and situational constraints. In the EEG study presented in Chapter 2 of this dissertation, we found that divergent (vs. convergent) processes in idea generation are associated with reduced delta power in a widespread network of brain areas. Although speculative, this could point at increased default mode network activity during divergent compared to convergent thinking (Jann et al., 2010). Second, altered activation of the default mode network may be related to creativity in ADHD. Reduced task-related deactivation of the default mode network has been observed in children with ADHD, possibly increasing the likelihood of attentional lapses (Liddle et al., 2011). This default mode network hyperactivity during performance of cognitive tasks was normalized by increasing task saliency, either by rewarding performance or by methylphenidate treatment (see also Volkow et al., 2004). Thus, the investigation of how activation and deactivation of the default mode networks and other large-scale networks interact during different creative processes (and in different groups of people) seems to be a promising avenue for future research.

**Concluding remarks**

Together, the studies presented in this dissertation advance our understanding of the neural and cognitive mechanisms underlying creativity, as well as the ability to study them, in several ways. First of all, the new paradigm that we presented for studying convergent and divergent processes in EEG settings allows researchers to measure these processes simultaneously, while keeping context and instructions constant, thereby increasing the comparability between these processes. Second, our model of dopaminergic modulation of creativity may guide future research on this topic. Third, our findings regarding creativity in ADHD help us understand when and how people with (symptoms of)
ADHD are more creative than others – knowledge that could help these people to take advantage of their symptoms in situations that ask for creativity. Finally, our findings on the effects of methylphenidate on creative processes contribute to a growing collection of knowledge that will allow people to make more informed decisions about whether to use such substances or not.

While these findings cast some light on the potential mechanisms at work in the creative brain, at the same time they open up at least as many new research questions as they answer. If I would be asked to summarize my four years of research into one main conclusion, I would state that, indeed, creativity is an elusive collection of neural processes that is hard to capture in the laboratory. Studying it required me to think outside the box many times and has taught me a great deal about my own creative brain. I hope that the findings and methods presented in this dissertation will inspire other researchers in their search for the neural processes that drive creativity. To use one of the hundreds of creativity quotes of obscure origin that roam the internet (something I managed to resist until the very end of this dissertation): “Making the simple complicated is commonplace; making the complicated simple, awesomely simple, that’s creativity” (Charles Mingus, American jazz musician, 1977).