Neuroscience and Biobehavioral Reviews 78 (2017) 13–23

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review article

# Creative cognition and dopaminergic modulation of fronto-striatal networks: Integrative review and research agenda

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ARTICLE INFO

*Keywords:*
Creativity
Dopamine
Divergent thinking
Fronto-striatal networks

ABSTRACT

Creative cognition is key to human functioning yet the underlying neurobiological mechanisms are sparsely addressed and poorly understood. Here we address the possibility that creative cognition is a function of dopaminergic modulation in fronto-striatal brain circuitries. It is proposed that (i) creative cognition benefits from both flexible and persistent processing, (ii) striatal dopamine and the integrity of the nigrostriatal dopaminergic pathway is associated with flexible processing, while (iii) prefrontal dopamine and the integrity of the mesocortical dopaminergic pathway is associated with persistent processing. We examine this possibility in light of studies linking creative ideation, divergent thinking, and creative problem-solving to polymorphisms in dopamine receptor genes, indirect markers and manipulations of the dopaminergic system, and clinical populations with dysregulated dopaminergic activity. Combined, studies suggest a functional differentiation between striatal and prefrontal dopamine: moderate (but not low or high) levels of striatal dopamine benefit creative cognition by facilitating flexible processes, and moderate (but not low or high) levels of prefrontal dopamine enable persistence-driven creativity.

## 1. Introduction

Compared to other species, humans have unsurpassed ability to explore, to seek and create novelty, and to enjoy it. Such ability to create and innovate allows humans to flexibly adapt to, and prosper in, rapidly changing environments, to increase social standing and reputation, to perform complex tasks, and to make high quality decisions (Hennessey and Amabile, 2010; Miller, 2000; Nijstad et al., 2010; Runco, 2004; Sternberg, 1999). Although creativity is often seen as rather elusive (Plucker and Renzulli, 1999; Runco, 2004), psychological science converges on an operational definition of creativity as the production of outcomes (e.g., ideas, products, services) that are original, yet potentially useful (Runco and Jaeger, 2012). Creative performance is influenced by a range of cognitive process(es) such as accessing remote associations and divergent thinking, exogenous factors such as extrinsic rewards and time pressure, and characteristics of the creative person such as approach orientation (in which motivation and behavior are regulated by, and directed towards, desired and appetitive stimuli), openness to experience, intelligence, and vulnerability to psychopathology (Amabile, 1996; Baas et al., 2008, 2016; Runco, 2004; Sternberg, 1999).

In the creativity literature, a distinction is made between 'little c' and 'big C' creativity (Gardner, 1993). Whereas 'big C' creativity refers to eminent creative achievements of brilliant scientists such as Marie Curie and Albert Einstein, of great inventors such as Thomas Edison, or of famous artists such as Emily Dickinson, Pablo Picasso, or The Beatles, 'little c' creativity refers to relatively mundane contributions and everyday creativity, expressed in people's novel use of language, their ability to create and apply new mental categories to organize experiences, and their ability to mentally manipulate objects (Kaufman and Beghetto, 2009; Ward et al., 1999). Here we focus on 'little c' creativity, for two reasons. First, it is important in day-to-day life: it helps us adapt to changing circumstances, to solve everyday problems, and to create new opportunities (Richards, 2007). Second, the cognitive processes that support 'little c' creativity may also operate in cases of 'big C' creativity (Guilford, 1950; Ward et al., 1999), and the study of 'little c'

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[1] This work was facilitated by a grant from the Netherlands Organization for Scientific Research (NWO-451-12-023) to Matthijs Baas.

http://dx.doi.org/10.1016/j.neubiorev.2017.04.007
Received 18 March 2016; Received in revised form 28 March 2017; Accepted 9 April 2017
Available online 15 April 2017
0149-7634/ 

creativity may therefore contribute to a better understanding of creative genius (Nijstad et al., 2010).[2]

Underlying the cognitive processes accounting for 'little c' creativity are neural circuitries that may be temporarily or more chronically (de-) activated. Uncovering such neural circuitries may thus offer a unifying framework for understanding how person and situation characteristics influence creativity. Recent advances in neurobiology and (cognitive) neuroscience converge on several candidate regions and networks in the human brain that seem to be involved in creative cognition, including prefrontal, parietal, and temporal circuitries, and the striatum (e.g., Abraham et al., 2012; Beaty et al., 2016; Mayseless et al., 2011). For instance, creativity is associated with the activation of prefrontal circuitries that are involved in the controlled manipulation of information and executive functioning (Abraham et al., 2012; Barr et al., 2014; Benedek et al., 2014; De Dreu et al., 2012; Dietrich and Kanso, 2010; Gonen-Yaacovi et al., 2013; Metuki et al., 2012). Creativity also seems to be related to the striatum, which is part of a sub-cortical network involved in reward processing, habitual behavior, and flexible updating of goal representations and switching between task strategies (Abraham et al., 2012; Cools and D'Esposito, 2011; Ikemoto, 2007; Mayseless et al., 2013; Zabelina et al., 2016). Interestingly, the striatum and prefrontal cortex are strongly interconnected and conditioned by the neurotransmitter dopamine (Alexander et al., 1986). Moreover, growing evidence from neurobiology shows that dopaminergic modulation of such fronto-striatal circuitries regulates the balance between flexibility and persistence (Cools et al., 2007), two key cognitive processes that support creativity (Nijstad et al., 2010).

The purpose of this review is two-fold. First, we review evidence for a functional differentiation between striatal and prefrontal dopamine: moderate (but not low or high) levels of striatal dopamine benefit creative performance by facilitating flexible processes, and moderate (but not low or high) levels of prefrontal dopamine enable persistence-driven creative outputs. Second, we aim to integrate and connect this possibility with research using standardized tests to measure creative cognition and performance conducted in social, personality, and clinical psychology. In combination, these two aims integrate recent insights into the neural underpinnings of creative cognition and performance, and provide a research agenda for further understanding the neurocognitive underpinnings of creativity.

We proceed as follows. Section II reviews contemporary scientific approaches to (measure) human creative cognition, suggesting that creative outputs derive from two distinct yet interrelated cognitive processes—flexibility (allowing people to consider different task approaches and unconventional perspectives) and persistence (enabling people to work on creative problems attentively and thoroughly over longer periods of time). Section III summarizes neurobiological work on dopaminergic modulation of fronto-striatal circuitries in relation to flexibility and persistent processing and a model of dopaminergic modulation of creativity via fronto-striatal brain circuitries is proposed. Section IV reviews the evidence for our model and integrates currently scattered and oftentimes indirect research evidence on the relationship between striatal and prefrontal dopamine activity, and flexibility and persistence in creativity. Section V examines knowledge gaps, avenues for future research, and possibilities for creative enhancement. Section V also addresses possible other neural networks and circuitries that assist creativity in addition to the fronto-striatal circuitries addressed here.

[2] The relationship between little c and big C creativity is not necessarily straightforward. For example, the relationship between creative performance on laboratory tasks and creative achievements outside of the laboratory (e.g., in the classroom, or in one's profession) can be rather weak (Baer, 2011a; Kim, 2008). A discussion of possible explanations in terms of measurement issues, the role of domain-specific expertise, and state/trait-based moderators is beyond the scope of this article (but see, e.g., Baer, 2011b; 2016; Kaufman, 2016; Runco and Acar, 2012; Simonton, 2007).

## 2. Demystifying creativity

To study creative cognition and its underlying processes, scientists have developed and used a range of tasks and measures, some of the more frequently used ones are shown in Table 1. Many of these tasks directly measure creative outputs – ideas or insights that are novel yet fitting and potentially useful – but also provide good insight into the underlying cognitive processes. Consider the widely used Alternative Uses Task, in which individuals write down as many unusual ways to use a common object, such as a brick or a tin can (Guilford, 1967). Ideas are scored in terms of *originality* (the extent to which the ideas are unusual and novel), and in terms of underlying cognitive processes as reflected in for example *fluency* (the number of generated ideas) and *flexibility* (the number of different conceptual categories that the ideas belong to). There is good evidence that both fluency and flexible processing benefit originality (Nijstad et al., 2010).

In addition to such open-ended idea generation tasks, creativity has been examined using insight tasks. Insight tasks typically require unexpected and unusual approaches or mental restructuring of information about a presented problem (both flexible and divergent processing), as well as the ability to engage in constrained and confirmatory search processes to identify the correct solution (Bowden et al., 2005; Cropley, 2006). Consider the frequently used Remote Associates Test, in which participants receive series of three words that are only remotely related to each other (e.g., *falling*, *actor*, *dust*) and are instructed to generate a word that relates to all of these three words (i.e., *star*) (Mednick, 1962). To find the correct solution, people rely on divergent thinking to sample potentially correspondent attributes and relations associated with the three provided words, but test a possible solution through convergent processing (Chermahini and Hommel, 2010; De Dreu et al., 2014; Folley and Park, 2005).

### 2.1. Two pathways to creativity

The processes listed in Table 1 may suggest that it is divergent, remote, and flexible thinking that promotes original ideation and creative problem solving. However, such intuition is best characterized as a half-truth according to the Dual Pathway to Creativity Model (De Dreu et al., 2008; Nijstad et al., 2010). The model expands on earlier work into creative cognition and problem-solving (e.g., Amabile, 1996; Ashby et al., 1999; Mednick, 1962; Simonton, 2003; Ward et al., 1999) and conjectures that creative outputs result from two distinct cognitive processes—flexibility versus persistence. The *flexibility pathway* includes a broad attentional scope (a tendency to perceive holistic and global rather than detailed structures), facilitated access to semantic concepts with lower *a priori* accessibility, divergent thinking, and flexible switching between perspectives (Mayseless et al., 2013; Runco et al., 2011; Silvia et al., 2008). As such, the flexibility pathway incorporates a range of (lower-level) cognitive processes and skills such as switching between cognitive sets or response rules (Alexander et al., 2007) and the inhibition of a dominant response in favor of a more appropriate response (Dreisbach and Goschke, 2004; also see Ashby et al., 1999; Nijstad et al., 2010).[3] Alone and in combination, these

[3] Within the cognitive neurosciences and psychology, the term flexibility is used to refer to a variety of cognitive processes or skills (Eslinger and Grattan, 1993; Ionescu, 2012; Zabelina et al., 2015), including switching between cognitive sets or response rules (Kehagia et al., 2010), inhibition of a dominant response in favor of a more appropriate response (Dreisbach and Goschke, 2004), manipulation of information in working memory (Durstewitz and Seamans, 2008), and goal-directed exploration (Cools, 2012). In addition, different types of flexibility associate with activity in different parts of the brain (e.g., Eslinger and Grattan, 1993; Ravizza and Carter, 2008). We refrain here from solving this definitional issue. However, to avoid confusion, we refer to flexibility as the ease with which people break the set of typical associations and consider different perspectives or alternatives during idea generation or problem solving (Ashby et al., 1999; Nijstad et al., 2010); *cognitive* flexibility is used to refer to the ease with which people can switch among different tasks, sets, strategies, or goal representations

**Table 1**
Creativity self-report and standardized performance measures.

| Sample measure | Sample item/description |
|---|---|
| Creative personality | |
| GPS[a] | Participants mark characteristics indicative of creative (e.g., original) and non-creative personality (e.g., modest). |
| Self-rated creativity | |
| CAQ[b] | Participants mark recognized and concrete creative achievements in ten domains (e.g., visual arts, sciences, music). Scores for each domain are summed together to yield a creative achievement score. |
| CDQ-R[c] | Respondents indicate their perceived level of creativity in different domains (e.g., How creative would you rate yourself in dancing?). |
| Creative products | |
| AUT[d] | Participants generate as many possible uses for an object (e.g., brick, tin can). Ideas coded for originality. |
| Brainstorm task[e] | Participants generate as many possible ideas about a given topic (e.g., improve the environment). Ideas are coded for originality. |
| Drawing test[f] | Participants draw as many possible figures using provided shapes (e.g., triangles). Drawings are rated for originality by coders. |
| Pasta task[g] | Participants generate as many possible new names for types of pasta given five examples with the same end letter. New names scored as divergent or convergent based on (different versus same) end letter. |
| Alien drawing task[h] | Participants draw an alien. Creativity determined by extent to which alien diverges from mammals (e.g., asymmetric body). |
| Collage building task[i] | Participants make a collage with provided material. Creativity of product rated by experts. |
| Poem[j] | Participants write a poem according to specified rules. Creativity of product rated by experts. |
| RAT[k] | Participants generate a word that connects three stimulus words (e.g., black, bean, break; answer: coffee). Correct solution: yes/no. |
| Candle task[l] | Participants have to support a candle on the wall using a candle, matches, and a box of tacks. Correct solution: yes/no. |
| Nine-dot problem[m] | Participants connect nine dots in a square array by drawing four straight lines. Correct solution: yes/no. |
| Creative processes | |
| AUT[d], Brainstorm task[e] | *Flexibility*: number of different conceptual categories that ideas fall into. *Persistence*: number of ideas within one category, time on task, level of semantic clustering during idea generation. |
| Pasta task[g] | *Flexibility*: number of divergent pasta names and switches between names with different end letters. *Persistence*: number of convergent pasta names and repetitions of names with the same end letter. |
| Category inclusion task[n] | *Flexibility*: tendency to rate non-typical exemplars of a category (e.g., camel) as members of that category (e.g., vehicle). |
| RAT[k], Candle task[l], Nine-dot problem[m] | Creative insight, requires both convergent and divergent thinking processes. |

[a] Gough Personality Scale; Gough (1979).
[b] Creative Achievement Questionnaire; Carson et al. (2005).
[c] Creativity Domain Questionnaire-Revised; Kaufman et al. (2009).
[d] Alternative Uses Task, Guilford (1967).
[e] Nijstad et al. (2010).
[f] Akinola and Mendes (2008).
[g] De Dreu et al. (2014).
[h] Rietzschel et al. (2007).
[i] Amabile (1996).
[j] Amabile (1996).
[k] Remote Associates Test; Mednick (1962).
[l] Duncker (1945).
[m] MacGregor et al. (2001).
[n] Isen et al. (1987).

facets of flexibility typically associate with creative insight performance and original ideation (Carson et al., 2003; Chermahini and Hommel, 2012; Cretenet and Dru, 2009; De Dreu et al., 2011; Gilhooly et al., 2007; Zabelina and Robinson, 2010).

In addition to the flexibility pathway, research uncovered a *persistence pathway* to creative cognition and performance. The persistence pathway includes more convergent, focused and systematic thinking and prolonged and incremental search processes. The basic insight is that although incremental search and systematic processes may initially lead to obvious and readily available ideas, they will result in novel ideas and solutions after more readily available ideas have been considered and discarded (Lucas and Nordgren, 2015; Ward et al., 1999). Relative to flexible processing, persistent processing requires more mental effort and cognitive resources (Baas et al., 2013; Chermahini and Hommel, 2010; De Dreu et al., 2012; Roskes et al., 2012). Indeed, more effortful and systematic creative thinking associates with activation in prefrontal circuitries (Benedek et al., 2014; Dietrich and Kanso, 2010; Gonen-Yaacovi et al., 2013), and with working memory capacity (Barr et al., 2014; Benedek et al., 2014; De Dreu et al., 2012; Lucas and Nordgren, 2015). Accordingly, stimulating (areas within) the PFC increases creative insight performance and enhances original ideation (Cerruti and Schlaug, 2009; Mayseless and Shamay-Tsoory, 2015; Metuki et al., 2012).

## 3. Flexibility and persistence: dopamine and the fronto-striatal network

Although flexible and persistent processing may independently lead to creative outputs, creativity most likely benefits from an interplay between flexibility and persistence in various stages of the creative process (Cropley, 2006; Nijstad et al., 2010; Zabelina and Robinson, 2010). For example, an individual may first engage in divergent thinking to identify many possible candidate solutions for a problem, and then examine a limited number of fruitful possibilities more fully through convergent analysis (Runco, 2004; Ward et al., 1999).

### 3.1. Processing modes and the fronto-striatal network

This calibrated switching between processing-modes requires some higher-level cognitive control (Dreisbach and Goschke, 2004; Zabelina and Robinson, 2010) and indeed, optimal creative performance benefits moderate rather than extreme levels of either flexibility or persistence. Whereas extreme flexibility without any focus results in distractibility and "weird" rather than creative ideas, excessive persistence without any flexibility produces rigid thinking and "boring" rather than creative ideas (Dreisbach and Goschke, 2004; Durstewitz and Seamans, 2008; Nijstad et al., 2010). Thus, optimal creative performance requires *both*

(*footnote continued*)
(Alexander et al., 2007). We emphasize that measures of *cognitive flexibility* positively associate with indicators of flexibility (Carson et al., 2003; Gilhooly et al., 2007; Zabelina and Robinson, 2010).

flexibility and persistence, and sub-optimal creativity emerges when flexibility is over-excited at the expense of persistence or, alternatively, persistence is over-excited at the expense of flexibility.

Growing evidence links creative cognition and performance, along with the regulation of the balance between flexibility and persistence, to activation in the fronto-striatal circuitry. This circuitry is comprised of several distinct but interconnected brain areas, two of which are particularly relevant to creativity: the striatum and the PFC. The subcortical striatum, the primary input structure of the basal ganglia, is involved in diverse functions, such as reward processing and habit learning, as well as flexibility-supporting processes including the updating of goal representations and the shifting of task strategies (Ikemoto, 2007; Kehagia et al., 2010). The PFC is generally known for processes associated with executive control of behavior, such as the maintenance of goal and task representations in working memory, planning, attentional control, and the suppression of distractors (Kane and Engle, 2002; Miller and Cohen, 2001). It is involved in persistence, controlled manipulation of information and executive functioning (Barr et al., 2014; Benedek et al., 2014; Dietrich and Kanso, 2010; Gonen-Yaacovi et al., 2013; Metuki et al., 2012).

If creative performance requires and relies on a proper balance between flexibility and persistence, and flexibility and persistence are associated with striatal and prefrontal activation, it follows that creative cognition and performance requires a balance within the fronto-striatal network, rather than excessive activation within either the striatum or the PFC. Indeed, outside of the domain of creative cognition, growing evidence reveals that the PFC and striatum are strongly interconnected (Alexander et al., 1986), and that the balance between persistent maintenance of goal and task representations in working memory, and the flexible updating of goal representations and shifting of task strategies, is conditioned by interactions between these brain areas (Cools et al., 2007; Frank et al., 2001; McNab and Klingberg, 2008). Furthermore, recent work indicates that divergent thinking is associated with increased gray matter density in several fronto-striatal areas, including the caudate nucleus of the striatum (Jauk et al., 2015; Takeuchi et al., 2010a), and stronger connectivity between the striatum and frontal areas (Erhard et al., 2014; Takeuchi et al., 2010b).

### 3.2. Dopaminergic pathways in the fronto-striatal network

Critical in this interplay between the striatum and the PFC is the neurotransmitter dopamine (Frank et al., 2001; Goldman-Rakic, 1992; Kellendonk et al., 2006; Krugel et al., 2009; Meyer-Lindenberg et al., 2005; Wallace et al., 2011). Synthesized in the subcortical ventral tegmental area (VTA) and substantia nigra, dopamine is projected onto a number of brain areas, including the striatum and PFC, via various dopaminergic pathways (Alexander et al., 1986). Together, as schematically illustrated in Fig. 1, these pathways form a complex network that regulates itself through reciprocal connections between brain areas and inhibitory autoreceptors.

Within this dopaminergic network, dopamine is projected onto striatal and PFC regions via the nigrostriatal and the mesocortical pathway (e.g., Durstewitz and Seamans, 2008; Frank et al., 2001).[4] The nigrostriatal pathway regulates dopamine levels first and foremost in the dorsal striatum in the basal ganglia (Alexander et al., 1986). There it facilitates attentional shifts and updating when new information in the environment becomes available (Frank et al., 2001; Kehagia et al., 2010; Leber et al., 2008; Meyer-Lindenberg et al., 2005). The mesocortical pathway, in contrast, originates in the VTA and innervates prefrontal areas where it supports working memory processes and sustained attention (Goldman-Rakic, 1992). Accordingly, dopamine levels in the PFC facilitate cognitive control and reduce distractibility (Durstewitz and Seamans, 2008; Vijayraghavan et al., 2007).

Much as increased flexibility seems to pair to reduced persistence and vice versa (Nijstad et al., 2010), increasing levels of dopamine in the striatum seem to associate with decreases in dopamine levels in the PFC, and vice versa (Akil et al., 2003; Meyer-Lindenberg et al., 2005). Put differently, within the fronto-striatal network, there appears to be a flexibility-persistence tradeoff that is modulated by striatal dopamine relative to prefrontal dopamine (Cools et al., 2007; Cools and D'Esposito, 2011; Dodds et al., 2008; Frank, 2005; Wallace et al., 2011). Evidence for this comes from studies that examined the effects of specific dopamine-enhancing drugs. Some drugs stimulate D1-type receptors, the most abundant receptor type in the PFC (Lidow et al., 1991), whereas other drugs stimulate D2-type receptors, which is most prevalent in the sub-cortical areas and the striatum (Camps et al., 1989). Whereas administering D1-stimulating drugs enhances working memory capacity and cognitive control (Durstewitz and Seamans, 2008; Vijayraghavan et al., 2007), D2-stimulating drugs promote cognitive flexibility (Dodds et al., 2009; Mehta et al., 2004). Moreover, whereas *overstimulating D1 receptors* results in rigid, perseverative thinking—new information is blocked and screened out and mental representations are no longer updated (Durstewitz and Seamans, 2008; Vijayraghavan et al., 2007), *overstimulating D2 receptors* associates with exceeding levels of distractibility (Durstewitz and Seamans, 2008; Kellendonk et al., 2006).

This dopamine-conditioned flexibility-persistence tradeoff within the fronto-striatal network has three important implications for our understanding of creative performance. The first implication is that when striatal dopamine exceeds prefrontal dopamine, the fronto-striatal network biases towards flexibility (Cools et al., 2007; Dodds et al., 2008), and creative performance may be the outcome of flexible rather than persistent processing. When, in contrast, prefrontal dopamine exceeds striatal dopamine, the fronto-striatal network biases towards persistence, and creative performance may be the outcome of persistent rather than flexible processing. Second, the balance between flexibility and stability is disturbed when dopaminergic activity in either the PFC or striatum is too high or low. That is, too much (little) dopaminergic activity in the striatum (PFC) may lead to distractibility, whereas too much (little) dopaminergic activity in the PFC (striatum) may lead to rigidity. This implies an inverted-U-shaped function between fronto-striatal dopamine and creative performance (see Fig. 1), so that intermediate levels of dopamine optimize task performance because of a proper balance between flexibility and persistence. Exceedingly low or high levels of dopamine impair performance because they drive either towards distractibility (i.e., flexibility without persistence), or to rigidity (persistence without flexibility) (also see Cools and D'Esposito, 2011; Durstewitz and Seamans, 2008; Vijayraghavan et al., 2007).

The third implication is that manipulating dopamine levels can have opposing effects in healthy participants with high and low initial dopamine levels (Arnsten, 1998; Cools and D'Esposito, 2011; Seamans and Yang, 2004). Whereas adding dopamine to someone with high baseline dopamine may result in distractibility, the same dose may optimize performance in someone with low baseline dopamine. For example, in patients with early Parkinson's disease, nigrostriatal dopamine levels are impeded while the mesocortical pathway is largely intact. Without medication, these patients show reduced cognitive flexibility relative to healthy controls. When treated with dopamine-triggering drugs, such impaired flexibility can be largely rescued (Cools et al., 2001; Frank, 2005; Swainson et al., 2000). Taken together, creative cognition is a function of the interaction between flexibility and persistence, which is controlled by dopaminergic modulation in fronto-striatal regions of the human brain. When striatal dopamine exceeds prefrontal dopamine, the fronto-striatal network biases towards

[4] Along with the mesocortical pathway, the mesolimbic pathway projects from the VTA to limbic areas and the ventral striatum. Mesolimbic DA mediates processes associated with appetitive motivation and reward prediction (Ikemoto, 2007; Schultz, 2002), and may indirectly influence creative performance through motivational processes. We will return to this issue in the discussion.

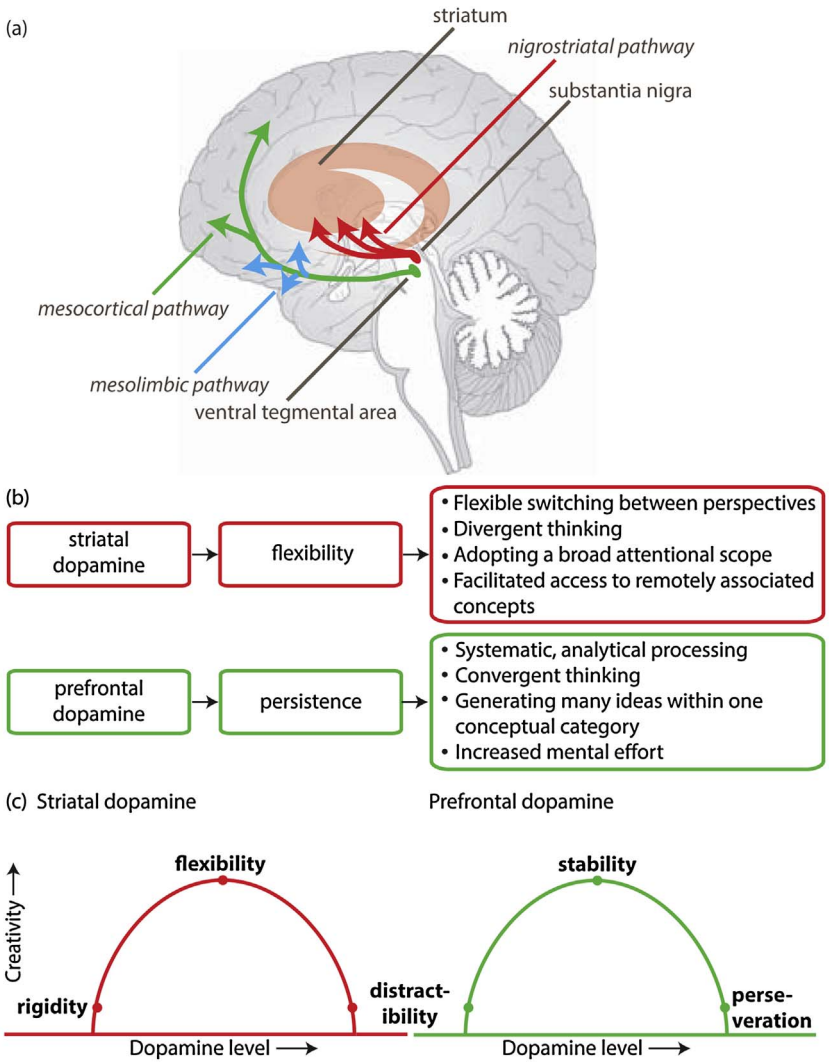


**Fig. 1.** Main dopaminergic networks in the brain and their proposed role in flexible and persistent creative processes. (a) Main dopaminergic pathways in the brain. (b) Proposed model of dopaminergic modulation of creativity through processes related to flexibility and persistence. (c) Hypothesized relationships between striatal and prefrontal dopamine levels and creative performance.

flexibility, and creative performance may result particularly from flexible processing and when prefrontal dopamine exceeds striatal dopamine, the fronto-striatal network biases towards persistence, and creative performance may result particularly from persistent processing. However, overexciting nigrostriatal dopamine relative to mesocortical dopamine links to over-excited flexibility relative to persistence, with distractibility and "weird" ideas as a probable outcome. Overexciting mesocortical dopamine relative to nigrostriatal dopamine links to over-excited persistence relative to flexibility, with rigidity of thought and "boring" ideas as a probably outcome. Put differently: the balance between flexibility and stability is disturbed when dopaminergic activity in either the PFC or striatum is too high or low. That is, too much (little) dopaminergic activity in the striatum (PFC) may lead to increased distractibility and "weird" ideas, whereas too much (little) dopaminergic activity in the PFC (striatum) may lead to increased rigidity and "boring" ideas.

## 4. Evidence for dopaminergic modulation of creative performance

Different lines of research support the possibility that fronto-striatal dopaminergic activity links to flexibility and persistence, and to creative cognition and performance in an inverted-U-shaped fashion (per Fig. 1). This section reviews this work. We begin with studies suggesting the involvement of striatal dopamine in modulating flexible processes, followed by a review of preliminary evidence suggesting a role for prefrontal dopamine in modulating persistent processes. These studies often target different aspects of the dopaminergic modulation of (interactions among) neural circuitries. Some studies examined (genetic and behavioral markers of) dopamine receptor availability, allowing for a rather precise inference about the functioning of the nigrostriatal versus mesocortical dopamine in creative cognition and performance. Other studies compare medication with dopamine agonist or antagonist to placebo treatments, thus allowing for causal inferences and, in some cases, inferences about the specific role of (nigro)striatal versus mesocortical and prefrontal dopamine. Finally, there are studies comparing healthy controls to patients with a disorder with strong dopaminergic dysregulation (e.g., Parkinson's disease, bipolar disorder; Johnson et al., 2012; Polner et al., 2015). Although each approach has its limitations, the available evidence combined points to a functional differentiation between striatal and prefrontal dopamine in modulating flexible and persistent creative processes, with creative performance as its key end-state.

### 4.1. Dopaminergic modulation of flexibility

First evidence for dopaminergic modulation of creativity in the

striatum comes from studies showing that dopamine D2 receptor functioning is related to creative performance on standardized tests. Although D2 receptors are also present in the PFC, they are 11 times more prevalent in the striatum (Camps et al., 1989). The role of dopaminergic D2 receptors in creativity is supported by findings from genetic studies showing that the dopamine D2 (DRD2) receptor gene is associated with enhanced verbal fluency, flexibility, and originality during divergent thinking (Reuter et al., 2006; Zhang et al., 2014a). Other studies considered spontaneous eye-blink rate—an indirect marker of D2 receptor availability (Groman et al., 2014). Resonating with the established inverted-U-shaped relationship between striatal dopamine and cognitive flexibility (e.g., Kellendonk et al., 2006), Chermahini and Hommel (2010, 2012) observed that participants with medium eye blink rates showed more flexibility in divergent thinking, whereas participants with either low or high eye blink rates were less flexible in divergent thinking. Finally, positive mood induction, presumably associated with increased dopamine release in the brain (Ashby et al., 1999; Dreisbach and Goschke, 2004), increased both eye blink rates and flexible divergent thinking, but in individuals with low baseline dopamine levels only (Chermahini and Hommel, 2012). Together, these studies suggest that dopamine D2 receptor functioning, which most strongly affects striatal activity, associates with creativity through flexibility and this relationship follows an inverted-U-shaped function. Further supporting this suggestion, a recent study showed that the effect of methylphenidate (a psychostimulant that enhances both dopamine and noradrenaline levels in the brain; Kuczenski and Segal, 1997) on divergent thinking depended on baseline levels of novelty seeking, a personality trait that is often associated with dopaminergic functioning (Gvirts et al., 2016; Depue and Collins, 1999). In this study, methylphenidate (vs. placebo) administration improved creativity in participants with low baseline levels of novelty seeking, but impaired performance in participants with high baseline levels of novelty seeking (Gvirts et al., 2016).

Other preliminary evidence for the role of striatal dopamine in flexibility and creativity comes from treatment studies in Parkinson's disease. In addition to deficient levels of noradrenaline (Scatton et al., 1983), patients with Parkinson's disease have depleted nigrostriatal dopamine concentrations, and impaired cognitive flexibility and creative flexibility on standardized tests (Canesi et al., 2012; Cools et al., 2001; Swainson et al., 2000). This flexibility impairment can be remedied by treatment with dopaminergic medication (Cools et al., 2001; Swainson et al., 2000), with enhanced creative performance as a possible outcome (Inzelberg, 2013). For example, in patients with beginning Parkinson's disease but not in healthy controls, divergent thinking abilities improved after they started taking dopaminergic medication (Polner et al., 2015), and medicated Parkinson's disease patients generated even more original ideas on a divergent thinking task than healthy controls (Faust-Socher et al., 2014).

### 4.2. Dopaminergic modulation of persistence

A number of studies established a link between creativity and dopaminergic polymorphisms, genetic variations that result in individual differences in dopamine functioning. Mayseless et al. (2013) compared non-carriers to carriers of the 7-repeat (7R) allele of the dopamine D4 (DRD4) receptor gene. This receptor gene is predominantly (but not exclusively) expressed in cortical areas and non-carriers have presumably higher prefrontal dopamine receptor functioning (Langley et al., 2004). Non-carriers were less flexible during idea generation, but because the originality of their ideas was left intact (Mayseless et al., 2013), although speculative, their original thinking most likely resulted from enhanced ability to persist.

Several other studies examined the creative correlates of genetic differences in the efficacy of catechol-O-methyltransferase (COMT), an enzyme that is involved in the breakdown of prefrontal dopamine (Matsumoto et al., 2003). The COMT Val158Met polymorphism influences cognitive control and working memory (Malhotra et al., 2002). In carriers of the low-activity COMT Val158*met* allele, prefrontal dopamine levels are higher than in carriers of the more active COMT Val158*val* allele. Low activity *met* carriers were less flexible than *val* carriers in adapting their behavior to changing task demands (Krugel et al., 2009), yet more fluent (Murphy et al., 2013; Runco et al., 2011; Zabalina et al., 2016) and original (Zhang et al., 2014b; Zabalina et al., 2016) during standardized creative tasks. In other words, those individuals with genetic predisposition to high prefrontal dopaminergic activity appear less flexible but more fluent and original in idea generation.

Dopaminergic modulation of persistence is further suggested by a study investigating the effects of tyrosine administration on convergent and divergent creative processes (Colzato et al., 2014). In addition to increasing noradrenaline levels in the brain (Hase et al., 2015), the amino acid L-Tyrosine acts as a precursor of dopamine (Acworth et al., 1988; Hase et al., 2015). Tyrosine counteracts the cognitive effects of sleep deprivation (Magill et al., 2003) and facilitates executive control during a cognitively demanding task (Colzato et al., 2013). Accordingly, tyrosine intake enhanced convergent but not divergent thinking on creative insight tasks (Colzato et al., 2014), suggesting that tyrosine supported, through enhanced prefrontal dopamine, the more persistence-related processes underlying creative performance.

### 4.3. Clues from creative processes in (Sub)clinical populations

If dopamine availability indeed modulates creativity and its underlying cognitive processes, we would expect chronic disturbances in the dopaminergic system to be associated with predictable deficits or improvements in creative performance. Two broad classes of psychiatric disorders—schizophrenia and schizotypal traits, and bipolar mood disorders—are well documented to exhibit dopaminergic abnormality. Schizophrenia is a mental illness that is characterized by psychotic symptoms (hallucinations and delusions; also termed positive symptoms), flattened affect and apathy (termed negative symptoms) and cognitive impairments, such as disorganized thought and working memory deficits (Laruelle et al., 2003). These symptoms result from extensive deregulation of the dopaminergic system. Dopaminergic hyperactivity in the striatum presumably underlies the positive symptoms of schizophrenia, whereas dopaminergic hypo-activity in the PFC has been proposed to give rise to negative symptoms and cognitive impairments (Durstewitz and Seamans, 2008; Kellendonk et al., 2006; Laruelle et al., 2003).

People with subclinical schizotypal traits (e.g., mild levels of perceptual aberrations, hallucinations, and eccentric behavior) have minor dopaminergic deregulation, as compared to those with full-blown schizophrenia. On standardized tests, these sub-clinical types display enhanced creative flexibility relative to healthy controls. For example, research shows striatal involvement in low latent inhibition, the lowered capability to filter out from attentional focus those stimuli that were previously experienced as irrelevant (Fletcher and Frith, 2009; Swerdlow et al., 2003). Low latent inhibition is associated with higher creativity through flexibility: during a creativity task, more seemingly irrelevant concepts and information enter working memory, which in turn increases the span of elements to work with, leading to more flexible and original responses (e.g., Acar and Sen, 2013; Carson et al., 2003). Importantly, low latent inhibition—which typically facilitates flexibility—is not only observed in psychotic schizophrenia patients (Baruch et al., 1988a), but also in healthy participants with a high number of schizotypal personality traits (Baas et al., 2016; Baruch et al., 1988b). Interestingly, however, the relationship between these schizotypal traits and creativity follows an inverted-U-shaped function. Subclinical positive symptoms associate with enhanced creativity (Acar and Sen, 2013; Carson et al., 2003; Folley and Park, 2005), whereas more severe schizophrenic symptoms associate with impaired creative performance on both verbal and visual divergent thinking tasks

(Abraham et al., 2007).

A similar inverted U-shaped relationship between dopamine availability and creativity is seen in studies involving individuals with bipolar disorder. In bipolar spectrum disorders, such as bipolar II disorder and cyclothymia, people alternate between episodes of depressed mood and hypomania (Berk and Dodd, 2005). During (hypo) manic episodes, patients are extremely energetic, sociable, impulsive, easily distracted, and have a decreased need for sleep (Johnson et al., 2012). People with (hypo)manic symptoms have impaired working memory and executive function (Cousins et al., 2009; Martínez-Arán et al., 2004), are highly sensitive to external reward cues and engage in more goal-directed activities (Alloy et al., 2008; O'Sullivan et al., 2011). It is typically associated with a hyperdopaminergic state in the brain and with structural differences in dopaminergic areas, including the striatum (Cousins et al., 2009; O'Sullivan et al., 2011; Strakowski et al., 2005). Healthy participants who report a large number of hypomanic symptoms report and show more creative performance than people without such symptoms (Baas et al., 2016; Furnham et al., 2008; Johnson et al., 2012). However, those individuals with more severe symptoms show similar levels of creative performance compared to people without such symptoms (Richards et al., 1988; Santosa et al., 2007; Srivastava et al., 2010).

Taken together, the results of studies with schizophrenic and schizotypal, as well as bipolar spectrum disorder, all resonate with the possibility that creative outcomes are conditioned by fronto-striatal dopamine in an inverted U-shaped fashion. While psychopathologies associate with disturbances in a range of neurobiological, cognitive, and motivational variables (Cousins et al., 2009; Ettinger et al., 2014), these studies provide otherwise convergent evidence for the possibility that dopamine modulates creative cognition and performance.

## 5. Summary and avenues for research

The flexibility and persistence pathways underlying creative performance (Nijstad et al., 2010) can be meaningfully traced to activation within and between striatal and prefrontal regions within the human brain, and dopaminergic modulation thereof. More specifically, nigrostriatal dopamine facilitates flexibility and controls the flow of new information into working memory, allowing people to switch between different task approaches and to consider more remotely associated concepts (cf. Dodds et al., 2009; Frank et al., 2001; McNab and Klingberg, 2008). Mesocortical dopamine, on the other hand, supports prefrontal functionalities such as the maintenance and manipulation of representations in working memory (De Dreu et al., 2012; Durstewitz and Seamans, 2008; Frank et al., 2001), allowing for persistence.

### 5.1. New insights

The present analysis provides three non-trivial insights. First, the relationships between dopamine and flexible and persistent creative processes seem to follow an inverted-U-shaped function: Too much (little) dopaminergic activity in the striatum (PFC) manifests in distractibility, whereas too much (little) dopaminergic activity in the PFC (striatum) manifests in rigidity (cf. Kellendonk et al., 2006; Seamans and Yang, 2004; Vijayraghavan et al., 2007). Both distractibility and rigidity are antithetic to creative cognition and performance. Rather than "just" dopamine, or "just" striatal or prefrontal activation, creative cognition and performance requires a balance within the dopaminergic fronto-striatal network, and depends on interactions among nigrostriatal and mesocortical dopamine. This leads to the second implication: The effects of increasing or reducing dopamine in the brain strongly depends on the participants' baseline dopamine levels. Specifically, up-regulating striatal dopamine in individuals with high (low) baseline levels can bias cognition and performance away from (towards) creativity and into (away from) distractibility. Likewise, up-regulating mesocortical dopamine in individuals with high (low) baseline levels can bias cognition and performance away from (toward) creativity and into (away from) rigid perseverance. Understanding this is crucial in new research, to which we turn shortly.

Because dopaminergic interventions interact with individual baseline levels of dopamine, a third and more actionable implication is that dopaminergic drugs, such as methylphenidate and amphetamine, provided to healthy people can both reduce or enhance creative performance (cf. Farah et al., 2009; Gvirts et al., 2016; Mehta et al., 2004). Likewise, whereas some neuropsychiatric disorders that are associated with excessive low creative thinking may benefit from dopamine treatment, such treatment may have adverse effects in disorders and pathologies that associate with high creativity. For example, individuals diagnosed with depression are typically low in creativity and flexible thinking (Baas et al., 2016), and may benefit, in terms of creative performance, from dopaminergic medication. Individuals diagnosed with mania, in contrast, are typically more flexible and creative (Baas et al., 2016) and may become exceedingly distractible when given dopaminergic medication.

The framework presented here fits and integrates scattered evidence from studies looking at genetic markers of dopamine receptors, effects of drug administration, indirect markers of dopaminergic activation, and psychopathological disturbances. At the same time, however, the evidence is oftentimes indirect, and parts of the framework receive stronger support than others. First, the available evidence is not always clearly distinguishable between the role of dopamine from that of other neuromodulators such as noradrenaline and serotonin. This is especially the case when considering neuropsychological pathologies and disorders, that typically have high co-morbidity (Baas et al., 2016; Borsboom and Cramer, 2013) and in which multiple neural systems and circuitries are involved and dysregulated. Another example is the finding that intranasal administration of oxytocin – a neuropeptide implicated in enhanced approach and reduced withdrawal tendencies (Carter, 2014; De Dreu and Kret, 2016) – promotes flexibility, divergent thinking, and creative insight performance (De Dreu et al., 2014). Most likely such creativity-promoting effects of oxytocin can be attributed to its effects on dopaminergic modulation in the striatum (De Dreu et al., 2015; Donaldson and Young, 2008), but it cannot be excluded that it (also) emerges because of reduced cortisol responses and fear signaling in the amygdalar-hippocampal circuit (Meyer-Lindenberg et al., 2011).

### 5.2. New research targets

Two specific targets of new research can be identified. First and foremost, new research is needed that focuses on obtaining *direct* evidence for dopamine involvement in creative processes. This could be achieved by manipulating dopamine levels in healthy individuals using dopaminergic agonists and antagonists, ideally by combining them in pretreatment studies (e.g., Gvirts et al., 2016; Piray et al., 2016; Van der Schaaf et al., 2014). Earlier work successfully used these methods to investigate the role of dopamine in cognitive flexibility (Alexander et al., 2007), latent inhibition (Swerdlow et al., 2003), and working memory (Cools et al., 2007). A next generation of studies could advance these basic insights to directly measure creative performance. Such new work could specifically test the hypothesis that drugs that predominantly elevate or decrease striatal dopamine levels will impact flexible thinking, with different effects in participants with high and low baseline dopamine levels (e.g., Chermahini and Hommel, 2012). Similarly, drugs that particularly regulate prefrontal dopamine can be expected to alter persistence-related processes, again depending on participants' baseline dopamine levels (Cools and D'Esposito, 2011; Durstewitz and Seamans, 2008).

Second, the present framework points to specific interactions between neuropharmacological manipulations of striatal and prefrontal dopamine levels on the one hand, and situational factors on the other. Especially the persistence pathway to creativity hinges on the availability of working memory capacity, and is for example blocked or

harmed by cognitive load and time pressure (De Dreu et al., 2012; Roskes et al., 2012). Accordingly, up-regulating prefrontal dopamine should promote creative performance, through enhanced persistent processing, more when working memory capacity is intact rather than loaded and when time pressure is mild rather than severe. In contrast, the flexibility pathway is easily activated when individuals feel happy, when an approach-orientation is activated, or when individuals adopt a long-term rather than short-term time-perspective (e.g., Baas et al., 2008; Cretenet and Dru, 2009; De Dreu et al., 2008, 2011). Under such benign circumstances, neuropharmacological enhancement of striatal dopamine may result in mere distractibility, rather than creativity-conducive flexibility.

### 5.3. Beyond the fronto-striatal network

We focused here on dopamine as the key neurotransmitter involved in creative cognition and behavior, and in particular dopaminergic modulation of the nigrostriatal and mesocortical pathways. We left aside the mesolimbic dopamine pathway, which also projects to parts of the striatum and prefrontal cortex (Alexander et al., 1986), and may influence creativity through its role in reward processing and appetitive motivation (Ikemoto, 2007; Schultz, 2002). Approach orientation—in which behavior is regulated towards rewarding stimuli—associates with dopaminergic activation (Depue and Collins, 1999), and is associated with greater flexibility, originality, and better insight performance when temporarily induced (Baas et al., 2011; Cretenet and Dru, 2009; Roskes et al., 2012), or measured as a chronic trait (Baas et al., 2013; Carver and White, 1994; De Dreu et al., 2011; Furnham et al., 2008). Thus, whereas the nigrostriatal and mesocortical dopamine pathways condition creative cognition, dopaminergic modulation of the mesolimbic pathway may be primarily involved in the motivation to perform creatively.

In addition to dopamine and dopaminergic circuitries, human behavior and cognition is modulated by other brain circuitries and a range of other neurotransmitters including, for example, serotonin, noradrenaline and acetylcholine (Cools, 2012; Robbins and Roberts, 2007). We singled-out dopamine because of the wealth of research on non-human animal behavior aligning with more recent work on human cognitive performance, because of the growing evidence for the role of dopaminergic modulation of creative cognition and performance and, finally, because of the specific function of dopamine in the set of neural structures that have been implicated in human creativity. However, while dopamine emerges as a prime candidate neurotransmitter influencing human creativity, future work should not shy away from exploring the role of other neurochemicals (Arnsten, 1998; Cools, 2012; De Dreu et al., 2015; Robbins and Roberts, 2007). In fact, prefrontal noradrenaline is thought to modulate the balance between “exploration” and “exploitation” behavior by facilitating both stabilization of salient information in working memory and attentional shifts to alternative opportunities in the environment (Aston-Jones and Cohen, 2005; Berridge and Waterhouse, 2003) and may enable the persistence pathway to creativity. In addition, creative ideas are not generated in a vacuum but build on existing knowledge and in interaction with the external environment (Baer, 2016; Nijstad et al., 2010). This requires the activation of other brain structures, including the parietal–temporal regions, that are involved in the processing of sensory input, memory retrieval, and language processing (e.g., Abraham et al., 2012; De Manzano et al., 2010; Shamay-Tsoory et al., 2011; Takeuchi et al., 2010b). Ultimately, creative cognition requires the dynamic interactions of large-scale brain systems (cf. Beaty et al., 2016).

### 5.4. Measuring creative cognition

Aside from the specific roles of dopamine in the fronto-striatal circuitry, our review highlights the importance of the way creativity is operationalized and measured. For example, standardized creativity tasks that capitalize on convergent and persistent processing, such as the remote associates test, may benefit more from neuromodulation of the PFC than creativity tasks that more strongly rely on flexibility and divergent thinking. In addition, tracking the involvement of dopamine and other neurotransmitters in creative, divergent and persistent thinking either requires the manipulation of neurotransmitters in which treated subjects perform creativity tasks. Alternatively, it can be tracked with PET studies (cf. De Manzano et al., 2010), although it could be difficult to fit the measurement of creative processes with the requirements of PET imaging (Abraham and Windmann, 2007; Boot et al., 2017). PET has very low temporal resolution and it requires the selection of suitable comparison tasks, which is challenging. In either case, a theory-driven, calibrated choice of tasks, designs, and procedures is necessary to further understand the sometimes inconsistent findings on the neurocognitive underpinnings of creativity (Dietrich and Kanso, 2010).

## 6. Concluding remarks

Creative performance is a function of both flexible and persistent processing, and the extent to which these pathways are engaged differs between individuals, and is conditioned by situational factors. Both individual differences in, and the temporary impact of situational factors on flexibility and persistence may be understood in terms of regulatory processes within and between components of the fronto-striatal network. Whereas dopaminergic activity in the striatum enables flexibility, dopaminergic modulation of the prefrontal cortex permits stability and persistence. Chronic disturbances in striatal and/or prefrontal dopamine regulation may be key to individual differences in creative performance, with severe dopaminergic dysregulation accounting for excessively low or high creativity in patients with, for example, Parkinson’s disease or schizophrenia. Likewise, more transient states such as mood, fatigue, or time pressure, permit or inhibit creativity because of dopaminergic activity in the striatum and/or the prefrontal cortex. Importantly, over-activation of the striatum relative to the prefrontal cortex manifesting itself in exceeding levels of distractibility, and under-activation leading to perseverant rigidity of thought. As such, rather than “just” increased dopamine regulation of striatal or prefrontal circuitries, it is the fine balance in dopaminergic regulation of the fronto-striatal network that permits and enables individuals to think and perform creatively.

## References

Abraham, A., Windmann, S., 2007. Creative cognition: the diverse operations and the prospect of applying a cognitive neuroscience perspective. Methods 42, 38–48.
Abraham, A., Windmann, S., McKenna, P., Güntürkün, O., 2007. Creative thinking in schizophrenia: the role of executive dysfunction and symptom severity. Cogn. Neuropsychiatry 12, 235–258.
Abraham, A., Beudt, S., Ott, D.V., von Cramon, D.Y., 2012. Creative cognition and the brain: dissociations between frontal, parietal–temporal and basal ganglia groups. Brain Res. 1482, 55–70.
Acar, S., Sen, S., 2013. A multilevel meta-analysis of the relationship between creativity and schizotypy. Psychol. Aesthet. Creat. Arts 7, 214–228.
Acworth, I.N., During, M.J., Wurtman, R.J., 1988. Tyrosine: effects on catecholamine release. Brain Res. Bull. 21, 473–477.
Akil, M., Kolachana, B.S., Rothmond, D.A., Hyde, T.M., Weinberger, D.R., Kleinman, J.E., 2003. Catechol-O-methyltransferase genotype and dopamine regulation in the human brain. J. Neurosci. 23, 2008–2013.
Akinola, M., Mendes, W.B., 2008. The dark side of creativity: biological vulnerability and negative emotions lead to greater artistic creativity. Pers. Soc. Psychol. Bull. 34, 1677–1686.
Alexander, G.E., DeLong, M.R., Strick, P.L., 1986. Parallel organisation of functionally segregated circuits linking basal ganglia and cortex. Annu. Rev. Neurosci. 9, 357–381.
Alexander, J.K., Hillier, A., Smith, R.M., Tivarus, M.E., Beversdorf, D.Q., 2007. Beta-adrenergic modulation of cognitive flexibility during stress. J. Cogn. Neurosci. 19, 468–478.
Alloy, L.B., Abramson, L.Y., Walshaw, P.D., Cogswell, A., Grandin, L.D., Hughes, M.E., Iacoviello, B.M., Whitehouse, W.G., Urosevic, S., Nusslock, R., Hogan, M.E., 2008. Behavioral approach system and behavioral inhibition system sensitivities and bipolar spectrum disorders: prospective prediction of bipolar mood episodes. Bipolar

Disord. 10, 310–322.
Amabile, T.M., 1996. Creativity in Context. Westview Press, Boulder, CO.
Arnsten, A.F.T., 1998. Catecholamine modulation of prefrontal cortical cognitive function. Trends Cogn. Sci. 2, 436–447.
Ashby, F.A., Isen, A.M., Turken, A.U., 1999. A neuropsychological theory of positive affect and its influence on cognition. Psychol. Rev. 106, 529–550.
Aston-Jones, G., Cohen, J.D., 2005. An integrative theory of locus coeruleus-norepinephrine function: adaptive gain and optimal performance. Annu. Rev. Neurosci. 28, 403–450.
Baas, M., De Dreu, C.K.W., Nijstad, B.A., 2008. A meta-analysis of 25 years of mood-creativity research: hedonic tone, activation, or regulatory focus? Psychol. Bull. 134, 779–806.
Baas, M., De Dreu, C.K.W., Nijstad, B.A., 2011. When prevention promotes creativity: the role of mood, regulatory focus: and regulatory closure. J. Pers. Soc. Psychol. 100, 794–809.
Baas, M., Roskes, M., Sligte, D., Nijstad, B.A., De Dreu, C.K.W., 2013. Personality and creativity: the dual pathway to creativity model and a research agenda. Soc. Personal. Psychol. Compass 7, 732–748.
Baas, M., Boot, N.C., Nijstad, B.A., De Dreu, C.K.W., 2016. Mad genius revisited: vulnerability to psychopathology, biobehavioral approach-avoidance, and creativity. Psychol. Bull. 142, 668–692. http://dx.doi.org/10.1037/bul0000049.
Baer, J., 2011a. How divergent thinking tests mislead us: are the Torrance Tests still relevant in the 21 st century? The Division 10 debate. Psychol. Aesthet. Creat. Arts 5, 309–313.
Baer, J., 2011b. Four (more) arguments against the Torrance tests. Psychol. Aesthet. Creat. Arts 5, 316–317.
Baer, J., 2016. Creativity doesn't develop in a vacuum. New Dir. Child Adolesc. Dev. 151, 9–20.
Barr, N., Pennycook, G., Stolz, J.A., Fugelsang, J.A., 2014. Reasoned connections: a dual-process perspective on creative thought. Think. Reason 21, 61–75.
Baruch, I., Hemsley, D.R., Gray, J.A., 1988a. Differential performance of acute and chronic schizophrenics in a latent inhibition task. J. Nerv. Ment. Dis. 176, 598–606.
Baruch, I., Hemsley, D.R., Gray, J.A., 1988b. Latent inhibition and psychotic proneness in normal subjects. Pers. Individ. Dif. 9, 777–783.
Beaty, R.E., Benedek, M., Silvia, P.J., Schacter, D.L., 2016. Creative cognition and brain network dynamics. Trends Cogn. Sci. 20, 87–95.
Benedek, M., Beaty, R., Jauk, E., Koschutnig, K., Fink, A., Silvia, P.J., Dunst, B., Neubauer, A.C., 2014. Creating metaphors: the neural basis of figurative language production. NeuroImage 90, 99–106.
Berk, M., Dodd, S., 2005. Bipolar II disorder: a review. Bipolar Disord. 7, 11–21.
Berridge, C.W., Waterhouse, B.D., 2003. The locus coeruleus–noradrenergic system: modulation of behavioral state and state-dependent cognitive processes. Brain Res. Rev. 42, 33–84.
Boot, N.C., Baas, M., De Dreu, C.K.W., Van Gaal, S., 2017. Widespread Neural Oscillations in the Delta Band Dissociate Convergent from Divergent Ideation During Creative Idea Generation. Unpublished Manuscript. University of Amsterdam.
Borsboom, D., Cramer, A.O.J., 2013. Network analysis: an integrative approach to the structure of psychopathology. Annu. Rev. Clin. Psychol. 9, 91–121.
Bowden, E.M., Jung-Beeman, M., Fleck, J., Kounios, J., 2005. New approaches to demystifying insight. Trends Cogn. Sci. 9, 322–328.
Camps, M., Cortés, R., Gueye, B., Probst, A., Palacios, J.M., 1989. Dopamine receptors in human brain: autoradiographic distribution of D2 sites. Neuroscience 28, 275–290.
Canesi, M., Rusconi, M.L., Isaias, I.U., Pezzoli, G., 2012. Artistic productivity and creative thinking in Parkinson's disease. Eur. J. Neurol. 19, 468–472.
Carson, S.H., Peterson, J.B., Higgins, D.M., 2003. Decreased latent inhibition is associated with increased creative achievement in high-functioning individuals. J. Pers. Soc. Psychol. 85, 499–506.
Carson, S., Peterson, J.B., Higgins, D.M., 2005. Reliability, validity, and factor structure of the Creative Achievement Questionnaire. Creat. Res. J. 17, 37–50.
Carter, C.S., 2014. Oxytocin pathways and the evolution of human behavior. Annu. Rev. Psychol. 65, 17–39.
Carver, C.S., White, T.L., 1994. Behavioral inhibition, behavioral activation: and affective responses to impending reward and punishment: the BIS/BAS scales. J. Pers. Soc. Psychol. 67, 319–333.
Cerruti, C., Schlaug, G., 2009. Anodal transcranial direct current stimulation of the prefrontal cortex enhances complex verbal associative thought. J. Cogn. Neurosci. 21, 1980–1987.
Chermahini, S.A., Hommel, B., 2010. The (b)link between creativity and dopamine: spontaneous eye blink rates predict and dissociate divergent and convergent thinking. Cognition 115, 458–465.
Chermahini, S.A., Hommel, B., 2012. More creative through positive mood? Not everyone!. Front. Hum. Neurosci. 6, 1–7.
Colzato, L.S., Jongkees, B.J., Sellaro, R., Hommel, B., 2013. Working memory reloaded: tyrosine repletes updating in the N-back task. Front. Behav. Neurosci. 7, 1–5.
Colzato, L.S., de Haan, A.M., Hommel, B., 2014. Food for creativity: tyrosine promotes deep thinking. Psychol. Res. 79, 709–714.
Cools, R., D'Esposito, M., 2011. Inverted-U-shaped dopamine actions on human working memory and cognitive control. Biol. Psychiatry 69, e113–25.
Cools, R., Barker, R.A., Sahakian, B.J., Robbins, T.W., 2001. Enhanced or impaired cognitive function in Parkinson's disease as a function of dopaminergic medication and task demands. Cereb. Cortex 11, 1136–1143.
Cools, R., Sheridan, M., Jacobs, E., D'Esposito, M., 2007. Impulsive personality predicts dopamine-dependent changes in frontostriatal activity during component processes of working memory. J. Neurosci. 27, 5506–5514.
Cools, R., 2012. Chemical neuromodulation of goal-directed behavior. In: Todd, P.M., Hills, T.T., Robbins, T.W. (Eds.), Cognitive Search: Evolution, Algorithms, and the Brain. Cambridge University Press, Cambridge, MA, pp. 111–124.
Cousins, D.A., Butts, K., Young, A.H., 2009. The role of dopamine in bipolar disorder. Bipolar Disord. 11, 787–806.
Cretenet, J., Dru, V., 2009. Influence of peripheral and motivational cues on rigid-flexible functioning: perceptual, behavioral, and cognitive aspects. J. Exp. Psychol. Gen. 138, 201–217.
Cropley, A., 2006. In praise of convergent thinking. Creat. Res. J. 18, 391–404.
De Dreu, C.K.W., Kret, M.E., 2016. Oxytocin conditions intergroup relations through upregulated in-group empathy, cooperation, conformity and defense. Biol. Psychiatry 79, 165–173.
De Dreu, C.K.W., Baas, M., Nijstad, B.A., 2008. Hedonic tone and activation level in the mood-creativity link: toward a dual pathway to creativity model. J. Pers. Soc. Psychol. 94, 739–756.
De Dreu, C.K.W., Nijstad, B.A., Baas, M., 2011. Behavioral activation links to creativity because of increased cognitive flexibility. Soc. Psychol. Personal. Sci. 2, 72–80.
De Dreu, C.K.W., Nijstad, B.A., Baas, M., Wolsink, I., Roskes, M., 2012. Working memory benefits creative insight, musical improvisation: and original ideation through maintained task-focused attention. Pers. Soc. Psychol. Bull. 38, 656–669.
De Dreu, C.K.W., Baas, M., Roskes, M., Sligte, D.J., Ebstein, R.P., Chew, S.H., Tong, T., Jiang, Y., Mayseless, N., Shamay-Tsoory, S.G., 2014. Oxytonergic circuitry sustains and enables creative cognition in humans. Soc. Cogn. Affect Neurosci. 9, 1159–1165.
De Dreu, C.K.W., Baas, M., Boot, N.C., 2015. Oxytocin enables novelty seeking and creative performance through upregulated approach: evidence and avenues for future research. Wiley Interdiscip. Rev. Cogn. Sci. 6, 409–417.
De Manzano, Ö., Cervenka, S., Karabanov, A., Farde, L., Ullén, F., 2010. Thinking outside a less intact box: thalamic dopamine D2 receptor densities are negatively related to psychometric creativity in healthy individuals. PLoS One 5, e10670.
Depue, R.A., Collins, P.F., 1999. Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation and extraversion. Behav. Brain Sci. 22, 491–569.
Dietrich, A., Kanso, R., 2010. A review of EEG, ERP: and neuroimaging studies of creativity and insight. Psychol. Bull. 136, 822–848.
Dodds, C.M., Müller, U., Clark, L., van Loon, A., Cools, R., Robbins, T.W., 2008. Methylphenidate has differential effects on blood oxygenation level-dependent signal related to cognitive subprocesses of reversal learning. J. Neurosci. 28, 5976–5982.
Dodds, C.M., Clark, L., Dove, A., Regenthal, R., Baumann, F., Bullmore, E., Robbins, TW, Müller, U., 2009. The dopamine D2 receptor antagonist sulpiride modulates striatal BOLD signal during the manipulation of information in working memory. Psychopharmacology 207, 35–45.
Donaldson, Z.R., Young, L.J., 2008. Oxytocin, vasopressin, and the neurogenetics of sociality. Science 322, 900–903.
Dreisbach, G., Goschke, T., 2004. How positive affect modulates cognitive control: reduced perseveration at the cost of increased distractibility. J. Exp. Psychol. Learn. Mem. Cogn. 30, 343–353.
Duncker, K., 1945. On problem solving. Psychol. Monogr. 58 (5).
Durstewitz, D., Seamans, J.K., 2008. The dual-state theory of prefrontal cortex dopamine function with relevance to catechol-o-methyltransferase genotypes and schizophrenia. Biol. Psychiatry 64, 739–749.
Erhard, K., Kessler, F., Neumann, N., Ortheil, H.J., Lotze, M., 2014. Professional training in creative writing is associated with enhanced fronto-striatal activity in a literary text continuation task. Neuroimage 100, 15–23.
Eslinger, P.J., Grattan, L.M., 1993. Frontal lobe and frontal-striatal substrates for different forms of human cognitive flexibility. Neuropsychologia 31, 17–28.
Ettinger, U., Meyhöfer, I., Steffens, M., Wagner, M., Koutsouleris, N., 2014. Genetics, cognition: and neurobiology of schizotypal personality: a review of the overlap with schizophrenia. Front. Psychiatry 5, 1–16.
Farah, M.J., Haimm, C., Sankoorikal, G., Chatterjee, A., 2009. When we enhance cognition with Adderall, do we sacrifice creativity? A preliminary study. Psychopharmacology 202, 541–547.
Faust-Socher, A., Kenett, Y.N., Cohen, O.S., Hassin-Baer, S., Inzelberg, R., 2014. Enhanced creative thinking under dopaminergic therapy in Parkinson disease. Ann. Neurol. 75, 935–942.
Fletcher, P.C., Frith, C.D., 2009. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. Nat. Rev. Neurosci. 10, 48–58.
Folley, B.S., Park, S., 2005. Verbal creativity and schizotypal personality in relation to prefrontal hemispheric laterality: a behavioral and near-infrared optical imaging study. Schizophr. Res. 80, 271–282.
Frank, M.J., Loughry, B., O'Reilly, R.C., 2001. Interactions between frontal cortex and basal ganglia in working memory: a computational model. Cogn. Affect Behav. Neurosci. 1, 137–160.
Frank, M.J., 2005. Dynamic dopamine modulation in the basal ganglia: a neurocomputational account of cognitive deficits in medicated and nonmedicated Parkinsonism. J. Cogn. Neurosci. 17, 51–72.
Furnham, A., Batey, M., Anand, K., Manfield, J., 2008. Personality, hypomania, intelligence and creativity. Pers. Individ. Dif. 44, 1060–1069.
Gardner, H., 1993. Seven creators of the modern era. In: Brockman, J. (Ed.), Creativity. Simon & Schuster, New York, pp. 28–47.
Gilhooly, K.J., Fioratou, E., Anthony, S.H., Wynn, V., 2007. Divergent thinking, strategies and executive involvement in generating novel uses for familiar objects. Br. J. Psychol. 98, 611–625.
Goldman-Rakic, P.S., 1992. Dopamine-mediated mechanisms of the prefrontal cortex. Sem. Neurosci. 4, 149–159.
Gonen-Yaacovi, G., De Souza, L.C., Levy, R., Urbanski, M., Josse, G., Volle, E., 2013. Rostral and caudal prefrontal contribution to creativity: a meta-analysis of functional imaging data. Front. Hum. Neurosci. 7, 1–22.
Gough, H.G., 1979. A creative personality scale for the Adjective Check List. J. Pers. Soc. Psychol. 37, 1398–1405.

Groman, S.M., James, A.S., Seu, E., Tran, S., Clark, T.A., Harpster, S.N., Crawford, M., Burtner, J.L., Feiler, K., Roth, R.H., Elsworth, J.D., London, E.D., Jentsch, J.D., 2014. In the blink of an eye: relating positive-feedback sensitivity to striatal dopamine D2-like receptors through blink rate. J. Neurosci. 34, 14443–14454.
Guilford, J.P., 1950. Creativity. Am. Psychol. 5, 444–454.
Guilford, J.P., 1967. The Nature of Human Intelligence. McGraw-Hill, New York.
Gvirts, H.Z., Mayseless, N., Segev, A., Lewis, D.Y., Feffer, K., Barnea, Y., Bloch, Y., Shamay-Tsoory, S.G., 2016. Novelty-seeking trait predicts the effect of methylphenidate on creativity. J. Psychopharmacol 0269881116667703.
Hase, A., Jung, S.E., Aan het Rot, M., 2015. Behavioral and cognitive effects of tyrosine intake in healthy human adults. Pharmacol. Biochem. Behav. 133, 1–6.
Hennessey, B.A., Amabile, T.M., 2010. Creativity. Annu. Rev. Psychol. 61, 569–598.
Ikemoto, S., 2007. Dopamine reward circuitry: two projection systems from the ventral midbrain to the nucleus accumbens-olfactory tubercle complex. Brain Res. Rev. 56, 27–78.
Inzelberg, R., 2013. The awakening of artistic creativity and Parkinson's disease. Behav. Neurosci. 127, 256–261.
Ionescu, T., 2012. Exploring the nature of cognitive flexibility. New Ideas Psychol. 30, 190–200.
Isen, A.M., Daubman, K.A., Nowicki, G.P., 1987. Positive affect facilitates creative problem solving. J. Pers. Soc. Psychol. 52, 1122–1131.
Jauk, E., Neubauer, A.C., Dunst, B., Fink, A., Benedek, M., 2015. Gray matter correlates of creative potential: a latent variable voxel-based morphometry study. Neuroimage 111, 312–320.
Johnson, S.L., Murray, G., Fredrickson, B., Youngstrom, E.A., Hinshaw, S., Bass, J.M., Deckersbach, T., Schooler, J., Salloum, I., 2012. Creativity and bipolar disorder: touched by fire or burning with questions? Clin. Psychol. Rev. 32, 1–12.
Kane, M.J., Engle, R.W., 2002. The role of prefrontal cortex in working-memory capacity, executive attention: and general fluid intelligence: an individual-differences perspective. Psychon. Bull. Rev. 9, 637–671.
Kaufman, J.C., Beghetto, R.A., 2009. Beyond big and little: the four c model of creativity. Rev. Gen. Psychol. 13, 1–12.
Kaufman, J.C., Waterstreet, M.A., Ailabouni, H.S., Whitcomb, H.J., Roe, A.K., Riggs, M., 2009. Personality and self-perceptions of creativity across domains. Imagin. Cogn. Pers. 29, 193–209.
Kaufman, J.C., 2016. Creativity 101. Springer, New York.
Kehagia, A.A., Murray, G.K., Robbins, T.W., 2010. Learning and cognitive flexibility: frontostriatal function and monoaminergic modulation. Curr. Opin. Neurobiol. 20, 199–204.
Kellendonk, C., Simpson, E.H., Polan, H.J., Malleret, G., Vronskaya, S., Winiger, V., Moore, H., Kandel, E.R., 2006. Transient and selective overexpression of dopamine D2 receptors in the striatum causes persistent abnormalities in prefrontal cortex functioning. Neuron 49, 603–615.
Kim, K.H., 2008. Meta-analyses of the relationship of creative achievement to both IQ and divergent thinking test scores. J. Creat. Behav. 42, 106–130.
Krugel, L.K., Biele, G., Mohr, P.N.C., Li, S.-C., Heekeren, H.R., 2009. Genetic variation in dopaminergic neuromodulation influences the ability to rapidly and flexibly adapt decisions. Proc. Natl. Acad. Sci. U. S. A. 106, 17951–17956.
Kuczenski, R., Segal, D.S., 1997. Effects of methylphenidate on extracellular dopamine, serotonin: and norepinephrine: comparison with amphetamine. J. Neurochem. 68, 2032–2037.
Langley, K., Marshall, L., Van den Bree, M., Thomas, H., Owen, M., O'Donovan, M., Thapar, A., 2004. Association of the dopamine D4 receptor gene 7-repeat allele with neuropsychological test performance of children with ADHD. Am. J. Psychiatry 161, 133–138.
Laruelle, M., Kegeles, L.S., Abi-Dargham, A., 2003. Glutamate, dopamine and schizophrenia. Ann. N. Y. Acad. Sci. 1003, 138–158.
Leber, A.B., Turk-Browne, N.B., Chun, M.M., 2008. Neural predictors of moment-to-moment fluctuations in cognitive flexibility. Proc. Natl. Acad. Sci. U. S. A. 105, 13592–13597.
Lidow, M.S., Goldman-Rakic, P.S., Gallager, D.W., Rakic, P., 1991. Distribution of dopaminergic receptors in the primate cerebral cortex: quantitative autoradiographic analysis using [3H]raclopride, [3H]spiperone and [3H]SCH23390. Neuroscience 40, 657–671.
Lucas, B.J., Nordgren, L.F., 2015. People underestimate the value of persistence for creative performance. J. Pers. Soc. Psychol. 109, 232–243.
MacGregor, J.N., Ormerod, T.C., Chronicle, E.P., 2001. Information processing and insight: a process model of performance on the nine-dot and related problems. J. Exp. Psychol. Learn. Mem. Cogn. 27, 176–201.
Magill, R.A., Waters, W.F., Bray, G.A., Volaufova, J., Smith, S.R., Lieberman, H.R., McNevin, N., Ryan, D.H., 2003. Effects of tyrosine, phentermine, caffeine D-amphetamine, and placebo on cognitive and motor performance deficits during sleep deprivation. Nutr. Neurosci. 6, 237–246.
Malhotra, A.K., Kestler, L.J., Mazzanti, C., Bates, J.A., Goldberg, T., Goldman, D., 2002. A functional polymorphism in the COMT gene and performance on a test of prefrontal cognition. Am. J. Psychiatry 159, 652–654.
Martínez-Arán, A., Vieta, E., Reinares, M., Colom, F., Torrent, C., Sánchez-Moreno, J., Benabarre, A., Goikolea, J.M., Comes, M., Salamero, M., 2004. Cognitive function across manic or hypomanic, depressed, and euthymic states in bipolar disorder. Am. J. Psychiatry 161, 262–270.
Matsumoto, M., Weickert, C.S., Akil, M., Lipska, B.K., Hyde, T.M., Herman, M.M., Kleinman, J.E., Weinberger, D.R., 2003. Catechol O-methyltransferase mRNA expression in human and rat brain: evidence for a role in cortical neuronal function. Neuroscience 116, 127–137.
Mayseless, N., Shamay-Tsoory, S., 2015. Enhancing verbal creativity: modulating creativity by altering the balance between right and left inferior frontal gyrus with tDCS. Neuroscience 291, 167–176.
Mayseless, N., Uzefovsky, F., Shalev, I., Ebstein, R.P., Shamay-Tsoory, S.G., 2013. The association between creativity and 7R polymorphism in the dopamine receptor D4 gene (DRD4). Front. Hum. Neurosci. 7, 1–7.
McNab, F., Klingberg, T., 2008. Prefrontal cortex and basal ganglia control access to working memory. Nat. Neurosci. 11, 103–107.
Mednick, S.A., 1962. The associative basis of the creative process. Psychol. Rev. 69, 220–232.
Mehta, M.A., Manes, F.F., Magnolfi, G., Sahakian, B.J., Robbins, T.W., 2004. Impaired set-shifting and dissociable effects on tests of spatial working memory following the dopamine D2 receptor antagonist sulpiride in human volunteers. Psychopharmacology (Berl.) 176, 331–342.
Metuki, N., Sela, T., Lavidor, M., 2012. Enhancing cognitive control components of insight problems solving by anodal tDCS of the left dorsolateral prefrontal cortex. Brain Stim. 5, 110–115.
Meyer-Lindenberg, A., Kohn, P.D., Kolachana, B., Kippenhan, S., McInerney-Leo, A., Nussbaum, R., Weinberger, D.R., Berman, K.F., 2005. Midbrain dopamine and prefrontal function in humans: interaction and modulation by COMT genotype. Nat. Neurosci. 8, 594–596.
Meyer-Lindenberg, A., Domes, G., Kirsch, P., Heinrichs, M., 2011. Oxytocin and vasopressin in the human brain: social neuropeptides for translational medicine. Nat. Rev. Neurosci. 12, 525–538.
Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202.
Miller, A.I., 2000. Insights of Genius: Imagery and Creativity in Science and Art. MIT Press, Cambridge, MA.
Murphy, M., Runco, M.A., Acar, S., Reiter-Palmon, R., 2013. Reanalysis of genetic data and rethinking dopamine's relationship with creativity. Creat. Res. J. 25, 147–148.
Nijstad, B.A., De Dreu, C.K.W., Rietzschel, E.F., Baas, M., 2010. The dual pathway to creativity model: creative ideation as a function of flexibility and persistence. Eur. Rev. Soc. Psychol. 21, 34–77.
O'Sullivan, N., Szczepanowski, R., El-Deredy, W., Mason, L., Bentall, R.P., 2011. fMRI evidence of a relationship between hypomania and both increased goal-sensitivity and positive outcome-expectancy bias. Neuropsychologia 49, 2825–2835.
Piray, P., Den Ouden, H.E.M., Van der Schaaf, M.E., Toni, I., Cools, R., 2016. Dopaminergic modulation of the functional ventrodorsal architecture of the human striatum. Cereb. Cortex. http://dx.doi.org/10.1093/cercor/bhv243.
Plucker, J.A., Renzulli, J.S., 1999. Psychometric approaches to the study of human creativity. In: Sternberg, R.J. (Ed.), Handbook of Creativity. Cambridge University Press, New York, pp. 35–60.
Polner, B., Nagy, H., Takáts, A., Kéri, S., 2015. Kiss of the muse for the chosen ones: de novo schizotypal traits and lifetime creative achievement are related to changes in divergent thinking during dopaminergic therapy in Parkinson's disease. Psychol. Aesthet. Creat. Arts 9, 328–339.
Ravizza, S.M., Carter, C.S., 2008. Shifting set about task switching: behavioral and neural evidence for distinct forms of cognitive flexibility. Neuropsychologia 46, 2924–2935.
Reuter, M., Roth, S., Holve, K., Hennig, J., 2006. Identification of first candidate genes for creativity: a pilot study. Brain Res. 1069, 190–197.
Richards, R., Kinney, D.K., Lunde, I., Benet, M., Merzel, A.P.C., 1988. Creativity in manic-depressives, cyclothymes, their normal relatives and control subjects. J. Abnorm. Psychol. 97, 281–288.
Richards, R., 2007. Everyday creativity: our hidden potential. In: Richards, R. (Ed.), Everyday Creativity and New Views of Human Nature. American Psychological Association, Washington, DC, pp. 25–54.
Rietzschel, E.F., De Dreu, C.K.W., Nijstad, B.A., 2007. Personal need for structure and creative performance: the moderating influence of fear of invalidity. Pers. Soc. Psychol. Bull. 33, 855–866.
Robbins, T.W., Roberts, A.C., 2007. Differential regulation of fronto-executive function by the monoamines and acetylcholine. Cereb. Cortex 17, i151–i160.
Roskes, M., De Dreu, C.K.W., Nijstad, B.A., 2012. Necessity is the mother of invention: avoidance motivation stimulates creativity through cognitive effort. J. Pers. Soc. Psychol. 103, 242–256.
Runco, M.A., Acar, S., 2012. Divergent thinking as an indicator of creative potential. Creat. Res. J. 24 (1), 66–75.
Runco, M.A., Jaeger, G.J., 2012. The standard definition of creativity. Creat. Res. J. 24, 92–96.
Runco, M.A., Noble, E.P., Reiter-Palmon, R., Acar, S., Ritchie, T., Yurkovich, J.M., 2011. The genetic basis of creativity and ideational fluency. Creat. Res. J. 23, 376–380.
Runco, M.A., 2004. Creativity. Annu. Rev. Psychol. 55, 657–687.
Santosa, C.M., Strong, C.M., Nowakowska, C., Wang, P.W., Rennicke, C.M., Ketter, T.A., 2007. Enhanced creativity in bipolar disorder patients: a controlled study. J. Affect. Disord. 100, 31–39.
Scatton, B., Javoy-Agid, F., Rouquier, L., Dubois, B., Agid, Y., 1983. Reduction of cortical dopamine, noradrenaline: serotonin and their metabolites in Parkinson's disease. Brain Res. 275, 321–328.
Schultz, W., 2002. Getting formal with dopamine and reward. Neuron 36, 241–263.
Seamans, J.K., Yang, C.R., 2004. The principal features and mechanisms of dopamine modulation in the prefrontal cortex. Prog. Neurobiol. 74, 1–58.
Shamay-Tsoory, S.G., Adler, N., Aharon-Peretz, J., Perry, D., Mayseless, N., 2011. The origins of originality: the neural bases of creative thinking and originality. Neuropsychologia 49, 178–185.
Silvia, P.J., Winterstein, B.P., Willse, J.T., Barona, C.M., Cram, J.T., Hess, K.I., Martinez, J.L., Richard, C.A., 2008. Assessing creativity with divergent thinking tasks: exploring the reliability and validity of new subjective scoring methods. Psychol. Aesthet. Creat Arts 2, 68–85.
Simonton, D.K., 2003. Scientific creativity as constrained stochastic behavior: the

integration of product, person and process perspectives. Psychol. Bull. 129, 475–494.
Simonton, D.K., 2007. Creativity: specialized expertise or general cog- nitive processes? In: Roberts, M.J. (Ed.), Integrating the Mind: Domain General Versus Domain Specific Processes in Higher Cognition. Psychology Press, Hove, England, pp. 351–367.
Srivastava, S., Childers, M.E., Baek, J.H., Strong, C.M., Hill, S.J., Warsett, K.S., ... Ketter, T.A., 2010. Toward interaction of affective and cognitive contributors to creativity in bipolar disorders: a controlled study. J. Affect. Disord. 125, 27–34.
Sternberg, R.J., 1999. Handbook of Creativity. Cambridge University Press, New York.
Strakowski, S.M., Delbello, M.P., Adler, C.M., 2005. The functional neuroanatomy of bipolar disorder: a review of neuroimaging findings. Mol. Psychiatry 10, 105–116.
Swainson, R., Rogers, R.D., Sahakian, B.J., Summers, B.A., Polkey, C.E., Robbins, T.W., 2000. Probabilistic learning and reversal deficits in patients with Parkinson's disease or frontal or temporal lobe lesions: possible adverse effects of dopaminergic medication. Neuropsychologia 38, 596–612.
Swerdlow, N.R., Stephany, N., Wasserman, L.C., Talledo, J., Sharp, R., Auerbach, P.P., 2003. Dopamine agonists disrupt visual latent inhibition in normal males using a within-subject paradigm. Psychopharmacology (Berl.) 169, 314–320.
Takeuchi, H., Taki, Y., Sassa, Y., Hashizume, H., Sekiguchi, A., Fukushima, A., Kawashima, R., 2010a. Regional gray matter volume of dopaminergic system associate with creativity: evidence from voxel-based morphometry. Neuroimage 51, 578–585.
Takeuchi, H., Taki, Y., Sassa, Y., Hashizume, H., Sekiguchi, A., Fukushima, A., Kawashima, R., 2010b. White matter structures associated with creativity: evidence from diffusion tensor imaging. Neuroimage 51, 11–18.
Van der Schaaf, M.E., Van Schouwenburg, M.R., Geurts, D.E.M., Schellekens, A.F.A., Buitelaar, J.K., Verkes, R.J., Cools, R., 2014. Establishing the dopamine dependency of human striatal signals during reward and punishment reversal learning. Cereb. Cortex 24, 633–642.
Vijayraghavan, S., Wang, M., Birnbaum, S.G., Williams, G.V., Arnsten, A.F.T., 2007. Inverted-U dopamine D1 receptor actions on prefrontal neurons engaged in working memory. Nat. Neurosci. 10, 376–384.
Wallace, D.L., Vytlacil, J.J., Nomura, E.M., Gibbs, S.E.B., D'Esposito, M., 2011. The dopamine agonist bromocriptine differentially affects fronto-striatal functional connectivity during working memory. Front. Hum. Neurosci. 5, 32.
Ward, T.B., Smith, S.M., Finke, R.A., 1999. Creative cognition. In: Sternberg, R.J. (Ed.), Handbook of Creativity. Cambridge University Press, New York, pp. 189–212.
Zabelina, D.L., Robinson, M.D., 2010. Creativity as flexible cognitive control. Psychol. Aesthet. Creat. Arts 4, 136–143.
Zabelina, D., Saporta, A., Beeman, M., 2015. Flexible or leaky attention in creative people?: Distinct patterns of attention for different types of creative thinking. Mem. Cogn. 44, 488–498.
Zabelina, D.L., Colzato, L., Beeman, M., Hommel, B., 2016. Dopamine and the creative mind: individual differences in creativity are predicted by interactions between dopamine genes DAT and COMT. PLoS One 11, e0146768.
Zhang, S., Zhang, M., Zhang, J., 2014a. An exploratory study on DRD2 and creative potential. Creat. Res. J. 26, 115–123.
Zhang, S., Zhang, M., Zhang, J., 2014b. Association of COMT and COMT-DRD2 interaction with creative potential. Front. Hum. Neurosci. 8, 216.