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Annual Research Review: Neural contributions to risk-taking in adolescence – developmental changes and individual differences

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Background: Risk-taking, which involves voluntary choices for behaviors where outcomes remain uncertain, undergoes considerable developmental changes during childhood, adolescence, and early adulthood. In addition, risk-taking is thought to be a key element of many externalizing disorders, such as ADHD, delinquency, conduct disorder, and substance abuse. In this review, we will discuss the potential adaptive and nonadaptive properties of risk-taking in childhood and adolescence. **Findings:** We propose that the changes in brain architecture and function are a crucial element underlying these developmental trajectories. We first identify how subcortical and cortical interactions are important for understanding risk-taking behavior in adults. Next, we show how developmental changes in this network underlie changes in risk-taking behavior. Finally, we explore how these differences can be important for understanding externalizing behavioral disorders in childhood and adolescence. **Conclusions:** We conclude that longitudinal studies are of crucial importance for understanding these developmental trajectories, and many of these studies are currently underway. **Keywords:** Risk-taking behavior; adolescence; developmental changes; individual differences; externalizing disorders; brain connectivity; ventral striatum.

Introduction

Many of the decisions we make in daily life involve an element risk. For example, having another coffee in a café may result in missing the train for a next appointment, or buying groceries at the new supermarket across the street may result in missing your favorite product. That is, in a risky choice the decision outcome carries a degree of uncertainty. A more formal definition of risk-taking – as typically used by economists – is choosing the option with the highest outcome variability (Figner & Weber, 2011), indicating that a risky choice may lead to greater benefits, but may also lead to larger negative outcomes at the expense of surety. In many examples the negative consequences of excessive risk-taking seem most salient, as is evident for car accidents, binge drinking, and gambling (Krmpotich et al., 2015). Contrary to common beliefs, however, risk-taking encompasses not only negative behavior; in some situations – or in some phases in life – it may be highly adaptive to take risks (Crone & Dahl, 2012). For example, risk-taking can lead to social benefits, such as forming new relationships (Willoughby, Good, Adachi, Hamza, & Tavernier, 2014) or maximizing financial outcomes (Peper, Koolschijn, & Crone, 2013).

Adolescence has often been described as a period of increased risk-taking (Steinberg et al., 2008). Epidemiological reports have observed an increase in risk-taking behavior in adolescence, such as for traffic accidents, crime rate, and substance abuse (Centers for Disease Control and Prevention Youth Risk Behavior Surveillance Survey – United States, 2011; Substance Abuse and Mental Health Services Administration Results from the 2010 National Survey on Drug Use and Health: Mental Health Findings NSDUH Series H-42, HHS Publication No. (SMA) 11–4667; see also Willoughby et al., 2014). There is a long debate in the literature about whether risk-taking is observed in *all* adolescents and as such is a normative developmental hallmark of adolescence, or is present in only a *subset* of adolescents who have already been susceptible to problem behavior in childhood (Bjork & Pardini, 2015). This debate dates back to theories in the early 20th century when adolescence was described as a period of storm and stress, characterized by conflict with parents, emotional outbursts, and risk-taking behavior (Hall, 1916). Personality theorists argued that risk-taking outbursts in adolescence were necessary for developing into healthy adults, and that the absence of this period of risk-taking would result in problem behavior later in life (Freud, 1969). In the last decades these views on adolescence have been more nuanced by arguing that not all adolescents take risks, and that risk-taking is not a necessity for healthy development (Arnett, 1999). The individual differences in risk-taking in adolescence remain an important question and there is currently no consensus on whether increased risk-taking, be it

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subtle or more extreme, is observed for all or some adolescents.

Theories of risk-taking in adolescence have been complicated even more by inconsistent findings in experimental research using laboratory tasks to measure risk-taking. Whereas some studies report monotonic decreases in risk-taking from childhood to adulthood (e.g., Crone, Bullens, van der Plas, Kijkuit, & Zelazo, 2008; Paulsen, Platt, Huettel, & Brannon, 2011; Van Duijvenvoorde, Jansen, Bredman, & Huizenga, 2012), other studies report adolescent-specific peaks in risk-taking (Burnett, Bault, Coricelli, & Blakemore, 2010; Figner, Mackinlay, Wilkening, & Weber, 2009), or no differences at all between children, adolescents, and adults (Van Leijenhorst, Westenberg, & Crone, 2008). A recent meta-analysis highlighted that adolescents and children take more risks than adults, but this depends partly on task demands (Defoe, Dubas, Figner, & van Aken, 2015). In part because of these discrepancies in findings, there is an emerging need to have more precise measurements of the processes involved in risk-taking and how these change in adolescence. Recent findings from the field of neuroscience have provided important new insight into how changes in neuroanatomic and neural activity through adolescence contribute to a potential rise in risk-taking behavior. In this review, we describe how a specific network in the brain, which encompasses the ventral striatum and prefrontal cortex (PFC), changes anatomically and functionally across the transition from childhood to puberty and from puberty into late adolescence and adulthood. Changes in this network are consistently related to risk-taking behavior (McClure, Laibson, Loewenstein, & Cohen, 2004; Peper, Mandl et al., 2013) and therefore we propose that a better understanding of this network may lead to better understanding of the dynamics of risk-taking in adolescence. There will be a specific focus on the role of pubertal hormones potentially driving parts of these changes (Peper & Dahl, 2013).

First, we will define current theories on risk-taking in adolescence. Second, we provide evidence that the ventral striatum and PFC are critically involved in risk-taking behavior. Third, we describe the structural changes that take place in this network and how this relates to individual differences in risk-taking. Fourth, we summarize the functional MRI studies that have focused on risk-taking in adolescence. Fifth, we describe functional connectivity studies in relation to risk-taking and argue that strengthening of the ventral striatum-PFC network is a central factor in explaining individual differences and developmental changes. Finally, we will discuss the implications of these changes for understanding individual trajectories in childhood disorders, along with the broader conclusions arising from the review.

Risk-taking in adolescence

Theories of risk-taking in adolescence have emerged from two lines. Traditionally, risk-taking has been interpreted as a purely cognitively driven process, and these theories postulated that risk-taking in adolescence was driven by the slowly developing cognitive control capacities of adolescents (Casey, 2015; Duckworth & Steinberg, 2015). There is strong evidence that the ability to use cognitive control, also referred to as executive functions, changes considerably during childhood and early adolescence, and reaches adult levels around mid adolescence (Crone, 2009; Luna, Padmanabhan, & O'Hearn, 2010). For example, children have more difficulties than adults in keeping information in working memory (Van Leijenhorst, Crone, & Van der Molen, 2007), controlling impulses (Durston et al., 2006), and switching between tasks (Morton, Bosma, & Ansari, 2009). These components of executive control follow different developmental trajectories, with response inhibition maturing in late childhood, task switching around early puberty, and working memory in midadolescence (Huizinga, Dolan, & van der Molen, 2006). Despite these consistent findings, a difficulty with this theory is that mid to late adolescents generally perform well on executive function tasks, whereas they are known to show highest levels of risk-taking (Steinberg, 2011). In addition, studies testing the understanding of risk and probability show that already young children have a clear understanding of probabilities (Huizenga, Crone, & Jansen, 2007; Jansen, van Duijvenvoorde, & Huizenga, 2012; Schlottmann & Anderson, 1994).

In the last two decades, researchers have increasingly acknowledged the important role that affective processes may play in risk-taking behavior (Casey, 2015; Crone & Dahl, 2012; Duckworth & Steinberg, 2015). For example, adolescents are particularly susceptible to risk-taking relative to adults if in a context where risks are unpredictable (Tymula et al., 2012), when rewards are encountered immediately (van Duijvenvoorde, Jansen, Visser, & Huizenga, 2010; Figner et al., 2009), when counterfactual emotions such as regret for not choosing the higher alternative are involved (Burnett et al., 2010), or when they are in the presence of peers (Gardner & Steinberg, 2005; Peake, Dishion, Stormshak, Moore, & Pfeifer, 2013). Recent frameworks point out that understanding risk-taking in adolescence involves a conceptualization of the interaction between the cognitive and affective processes involved (Duckworth & Steinberg, 2015). These frameworks are well grounded in neurodevelopmental models, often referred to as dual processing models. These models propose that under emotionally arousing situations adolescents may be more prone to be influenced by affective states compared to children and adults, whereas under emotionally calm situations they are more prone to make cognitively driven choices

(Somerville, Jones, & Casey, 2010; Steinberg et al., 2008; but see Pfeifer & Allen, 2012). Recent extensions of the neurodevelopmental dual processing models are focusing on characterizing developmental changes in neural circuits (Casey, 2015), the flexible recruitment of cognitive control depending on motivational states (Crone & Dahl, 2012), and the importance of decision context and (social) opportunity for risk in different phases of life (Defoe et al., 2015; Willoughby et al., 2014).

Biological perspective on risk-taking

Studies that have focused on the representation of reward values in the brain have typically focused on the ventral striatum and the ventromedial prefrontal cortex (VMPFC). The striatum is roughly divided into a dorsal part, which is related to a control network, involved in action selection, maintaining future goals, and inhibiting prepotent responses, and a ventral part, which is related to a valuation network involved in decision making, learning, and motivated behavior (van den Bos, Rodriguez, Schweitzer, & McClure, 2014). The VMPFC, a region located in the PFC, comprises part of the medial orbital frontal cortex and portions of the medial PFC. As part of a cortical-basal ganglia circuit, the connectivity of the VMPFC positions it well to integrate and represent the value that is expected from choice (O'Doherty, 2011). That is, the VMPFC is heavily interconnected with the ventral striatum, a structure important for reward processing and learning (Haber & Knutson, 2010). For example, an analysis of brain regions involved in risk-taking and reward processing in the neurosynth database (an online meta-analysis data base) shows that the contributions of the striatum and VMPFC to risk-taking and reward processing are robustly documented (Figure 1). As such understanding the developmental changes in striatum-VMPFC structure, function, and connectivity is highly relevant for understanding the developmental changes and individual differences in risk-taking.

The neuroscience of risk-taking has initially been informed by neuropsychological studies involving patients with damage to specific parts of the PFC. The famous case study of Phineas Gage describes the case of a railroad worker who was hit by an iron rod that damaged a large part of the VMPFC. While most of his cognitive functions remained intact, he displayed personality differences, which were associated with problems with emotional temper, anger outbursts, and impulsivity (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). As then, neuropsychological studies have consistently shown that patients with damage to the VMPFC are impaired in risky decision making.

A classic task to measure risk-taking in VMPFC patients is the Iowa Gambling Task, which measures reward sensitivity and future orientation (Bechara, Damasio, Tranel, & Damasio, 2005). The task involves four decks of cards where participants are free to sample from all decks to maximize their profit. Two cards are associated with high rewards on each trial and two cards are associated with low rewards on each trial, but all cards also involve unpredictable losses. Unbeknownst to the participants, the cards with the highest rewards are also associated with the highest losses, and are therefore disadvantageous in the long run. Healthy individuals, as well as VMPFC patients, first sample from all decks and show a quick preference for the high immediate reward decks. However, healthy individuals learn over trials to avoid the high immediate reward decks, because they experience that these decks are disadvantageous in the long run. VMPFC patients, in contrast, fail to learn to switch decks, and continue sampling from the high reward–high loss decks (Bechara, Tranel, & Damasio, 2000). These findings were interpreted to suggest that the VMPFC is a crucial region for future orientation, and that damage to this area of the brain results in risk-taking, especially when contingencies are uncertain. A modification of the task showed that patients with damage to VMPFC have specific difficulties with reversal learning, suggesting that they have difficulty with switching from what was first seen as a good habit (Fellows & Farah, 2005). Importantly, these deficits in value-based decision making were observed in the absence of working memory deficits (Bechara, Damasio, Tranel, & Anderson, 1998). Intriguingly, the developmental studies have shown that performance of adolescents on the Iowa Gambling Task mirrors the performance of VMPFC patients. That is to say, adolescents also show a preference for decks with immediate reward even though this results in losses in the long run (Crone & van der Molen, 2004; Smith, Xiao, & Bechara, 2012). Note, however, that there are individual differences in decision strategy on the Iowa Gambling Task, and some children and adolescents may be very capable of avoiding frequent losses (van Duijvenvoorde et al., 2010; Huizenga et al., 2007).

Even though these neuropsychological patient studies are important for understanding the role of VMPFC in risk-taking, these studies could not separate how the brain responds to the many different processes that are involved when individuals take risks. Methods to examine these processes in more detail are – for example – relating risk-taking to connectivity paths in the brain through white matter connections, through functional connectivity, or by relating risk-taking to neural activity during task performance.

One way to study striatal and PFC communication in relation to risk-taking has been by use of anatomical connectivity. This anatomical connectivity is established through white matter bundles consisting of (myelinated) axons. Using diffusion tensor imaging, white matter connections and its microstructural properties can – indirectly – be studied (Jones,

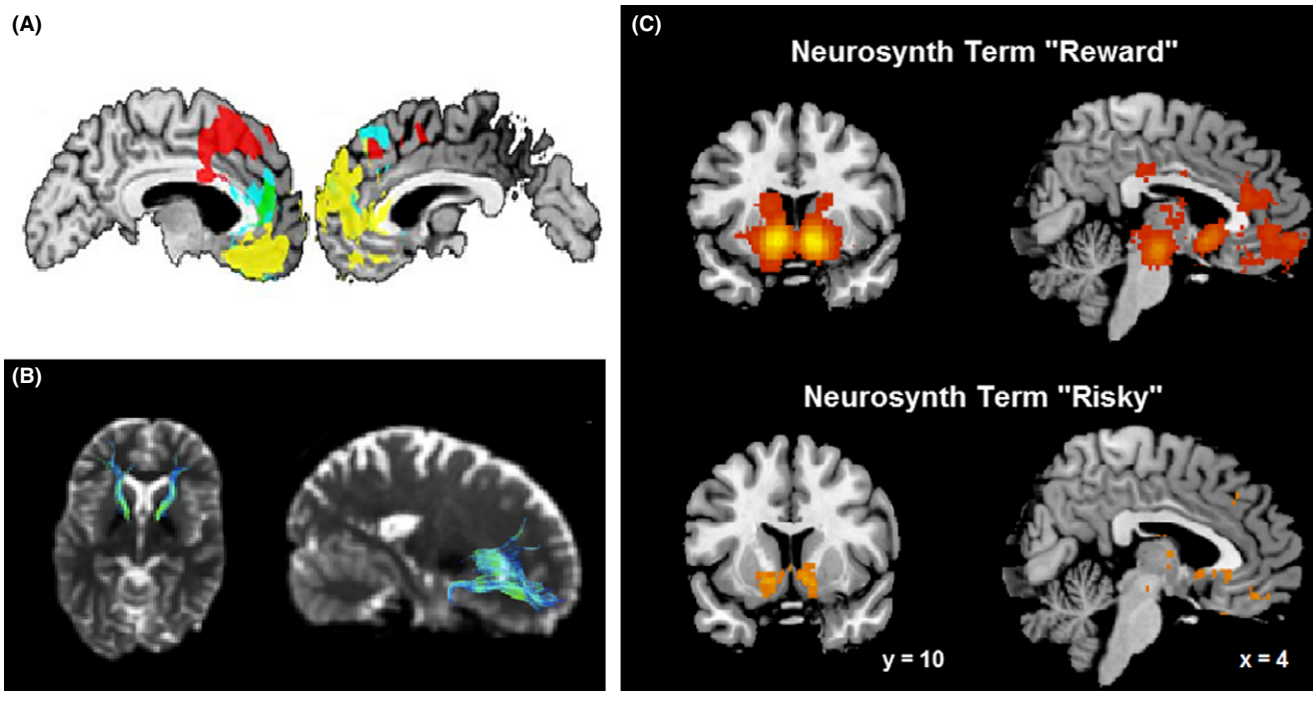

Figure 1 Different methods point toward a crucial role of striatum-ventromedial prefrontal cortex implicated in risk-taking. (A) Lesion mapping method, showing in yellow the region that corresponds with deficits on Iowa Gambling Task performance (see text for explanation; Glascher et al., 2012), (B) white matter connectivity path that corresponds to regulation of impulsivity (Peper, Koolschijn et al., 2013, with permission), and (C) Neurosynth meta-analysis of fMRI activations associated with the search term 'risky' or 'reward'. Results from neurosynth using the search term 'risky' (reverse inference, FDR-corrected 0.01) based on 55 studies. Results from neurosynth using the search term 'reward' (reverse inference, FDR-corrected 0.01) based on 560 studies

2008). This technique is particularly informative when testing the dual processing hypothesis, which states that increased risk-taking is mediated by less control of the PFC over striatal brain areas. Specifically, this hypothesis suggests that there is an imbalance between both brain systems, suggesting abnormal communication through white matter pathways. Results from studies examining these anatomical tracts in relation to the developmental changes in risk-taking and reward processing will be further discussed in Developmental changes and individual differences in structural connectivity and brain morphology related to risk-taking.

In addition, over the past two decades, a series of fMRI studies have used carefully controlled task manipulations to separate neural activations of processes such as risk, expected value, reward anticipation and reward valuation that constitute components of risky choice. These studies have consistently reported a role for both the striatum and the PFC when making risky choices and when processing rewarding outcomes (see Figure 1). The developmental changes in task-related neural activation and the decomposition of risky choice-related processes are further discussed in Developmental changes and individual differences in functional activity related to risk-taking. In Developmental changes and individual differences in functional connectivity related to risk-taking, we will discuss age-related change in intrinsic functional connectivity measures, a technique based on resting-state connectivity analyses, in which we focus on findings relevant to risk-taking.

A biological factor to consider that may trigger some of the changes in risk-taking in adolescence is the influence of hormonal changes such as testosterone. Testosterone is mainly produced by the testes in males, but also by the ovaries and adrenal glands in females (together producing about 1/10 of the amount compared to males). In adults, various studies on endogenous testosterone as well as testosterone administration have reported that higher levels of testosterone are related to more economic and noneconomic risk-taking (Apicella, Carre, & Dreber, 2015). For example, testosterone levels are related to financial risk-taking (Coates & Herbert, 2008), risk aversion at intermediate levels but not at low or high levels (Stanton et al., 2011), and may have a more general role in social status-seeking behavior (Boksem et al., 2013). In addition, testosterone levels influence neural activity in the ventral striatum in risk-taking paradigms in adults (Hermans et al., 2010). Given the massive changes in hormone levels during adolescent development, it is likely that these changes have an important shaping role in brain organization (i.e., have irreversible effects on brain morphology) and PFC-striatum connectivity, which may have implications for risk-taking behavior (Peper & Dahl, 2013). These studies are described in more detail below.

Developmental changes and individual differences in structural connectivity and brain morphology related to risk-taking

Alterations in risk-taking during adolescence have been attributed to changes in brain morphology. In general, albeit time – and region – specific, gray and white matter undergo considerable changes from childhood into adulthood: An overall reduction in gray matter volume and cortical thickness (i.e., neurons and its dendrites, but also intracortical myelin and glial cells (Mills & Tamnes, 2014) takes place (Raznahan et al., 2011; Wierenga, Langen, Oranje, & Durston, 2014) as well as an increase in white matter volume (i.e., myelinated axons; Schmithorst & Yuan, 2010) and changes in organization of white matter connections (Lebel & Beaulieu, 2011; Simmonds, Hallquist, Asato, & Luna, 2014). These normative changes in brain structure are thought to reflect fine-tuning and specialization of neuronal networks and underlie refinement of motor functioning, higher order cognition and cognitive control (Bava et al., 2010).

The striatum-PFC white matter tracts have previously been related to impulsivity control in the delay of gratification task in adults (van den Bos et al., 2014; Peper, Mandl et al., 2013). This task captures an individual's sensitivity to choose an immediate lower reward over a larger delayed reward. Typically, the task involves multiple choices between options such as '10 dollars today or 20 dollars next week'. By varying the amount and time delay it is possible to estimate an individual's tendency to discount delayed rewards. Individuals who are more inclined to select the immediate reward are characterized as more impulsive, whereas individuals who select the delayed reward are characterized as less impulsive. Studies that tested for the relation between delay discounting impulsivity and PFC-striatum connectivity in healthy adults reported that higher integrity of fronto-striatal white matter tracts is related to better behavioral control (less impulsivity) in the delay of gratification paradigm (van den Bos et al., 2014; Peper, Mandl et al., 2013). We recently found that the integrity of these fronto-striatal white matter bundles substantially increases across adolescence (Achterberg et al., in press), and that this developmental increase in white matter is accompanied by an improvement in impulse control over time. That is to say, the relation between the age-related increase in the ability to delay gratification was partly mediated by the strength of the connections between the striatum and the PFC (see also Van den Bos, Rodriguez, Schweitzer, & McClure, 2015).

A second way in which we have previously related brain structure change to risk-taking is by relating gray matter volume in the orbitofrontal cortex and risk-taking on the Balloon Analogue Risk-taking (BART) task (Figure 2). The BART is a computerized task in which participants can inflate a balloon by pressing a button and each button press relates to an increase in money. However, if the balloon explodes, all the money is lost. Therefore, it is key to maximize the reward without pumping too long to avoid explosion. The task is well known for its relations with daily life risk-taking in adolescence (Lejuez et al., 2002). BART risk-taking increases between childhood and adolescence, shows a peak in midadolescence, and a decrease in risk-taking in early adulthood (Braams, van Duijvenvoorde, Peper, & Crone, 2015). The age-related change in risk-taking was mediated by gray matter morphology of the orbitofrontal cortex (Peper, Koolschijn et al., 2013), such that a relatively fast development of the medial orbitofrontal cortex decreased risk-taking in girls, but increased risk-taking in boys. These data suggest that the orbitofrontal cortex accounts for some of the variance in risk-taking development, but does so in sexually dimorphic way.

How do these systems get triggered to change in adolescence? Animal studies have shown that testosterone is able to directly affect structural brain development, for instance, by influencing axonal diameter (Pesaresi et al., 2015), myelination (Melcangi et al., 2003), and number of synapses (Cooke, Breedlove, & Jordan, 2003). Adolescence is hallmarked by an increased production of sex steroid hormones (Grumbach & Styne, 1998). Therefore, one possible hypothesis is that adolescent increases in testosterone relate to increased risk-taking and decreased behavioral control through an effect on gray matter and white matter connections (Peper, Pol, Crone, & van Honk, 2011).

Indeed, human neuroimaging studies across several modalities are now being published that demonstrate an association between increased adolescent testosterone and decreased white matter integrity within the PFC and between the PFC and subcortical areas (Herting, Maxwell, Irvine, & Nagel, 2012; Menzies, Goddings, Whitaker, Blakemore, & Viner, 2015; Peper, de Reus, van den Heuvel, & Schutter, 2015), as well as increased gray matter volume in the orbitofrontal cortex (Peper, Koolschijn et al., 2013). With respect to behavioral risk-taking tendencies, testosterone effects on reduced brain connectivity are related to increased alcohol use (Peters, Jolles, Van Duijvenvoorde, Crone, & Peper, 2015), increased aggression (Peper et al., 2015), and increased laboratory risk-taking (Peper, Koolschijn et al., 2013). Even though effects could be demonstrated in both sexes, the effects were most pronounced in boys. These studies provide evidence that adolescent testosterone changes mediate the relation between brain structure and risk-taking behavior.

The exact neuronal mechanisms, however, of how testosterone affects risk-taking through PFC morphology and connectivity remain unclear. Possibly, testosterone directly influences PFC morphology, as

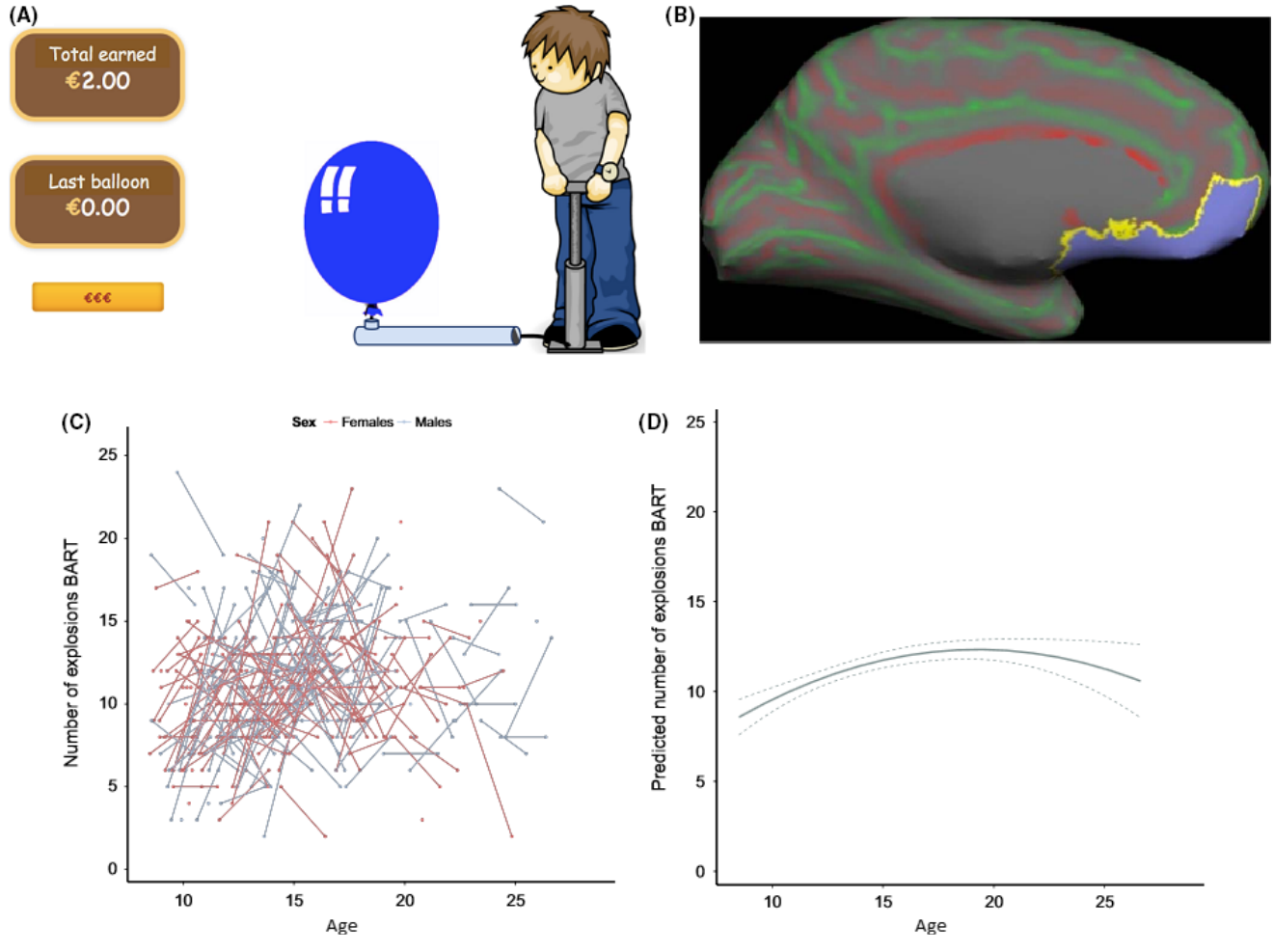

Figure 2 Example of a Balloon Analogue Risk-taking (BART) task trial (adapted from Lejuez et al., 2002). (A) By mouse clicking on the pump, the balloon was inflated, and 0.05€ was gained for each pump. The total amount of collected money on each trial was stored in a temporary bank (not displayed on the screen). Participants could decide to stop inflating the balloon at any time and collect their money by clicking the €€€ button. Then, their money was transferred to the permanent bank (accompanied by a slot-machine sound), and the amount was displayed on the screen. When the balloon exploded, the computer played a 'pop' sound, and the temporarily saved money on that trial was lost. (B) The medial orbitofrontal cortex (OFC): we found that a smaller medial OFC volume in boys and larger OFC surface area in girls related to more risk-taking. A mediation analysis indicated that OFC morphology partly mediates the association between testosterone level and risk-taking, independent of age (from Peper, Koolschijn et al., 2013; with permission). (C, D) Longitudinal graphic representation of age at both time points and total number of explosions in the BART on both time points. Individual subjects are represented by individual lines (C). Subjects measured only once are represented by points. Predicted values for total numbers of explosions in the BART based on the optimal fitting model (D). Dotted lines represent 95% confidence interval (from Braams et al., 2015, with permission)

receptors for androgens such as testosterone are found in the PFC (Finley & Kritzer, 1999). Alternatively, indirect effects of testosterone on neurotransmitter systems within the PFC such as the serotonin system (Handa, Hejna, & Lorens, 1997) or the dopamine system (Aubele & Kritzer, 2012) could also account for individual differences in reward-related risk-taking behavior. Moreover, a recent study suggests that testosterone is positively related to risk-taking but only among individuals low in cortisol (Mehta, Welker, Zilioli, & Carre, 2015), underscoring a 'dual-hormone hypothesis' pertaining to risk-taking tendencies (Dabbs, Jurkovic, & Frady, 1991).

In sum, structural connectivity and sex hormones are important biological factors in explaining individual differences in risk-taking tendencies. Furthermore, the developmental changes in these factors relate to changes in risk-taking behavior across adolescence.

An important factor in all these studies is the question of whether adolescent-specific changes in risk-taking are excessive (i.e., reckless sensation-seeking behavior), or merely a form of adaptive exploratory behavior. It has been found that relatively high secretion of testosterone in adolescent girls is related to increased monetary gains on a risk-taking task (Peper, Koolschijn et al., 2013) and to less self-reported anxiety (Peper et al., 2015). It might therefore be speculated that – at least in girls – relatively high testosterone relates to adaptive exploratory behavior. That is to say, adolescence is associated with quickly changing environmental demands that require adaptive skills and high flex-

ibility. Risk-taking is associated with explorative behavior, linked to obtaining personal autonomy and identity based on experience (Romer, 2010). As such, adolescents who engage in high levels of –adaptive- risk-taking should also be more likely to learn from the negative consequences of their behavior.

Two recent studies support this notion, by suggesting that testosterone levels modulate risky behavior in an adaptive way (Apicella et al., 2015). Moreover, risk-taking was higher in late adolescents with higher integrity of white matter connections (Kwon, Vorobyev, Moe, Parkkola, & Hamalainen, 2014). The authors also argued that more risk-taking relates to 'more mature' white matter connections and could be interpreted as normal development. These findings suggest a broader interpretation of risk-taking in adolescence, which can both be adaptive or maladaptive.

Developmental changes and individual differences in functional activity related to risk-taking

A wealth of studies has examined risk-taking in relation to neural activity during task performance. To understand how the striatum and PFC are involved in developmental changes in risk-taking, studies have used fMRI to test task-related activity in these brain regions. In these studies, processes involved in risk-taking have been studied in more detail, for example, by differentiating between processes in different phases of risk-taking, such as reward anticipation, reward valuation during choice, and reward-related processing during outcomes. These findings have been informative with respect to how subregions in the striatum-PFC network are sensitive to different aspects of risk-taking.

Reward anticipation

To gain insight into reward anticipation, studies used simple paradigms with control conditions where choices had no consequence on outcome (e.g., press the button when a stimulus appears). These studies reported that the ventral striatum is active when anticipating and when receiving rewards, relative to no rewards or losses (Van Leijenhorst, Zanolie et al., 2010). Reward anticipation in the ventral striatum was heightened in 13–17-year-old adolescents, compared to 8–11-year-old children and 18–29-year-old adults in response to the same level of reward (Galvan et al., 2006; Van Leijenhorst, Zanolie et al., 2010). Heightened reward anticipation activity in the striatum was also observed in 12–16-year-old adolescents, compared to adults, in a study that used a choice and no-choice gambling task, but this difference was only found in the no-choice condition (Jarcho et al., 2012). Thus, in terms of reward sensitivity there is robust evidence that reward sensitivity shows adolescent-specific heightened activity, which may indicate that adolescents are more driven by rewards. It should be noted that this heightened response in the striatum of adolescents has not been found in more complex reward-anticipation paradigms, in which the delivery of reward remains uncertain or dependent on performance (Bjork, Smith, Chen, & Hommer, 2010; Hoogendam, Kahn, Hillegers, van Buuren, & Vink, 2013). This may indicate that, when adolescents anticipate predictable rewards, they show more activation in the ventral striatum than children or adults, but that this is not the case when adolescents anticipate on unpredictable rewards. Future studies need to unravel the specific role of predictability of rewards in targeted task paradigms.

Reward valuation during choice

Next, studies used risky choice paradigms to examine not only reward anticipation, but also the neural activity that is involved when *taking* voluntary risks. When participants are faced with a risky decision, they often need to decide between a certain chance of getting a small reward (safe choice), and an uncertain chance of getting a high reward (risky choice). A large neuroimaging study tested how children, adolescents and adults make choices when faced with safe versus risky choices, and the neural activity associated with each choice. It was found that safe choices were associated with activation in the dorsolateral PFC, whereas risky choices were associated with activation in the VMPFC (Van Leijenhorst, Moor et al., 2010). In addition, a lower part of the VMPFC/subgenual anterior cingulate cortex (ACC; the subcallosal cortex), a region previously implicated in affective processing, was more active in 12–17-year-old adolescents compared to 8–10-year-old children and 18–25-year-old adults when taking high reward risks. These findings suggest again a unique affective coding of rewards in adolescence in a region that lies at the intersection of the VMPFC and the ventral striatum. A study that used a comparable design found that adults showed more activation in the dorsal ACC and ventral lateral PFC than adolescents when taking risks (Eshel, Nelson, Blair, Pine, & Ernst, 2007). Together, these findings suggest that adults rely more on PFC regions that are important for deliberative processing when making risky choices, whereas adolescents show stronger recruitment of VMPFC regions that are important for affective processing.

From these studies it is, however, not yet clear which component of choice drives these effects. That is, greater risks are typically associated with greater rewards, and thus a risky choice may be driven by insensitivity to risk or a greater sensitivity to reward. One way to disentangle different influences in valuation is to decompose risky choice using a formal

framework in combination with a task that independently varies risk and reward. A recent study followed a risk-return model to specify the influence of changing risks (outcome variability) and changing returns (expected value) on the choices of children (8–12-year-olds), adolescents (16–19-year-olds), and adults (25–35-year-olds). It was observed that return-sensitivity was related to increased activation in the VMPFC, which increased monotonically across age groups (but see Barkley-Levenson and Galvan (2014) for an adolescent-specific increase specifically in ventral striatum activation during return-valuation). On the other hand, risk sensitivity related to activation in the anterior insula and dorsal medial PFC, which peaked in the adolescent age group (van Duijvenvoorde, Huizenga et al., 2015). These results confirm the role of the VMPFC in valuation during choice in developmental populations, although adolescents showed a distinctive neural response to risk relative to children and adults. These findings confirm the benefits of using more advanced modeling approaches and also show adolescent-specific change in a wider affective network than PFC-striatum connectivity. Future studies should unravel how PFC-striatum responses interact with insula activity during risk-taking.

Processing of outcomes

After taking risks, individuals typically receive a reward or loss following their choices. The rewarding outcome also results in activation in the ventral striatum. Several studies reported that this reward response in the ventral striatum is higher in adolescents (typically 13–17-year-olds) compared to children and adults (Ernst et al., 2005; Galvan et al., 2006; Padmanabhan, Geier, Ordaz, Teslovich, & Luna, 2011; Van Leijenhorst, Moor et al., 2010; Van Leijenhorst, Zanolie et al., 2010), although not all studies have reported heightened activity (Bjork, Smith et al., 2010). A study that included different reward conditions, winning for self, for a friend or for an unknown other, showed that this heightened activity during adolescence was specific for self-relevant gains (Braams, Peters, Peper, Guroglu, & Crone, 2014).

Recently, the adolescent peak in neural activity in the ventral striatum to rewards was confirmed in a longitudinal study in which 254 adolescents between ages 8–27 years were scanned twice when performing a gambling task, with a 2 year period between the two scans (Braams et al., 2015). This study also allowed a better understanding of which behavioral tendencies co-vary with the neural response over time. It was found that the drive for reward, as measured with the BIS/BAS self-report questionnaire, in particular varied with the ventral striatum response to rewards. A similar sensitivity to individuals' ventral striatum response was observed in relation to reward sensitivity in a separate longitudinal study (van Duijvenvoorde et al., 2014). These longitudinal results provide a unique opportunity for relating changes in neural activation to changes in behavior over time. Even though most studies focused on rewarding and positive outcomes, some studies have also focused on other types of salient outcomes. For example, it was found that adolescents also show elevated activation compared with adults in the ventral striatum following an aversive event in the form of nontasty juice in comparison to a neutral baseline (Galvan & McGlennen, 2013). These findings suggest that the ventral striatum is more sensitive to affective learning signals in adolescence in general. Future studies should examine different types of feedback (reward, loss, pleasurable stimuli, aversive stimuli), and their relation to learning and behavioral adjustment, in more detail (Figure 3).

Although some consistent findings emerge, these studies also present some contradictory findings of adolescent-specific patterns of neural activation. Besides the use of different task paradigms, age-ranges, and baseline contrasts (Galvan, 2010; Richards, Plate, & Ernst, 2013), these contradictory findings may also point to the large individual differences within the adolescent age group. In addition, many studies report a monotonic decrease in risk-taking behavior between childhood and adulthood, while reporting a specific adolescent peak in neural activity (Galvan et al., 2006; Van Leijenhorst, Moor et al., 2010). Closer inspection of these behavioral patterns seems to suggest that adolescents often show larger variability in behavior (Ripke et al., 2012). A prior neuroimaging study reported large individual differences in adolescents' risk sensitivity ranging from very risk averse to risk-seeking, whereas adults were more uniformly risk averse (van Duijvenvoorde, Huizenga et al., 2015). Possibly, adolescents are more sensitive than adults to contextual cues, or show different types of sensitivity to context, such as social rewards (Willoughby et al., 2014). This may be a result of a transition period during adolescence in which they may be more susceptible to environmental influences (Crone & Dahl, 2012). Future approaches combining model-based influences with different experimental conditions may be a promising method to highlight these influences across development and to specify the extent of individual differences across developmental stages.

These imaging studies reviewed above have demonstrated regional age-related differences in neural activation during the process of risk-taking or reward processing. In general, the studies confirm adolescent-specific heightened activity in the striatum, both when anticipating rewards and when receiving rewards. In addition, the studies show that when *taking* risks (i.e., when evaluating different options and making voluntary choices), adolescents show heightened activity in regions that are thought

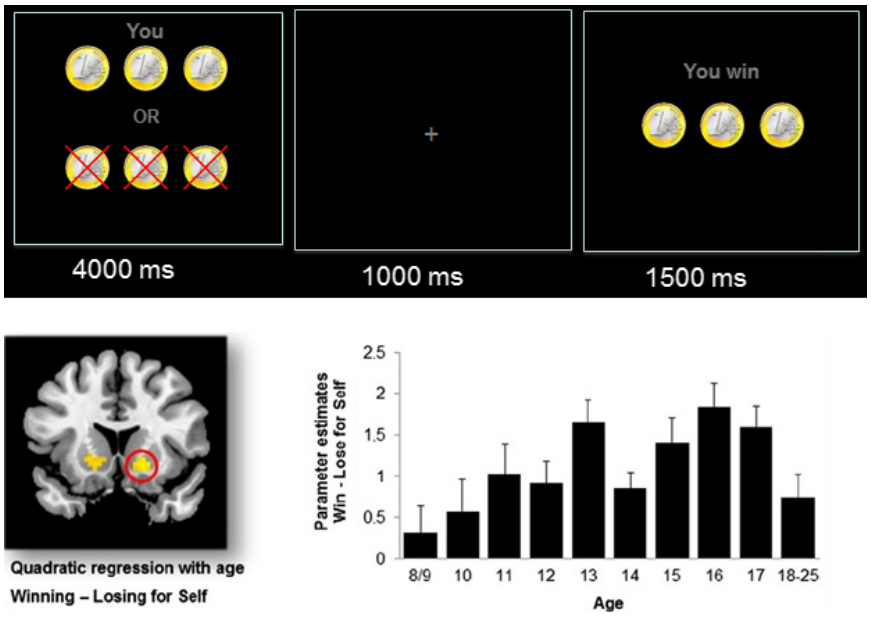

Figure 3 Example stimulus display and adolescent peak in ventral striatum activity in a gambling task with an equal probability of rewards and losses. Displayed is the ventral striatum brain region that shows an adolescent peak in reward responsiveness (adapted from Braams et al., 2014)

to work closely together with the PFC-striatum network, such as the subgenual ACC (which lies as the intersection of the striatum and PFC) and the insula. Together, these findings highlight both the consistent findings (in terms of heightened striatum activity during outcome processing), as well as the complexity (in terms of different regions working together when making choices) of understanding risk-taking development.

An additional question is to what extent connectivity between the striatum and PFC regions changes from trial-to-trial during choice or outcome. This question has been examined in imaging studies by examining connectivity patterns in adolescents of different ages. Although there are still only a few studies that have used this approach, several of these studies have reported an age-related increase in functional connectivity between the ventral striatum and medial PFC when receiving positive feedback relative to negative feedback (van den Bos, Cohen, Kahnt, & Crone, 2012) and gains compared to losses (van Duijvenvoorde et al., 2014). Even though this connectivity between ventral striatum and medial PFC was not related to increased risk-taking in the latter study (van Duijvenvoorde et al., 2014), the strength in connectivity between the ventral striatum and anterior insula attenuated individuals' risky decision making, indicating a regulatory role of this network. In addition, a recent longitudinal study reported that decreases in functional connectivity between the striatum and medial PFC between ages 15- and 17-years, were associated with decreases in risk-taking behavior (Qu, Galvan, Fuligni, Lieberman, & Telzer, 2015). Taken together, these functional connectivity patterns provide important additional information above an approach that only focuses on regional activity patterns over age. That is to say, regional activity and connectivity measures provide complementary information with respect to how certain brain structures are sensitive to rewards, and how brain regions interact when valuing rewards. Future studies are necessary, however, to understand these connectivity patterns in relation to behavioral changes in more detail.

Developmental changes and individual differences in functional connectivity related to risk-taking

A relatively new way to examine the relation between risk-taking and striatum-PFC connectivity is by examining functional activity during rest. Task-free connectivity patterns, also known as resting-state connectivity, assess synchronous activity between brain regions when subjects are awake but resting, and focuses on spontaneous low frequency fluctuations in the blood oxygen level dependent signal (Fox & Raichle, 2007). A great advantage of resting-state connectivity analysis is that it allows us to study age-related changes in intrinsic functional connectivity in children of all ages due to their minimal attentional demands. Also, resting-state analyses allow for both exploratory, data-driven analyses, such as independent component analyses, as well as the testing of specific hypotheses by use of seed-based approaches. Taken together, resting state connectivity is a powerful method that provides insight into the functional architecture of neural connections (see also Ernst, Torriso, Balderston, Grillon, & Hale, 2015).

A prior study examining resting state connectivity across adolescence aimed to test linear and nonlinear changes in a reward-based network. This study focused on the nucleus accumbens (a subregion of the ventral striatum) and used resting state data from 269 participants ranging between ages 8 and 25 years. Results showed that connectivity between the striatum and a ventral part of the medial PFC decreased with age. That is, specifically in the younger children functional connectivity between the striatum and VMPFC was high, and this connectivity declined into adulthood (Van Duijvenvoorde, Achterberg, Braams, Peters, & Crone, 2016). The study related the functional connectivity strength between the striatum and VMPFC to individual differences in self-report reward valuation, and found that individuals, who enjoyed winning money more, had stronger functional coupling between the striatum and VMPFC. This relation mediated the observed age-related decrease in winning pleasure (Van Duijvenvoorde, Achterberg et al., 2016). Similarly, a recent study using a relatively similar age range and sample (66 participants, ages 4–23 years; Fareri et al., 2015) also observed a decrease in functional connectivity between the striatum and VMPFC, particularly a subgenual part of the ACC. In this study, the age-related decrease was associated with increases in endogenous testosterone. Tentatively these results may suggest that a decline in positive coupling between these regions indicates a functional specialization of value-based processes, which may be partly driven by testosterone. The functional roles of the subgenual ACC versus the striatum may become more differentiated with age (e.g., Christakou, Brammer, & Rubia, 2011), although this will need to be tested in further studies.

Second, van Duijvenvoorde et al. (2014) observed a strengthening of connectivity between the striatum and dorsal regions of the medial PFC across adolescence (see Figure 4), although this strengthening was not related to reward valuation. This increase in functional connectivity between the striatum and the dorsal medial PFC may reflect an increase in top down regulation over the striatum. Several prior studies have suggested that there are different roles of the medial PFC along the dorsal to ventral axes (Beckmann, Johansen-Berg, & Rushworth, 2009; Crone, 2014). In a functional neuroimaging study using a two-choice probabilistic gambling task it was found that VMPFC activity was related to increased risk-taking behavior, whereas dorsal medial PFC activity was related to decreased risk-taking behavior (Van Leijenhorst, Moor et al., 2010). Together, these results suggest that in late childhood functional coupling between the striatum and VMPFC may result in increased reward valuation, and with development increasing functional specialization leads to a decoupling of these regions that supports reward-based learning and controlled behavior. In addition, age-related increases in functional integration between the striatum and the more dorsal MPFC regions may also lead to more regulated behavior.

A third study examined functional connectivity between the VMPFC and two subcortical regions: the striatum and the amygdala, in relation to alcohol use. It is well documented that alcohol use increases in adolescence and this is often considered a real life indicator of risk-taking. Advanced pubertal development and higher testosterone levels were associated with more alcohol intake, and activity in the striatum was related to more recent alcohol use in adolescence (Braams et al., in press). Therefore, in a recent study the hypothesis was tested that VMPFC connectivity to these subcortical regions would be related to alcohol use. Contrary to expectations there was no relation between VMPFC and striatum connectivity and alcohol use, but the connectivity between VMPFC and amygdala was a strong predictor of alcohol use (Peters et al., 2015). Several prior studies already reported that the amygdala is important for predicting alcohol use, and the triadic imbalance model of adolescent brain development also points to an important role of the amygdala in adolescent decision making (Richards et al., 2013). Thus, an important direction for future studies is to extend the model to other affective brain structures, especially when aiming to explain more complex daily life risk-taking. For example, a study that compared risk-taking adolescents with nonrisk-taking adolescents showed that risk-taking adolescents were characterized by connectivity between the

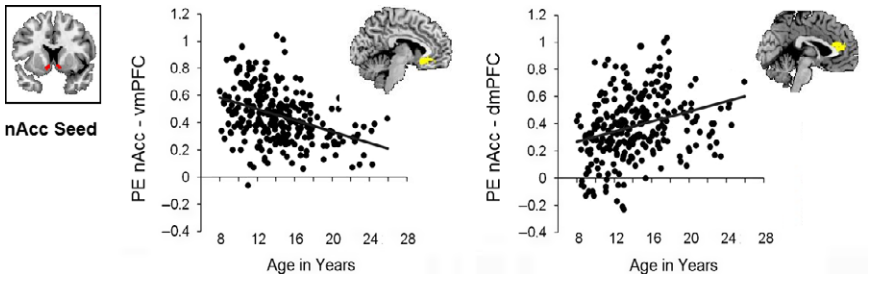

Figure 4 Resting-state connectivity between the nucleus accumbens (nAcc) seed and the ventral medial prefrontal cortex (VMPFC) and dorsal medial PFC (dmPFC) across adolescence. Dots display extracted parameter estimates in brain regions sensitive to age-related change as observed in a whole-brain seed-based analysis (From Van Duijvenvoorde, Achterberg et al., 2016, with permission)

amygdala and PFC, whereas nonrisk-taking adolescents showed stronger connectivity between the striatum and dorsal, but not ventral, medial PFC (DeWitt, Aslan, & Filbey, 2014).

How do individual differences inform what we know about childhood disorders?

Youth with disruptive behavioral disorders typically show more risk-taking problems compared to their age-matched peers (Bjork & Pardini, 2015). Dual-processing models have suggested that risky behavior, such as alcohol consumption, illegal drug use or committing minor crimes, start in adolescence, which creates a period of risk for healthy development (Casey & Jones, 2010; Somerville et al., 2010). Indeed, many psychiatric illnesses, such as substance abuse, anxiety or depression, have their onset in adolescence, suggesting that the reorganization in brain development during this period may create vulnerabilities (Paus, Keshavan, & Giedd, 2008). However, it is not yet well determined if externalizing disorders reflect an extreme on a continuum of risk-taking behavior or represent a different underlying developmental trajectory (Plichta & Scheres, 2014).

Several studies have examined the relation between activation in the ventral striatum to rewards and individual risk-taking propensity and reported a positive relation in healthy adolescents (van Duijvenvoorde et al., 2014; Galvan, Hare, Voss, Glover, & Casey, 2007). Greater activation in the ventral striatum to rewards was also observed in individuals who score high on sensation-seeking, although a family history of alcohol had no effect (Bjork, Knutson, & Hommer, 2008). Together, these findings suggest that ventral striatum responsiveness is higher for individuals who are more risk-seeking. A longitudinal structural brain development study showed that adolescents with smaller ventral striatum volume were more likely to have initiated alcohol use 2 years later (Urosevic et al., 2015). How connectivity between the striatum and PFC relates to risk-taking behavior exactly remains poorly understood, with inconsistent findings between studies (Berns, Moore, & Capra, 2009; Jacobus et al., 2013). Possibly, these effects are dependent on the developmental time course, the subregion within the striatum and PFC that is being studied, or whether the tasks measures dangerous or adaptive types of risk-taking.

There is some evidence in favor, and some evidence against the hypothesis that disruptive behavioral disorders, or externalizing disorders, are extremes of the normal variation in risk-seeking behavior. In a large study including 17–18-year-old adolescents with ADHD, their siblings, and adolescents without ADHD, it was found that on a reward task, the ADHD participants showed heightened activity in both the striatum and the VMPFC relative to adolescents without ADHD, with an intermediate patters for siblings of adolescents with ADHD (von Rhein et al., 2015). Furthermore, increased functional connectivity between the nucleus accumbens and the PFC has been found to be associated with greater impulsivity in children with ADHD (Costa Dias et al., 2013). Interestingly, heightened brain activity within the striatum and/or PFC is not always observed in ADHD: for instance, adults with ADHD show reduced reward responses in the striatum (Kappel et al., 2015). Thus, it is possible that the reward sensitivity in ADHD expresses itself differently over the course of development (Plichta & Scheres, 2014).

Another study compared 13–17-year-old adolescents with externalizing disorders (conduct disorder, oppositional defiant disorder, and ADHD) with healthy age-matched adolescents on a reward anticipation and receipt task. This study showed heightened activity in the ventral striatum in adolescents with externalizing disorders specifically to receiving reward outcomes (Bjork, Chen, Smith, & Hommer, 2010). However, yet another study on 10–17-year-old adolescents with externalizing disorders (conduct disorder and oppositional defiant disorder), observed reduced activity in the ventral striatum to reward cues relative to a healthy control group (White et al., 2014).

Finally, adolescents age 14–18-years with substance abuse disorder showed no differences in ventral striatum activity on a risk-taking task, but showed reduced activity in VMPFC when experiencing rewards, and heightened activation in lateral orbitofrontal cortex when receiving unexpected losses (Crowley et al., 2010). These differences between studies may depend on several individual difference factors such as the presence or absence of callous-unemotional traits. However, despite the striatum-VMPFC differences in clinical groups, the studies often show inconsistent findings in terms of the direction of the differences. There is a need for studies that include children, adolescents, and adults, with and without externalizing disorder, to test the question of whether reward sensitivity is specific for a certain developmental phase and to unravel the role of individual differences in personality traits.

Conclusions and future perspectives

This review discussed how ventral striatum-PFC connections relate to the development of risk-taking behavior in adolescence. We discussed the role of structural morphology, structural connections, functional connections, and task-related activity in explaining individual patterns of risk-taking behavior, in the context of the dual processing model of adolescent brain development (Somerville et al., 2010). Even though the relation between the ventral striatum and the PFC in relation to risk-taking is well established in adults, only recently have researchers

extended these methods to the question how adolescent risk-taking changes across development. As structural and functional connectivity between striatum and dorsal medial and lateral PFC increases, as is observed across adolescent development, individuals become less impulsive. In addition, as functional connectivity between striatum and VMPFC decreases (or becomes more independent), as is observed across adolescence, individuals become less reward sensitive. Thus, as children grow up, the network changes considerably which is associated with more future oriented, less impulsive choice.

Intriguingly, neural activity in both the ventral striatum and VMPFC is heightened in midadolescence relative to childhood and adulthood, although this is dependent on specific task conditions (Richards et al., 2013). Together, these findings suggest that in the process of brain maturation, specifically the maturation of ventral striatum-PFC connectivity, there is possibly enhanced sensitivity of this reward-seeking and valuing network, which is associated with a peak in certain types of risk-taking behavior (Braams et al., 2015; Figner et al., 2009). Possibly, a temporarily less inhibited system allows adolescents to explore their environment, seek out new relations and differentially value information that they receive (Crone & Dahl, 2012).

Going back to the long-standing debate whether risk-taking is observed in *all* adolescents and as such is a developmental hallmark of adolescence, or whether it is present in only a *subset* of adolescents who have already been susceptible to problem behavior in childhood (Bjork & Pardini, 2015), the results suggest some heightened reward sensitivity, at least in neural activity (Braams et al., 2015), in most adolescents. However, in terms of actual risk-taking behavior, research to date remains inconclusive (Willoughby et al., 2014), and the relation between reward sensitivity and risk-taking behavior is not yet fully understood. It remains an important question when increased risk-taking is part of normative development and when it is an indication for potential negative outcomes, such as sensitivity for developing disruptive behavior or substance dependency.

One potential way of investigating this in further detail will be to combine longitudinal research with a relatively young (preadolescent) sample. Consequently, (neuro)biological characteristics of children who develop extreme or pathological forms of risk-taking could be detected when moving into adolescence, compared to children who do not develop extreme forms of risk-taking. Stemming from this review, reduced striatum-PFC connectivity and enhanced reward sensitivity as well as increased sex steroid hormone secretion are likely candidates for these biological characteristics.

A second crucial question concerns whether the heightened reward sensitivity causes only vulnerabilities for excessive 'bad' risk-taking behavior, or is also potentially a driver for positive, motivated behavior. For example, friendly emotional faces are also typically associated with ventral striatum activity which is stronger for adolescents than for children and adults (Somerville, Hare, & Casey, 2011). In addition, peers enhance neural activity in the ventral striatum in adolescents (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011), but peers also reinforce positive helping and donating behavior (Van Hoorn, Van Dijk, Meuwese, Rieffe, & Crone, 2015). It was previously found that adolescents who have stronger family obligations show more activity in the ventral striatum when they donate money to the family (Telzer, Fuligni, Lieberman, & Galvan, 2013), suggesting that heightened emotional reactivity in the striatum can also be associated with the rewarding feeling of caring for others.

One way to examine this question in more detail is by moving beyond only testing snap shots in development, and focusing on individual trajectories using longitudinal measures. There are now several longitudinal studies in the literature in which it was found that individual differences in reward-seeking behavior or risk-taking propensity are important covariates of neural activity in ventral striatum and VMPFC (Braams et al., 2015; van Duijvenvoorde et al., 2014). In addition, longitudinal measures have the potential to unravel the question of whether neural architecture predicts behavioral outcomes or vice versa. For example, it remains an important question whether substance abuse influences subsequent brain development or whether certain trajectories of brain development create a vulnerability factor for developing substance abuse.

Finally, understanding risk and reward sensitivity over the course of development will prove extremely valuable for understanding the emergence of child- and adolescent-specific impulsivity disorders, because these studies often suffer from inconsistent findings across studies (Plichta & Scheres, 2014). A crucial direction for future studies will be to use multi-model approaches, which will gain insight into how functional and structural connectivity develop relative to each other. Several of these approaches were illustrated in this review. In addition, it will be important to measure a wide range of risk-taking behaviors, such as dangerous versus adaptive risk-takers, and include control-related and affective processes together. The dynamic inter-relations between these processes will be one of the key focus points for future research.

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Key points

- This review describes the most recent insights in neural processes involved in risk-taking, with a focus on brain structure, function, and connectivity, in healthy adolescents and adolescents with impulsivity disorders.
- The review moves beyond a discussion of regional functional activity, and puts emphasis on connectivity patterns as important determinants for age changes and individual differences.
- Specifically ventral striatum – prefrontal cortex connectivity is related to individual differences in risk-seeking behavior. Both regional activities and connectivity patterns provide complementary information on risk-taking development.
- Longitudinal measurements of ventral striatum – prefrontal cortex activity and connectivity will prove crucial for our understanding of the emergence of impulsivity disorders, such as ADHD and conduct disorder.

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