Review article

What motivates adolescents? Neural responses to rewards and their influence on adolescents' risk taking, learning, and cognitive control

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A B S T R A C T

Adolescence is characterized by pronounced changes in motivated behavior, during which emphasis on potential rewards may result in an increased tendency to approach things that are novel and bring potential for positive reinforcement. While this may result in risky and health-endangering behavior, it may also lead to positive consequences, such as behavioral flexibility and greater learning. In this review we will discuss both the maladaptive and adaptive properties of heightened reward-sensitivity in adolescents by reviewing recent cognitive neuroscience findings in relation to behavioral outcomes. First, we identify brain regions involved in processing rewards in adults and adolescents. Second, we discuss how functional changes in reward-related brain activity during adolescence are related to two behavioral domains: risk taking and cognitive control. Finally, we conclude that progress lies in new levels of explanation by further integration of neural results with behavioral theories and computational models. In addition, we highlight that longitudinal measures, and a better conceptualization of adolescence and environmental determinants, are of crucial importance for understanding positive and negative developmental trajectories.

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Thought by itself moves nothing

Adolescence is one of the periods in life well known for its changes in motivated, goal-directed, behavior. These changes are thought to result in a greater emphasis on potential rewards, such as a heightened behavioral motivation to obtain rewards and a heightened arousal in response to rewards (Galván, 2010). As a crucial aspect of almost all behavior and actions, motivated behavior has been investigated from a number of perspectives including economics, sociology, psychology, and neuroscience. Recent discoveries in neurocognitive research have led to new perspectives on the dynamic changes in motivated behavior during adolescence. In the current review we will discuss changes in adolescent’s reward sensitivity by reviewing recent cognitive neuroscience findings in relation to behavioral outcomes.

1. Adolescence as a period of change in motivated behavior

Adolescence is defined as an important transitional period between childhood and adulthood in which individuals gain independence and develop mature social goals (Crone and Dahl, 2012). As such, adolescence represents a developmental time window characterized by strong needs for exploration, forming new relationships, increasing intimacy, and rapid adjustment to changing social environments. Although the age range of adolescence differs between countries and cultures, it is generally agreed upon that in Western societies adolescence approximately spans the period between ages 10 and 22 years. Puberty is an important phase of adolescence and characterized by a rapid rise in gonadal hormones (Blakemore et al., 2010). These hormones are released through the hypothalamus-pituitary-gonadal axis and have a large influence on bodily characteristics and brain development (Peper and Dahl, 2013; Goddings et al., 2014). Pubertal development reaches a plateau in mid-adolescence at approximately age 15–16 years (Braams et al., 2015). The end phase of adolescence, however, is culturally defined and is reached when individuals have achieved mature social and personal responsibilities (e.g., Cohen et al., 2016).

Adolescence has traditionally been interpreted as a period of changes in motivated behavior. Some of the earliest theories in developmental psychology have described adolescence as a period of storm and stress, including conflict with parents, mood disruptions and a variety of risk behaviors, which were thought to be universal and biological (Arnett, 1999; Hall, 1904). Epidemiological reports have indeed observed an increase in risk taking behavior in adolescence, such as a higher incidence of traffic accidents, delinquency, and substance abuse (see Eaton et al., 2011; Willoughby et al., 2013). To reconcile these findings with the general cognitive increase observed in adolescence (e.g., Crane 2009), research tuned to the role of affective-motivational processes in understanding changes in adolescent’s behavior (e.g., Boyer, 2006). Developmental studies reported a surge in sensation seeking during adolescence (Zuckerman, 1994) that may lead to increases in peer influence and risky behavior (Steinberg et al., 2008; Romer, 2010). Recent approaches have linked these changes in motivated behavior to the long-lasting neurodevelopmental changes across adolescence. Since the discovery that brain development continues throughout adolescence, research on adolescent brain development has expanded enormously. It was discovered that adolescence is a period of continuous changes in brain morphology. In grey matter there is an overall reduction in volume and cortical thickness (Giedd et al., 1999; Gogtay et al., 2004; Tamnes et al., 2010; Raznahan et al., 2011; Mills and Tamnes, 2014; Wierenga et al., 2014), which is thought to reflect synaptic pruning, occurring in a time- and region-specific manner (Huttenlocher 1990; Petanjek et al., 2011). At the same time white matter volume increases and undergoes changes in the organization of its connections (Lebel and Beaulieu, 2011; Simmonds et al., 2014). Together, these changes allow for greater specialization and strengthening of connectivity between brain regions. These changes in structural development are paralleled by changes in functional activity in the brain, such as activation in response to motivationally salient events (for an overview of studies, see Crone and Dahl, 2012; Crone et al., 2016), and changes in subcortical and cortical functional connectivity during rest (Gabard-Durnam et al., 2014; Fareri et al., 2015b; van Duijvenvoorde et al., 2016).

The structural and functional age-related changes in the brain have been summarized in neurocognitive models, which suggest that adolescent motivation is particularly tuned towards rewarding stimuli. According to these models, which are also referred to as dual processing or imbalance models, adolescent brain development can be described as an imbalance in neural maturation patterns between the cortical-control system (including brain regions such as the prefrontal cortex), important for the regulation of thoughts and emotion, and the limbic system (including brain regions such as striatum and amygdala), important for affective-motivational functioning (e.g., Ernst, 2014; Somerville et al., 2010; Steinberg, 2008). During adolescence the reactivity of the affective-motivational system may be particularly heightened and—depending on adolescents’ motivations—may override controlled responses in emotionally-salient contexts (Somerville et al., 2010; Crone and Dahl, 2012). Consistent with these neurocognitive models, behavioral literature on adolescent risk-taking has indicated that in emotionally arousing situations, adolescents are more prone to taking risks. For instance, a recent meta-analysis showed that heightened risk taking in adolescents compared to adults, although particularly in contexts when rewards are encountered immediately (Defoe et al., 2015; see also Figner et al., 2009; van Duijvenvoorde et al., 2010). A similar finding is observed when adolescents are in the presence of peers (Gardner and Steinberg, 2005; Peake et al., 2013), or when approaching unknown situations (Blankensteijn et al., 2016; Tymula et al., 2012).

New models in this tradition increasingly acknowledge the complexity of brain and behavioral changes across adolescence. In order to understand changes in adolescent motivated behavior these models stress a) the importance of studying brain-connectivity within and between limbic, affective-motivational, and cognitive control brain circuits (Casey, 2015; Casey et al., 2016), b) the influence of social context on observed neural sensitivities (Shulman et al., 2016; Nelson et al., 2016), and c) the importance of pubertal developmental changes and cortical-subcortical flexible interactions (Crone and Dahl, 2012). More specifically, the first model (Casey, 2015) highlights the hierarchical development of brain circuitry, in which development and sensitivity of subcortical circuits (e.g. subcortical–subcortical connections) precede others (e.g., top-down cortical control connections), in order to develop. The second model highlights that an imbalance between cortical and subcortical brain systems may not occur under all circumstances and depends on contextual factors, such as peer presence (Shulman et al., 2016). Finally, it has been proposed that increases in pubertal hormones at the onset of puberty trigger the limbic system to
flexibly recruit cortical control regions, depending on the motivational goal of the behavior that is displayed (Crone and Dahl, 2012). Illustrations of a number of these recent models can be found in Fig. 1.

In summary, neurocognitive models regard adolescence as a key developmental period for changes in motivation and have defined adolescence to coincide with a particularly greater sensitivity towards potential and obtained rewards. On the other hand, these models have been characterized as heuristic models that provide general and relatively unfalsifiable hypotheses on developmental patterns (Pfeifer and Allen, 2012; van den Bos and Eppinger, 2016). Future specifications of the models should describe how to derive specific and testable hypotheses. The importance of these models, however, has been evident and an exciting step forward is moving into specifying models to match behavioral and neural changes across development.

A greater reward-related motivation in adolescence has been predominantly linked to behaviors such as aberrant decision-making and risk taking such as alcohol abuse, substance use problems, and excessive risk taking. However, changes in reward-related motivation may additionally lead to exploring new environments, achieving high social ranks, or experimenting with new social roles in society (Crone and Dahl, 2012). Thus, reward-sensitivity may be equally important for understanding positive developmental trajectories in learning, sports, and social behavior (Dahl and Vanderschuren, 2011; Telzer, 2016). In this review, we will discuss the influence that rewards may have on adolescent’s adaptive and maladaptive behavior across different domains in a neurocognitive perspective. To this end, we first describe brain regions involved in processing of rewards in adults and adolescents, with a specific focus on core reward regions, such as the striatum and its connected circuitry. Second, we discuss the functional changes in reward circuitry related to (mal)adaptive risk taking and decision making during adolescence. Third, we describe how reward circuitry is related to adolescent (mal)adaptive cognitive control and learning. In both domains we will consider social context as an important moderator of adolescent’s behavior. Fourth, we will discuss the potential for using a model-based approach, which allows for the development of specific, testable hypotheses of decision-making and learning in adolescence. Finally, we will discuss the challenges for studying adolescence as a key transition period for motivated behavior, along with the broader implications and conclusions arising from this review.

2. The neuroscience of reward sensitivity

Reward processing in the brain has been examined from a variety of perspectives. Animal research has shown that dopamine innervated regions such as the basal ganglia and its cortical targets (i.e., cortico-striatal loops) are central to basic reward processing and motivated behavior (e.g., Haber and Knutson, 2010). In both animal and human research, several major dopamine-rich pathways have been identified, including the mesolimbic and mesocortical pathways. These pathways project from the midbrain ventral tegmental area to the nucleus accumbens (mesolimbic dopamine pathway) and from the ventral tegmental area to parts of the prefrontal cortex (mesocortical dopamine pathway).

In humans, functional magnetic resonance imaging (fMRI) has been used extensively to examine functional activation in the brain during the anticipation and the receipt of reward (see Richards et al. (2013) for an excellent review). Several meta-analyses, which include reward-processing across a wide-range of decision paradigms, have consistently reported activation in a neural reward network including structures of the subcortical limbic system (Bartra et al., 2013; Cauda et al., 2011; Diedenhof et al., 2012; Liu et al., 2011; Sescousse et al., 2013; see also Fig. 2). Furthermore, these meta-analyses demonstrated activation in the medial prefrontal cortex (PFC), the orbitofrontal cortex (OFC) and ventral medial PFC, as well as in the insula, the anterior cingulate cortex (ACC), the posterior cingulate cortex (PCC), the inferior parietal lobule and regions in the lateral PFC (e.g., Liu et al., 2011). In another meta-analytic study, Sescousse et al. (2013) argued that overlapping neural networks are involved in coding both primary and secondary reward values. In adults, neural correlates of primary (such as food, sex and shelter) and secondary rewards (such as money or power) were found in the ventral striatum, the thalamus, the OFC, the insula, and the amygdala (see Fig. 2). These findings indicate that rewards elicit a consistent pattern of functional activation across dopaminergic innervated reward regions in the human brain.

To directly assess the relation between dopamine responses and blood-oxygenated level-dependent (BOLD) brain activation, a recent study used an innovative combination of optogenetic methods (allowing to experimentally control activation of neurons) and functional MRI in rats (Ferenczi et al., 2016). Findings showed, for the first time, that dopamine neuron stimulation was directly related to striatal BOLD activity. Also, the influence of the cortical circuitry over subcortical activation was evidenced by the finding that increased medial PFC excitability reduced striatal-midbrain coupling, and in this way suppressed reward-seeking behavior. These findings highlight the importance of the interaction between cortical and subcortical circuitry in driving motivated, reward-driven, behavior and present an important model for human data (see also Casey, 2015 for a discussion of translational models in adolescent’s motivated behavior).

When considering the development of these reward networks, animal research has shown marked changes in the expression and pruning of dopamine receptors during the transition into and out of adolescence (Spear, 2000; Spear, 2011), which may underlie developmental changes in adolescent’s reward-related behavior and neural activation (Luciana and Collins, 2013). Developmental changes in the dopamine system are, however, not only character-
ized by changes in the receptor density, but also by changes in the different firing modes (phasic/tonic) of dopamine neurons, and by changing interactions with other neurotransmitter systems (particularly glutamate and GABA) that may be relevant for motivated behavior (Ernst and Luciana, 2015). The complexity of this system is evident. A remaining challenging step is therefore to apply these insights of neurotransmitter remodeling in order to understand changes in adolescent motivated behavior (e.g. Luciana et al., 2012; Doremus-Fitzwater et al., 2011).

3. Reward sensitivity in adolescence

The specific neural architecture of reward processing changes across adolescence has been extensively studied across a large variety of paradigms. A recent meta-analysis pooled data from 26 studies and more than 800 individuals to identify the network of brain regions involved in adolescent reward processing (Silverman et al., 2015). Interestingly, adolescents and adults recruited overlapping brain regions, including the ventral striatum, the insula, and the posterior cingulate cortex. However, adolescents generally showed an increased likelihood of activation in these subcortical and cortical reward-related regions.

Activation of these reward regions across adolescence may depend on interactions with long-maturing brain regions such as the lateral PFC and parietal cortex, which have been typically related to a series of cognitive control functions (e.g., response inhibition, relational reasoning, working memory, and learning) (Crone 2009; Luna et al., 2010; Niendam et al., 2012; Diamond, 2013). Additionally, co-activations are regularly observed between reward-related regions and brain regions important for perspective taking, mentalizing and social behaviors, including the medial PFC and the temporal-parietal junction (Blakemore et al., 2010; Mills et al., 2014). Taken together, activity in core reward-regions is likely dependent on a larger interconnected network of subcortical and cortical regions in driving behavioral outcomes. These interactions, and their developmental changes, will be highlighted in some of the reviewed studies.

4. Adolescent reward-sensitivity and risk taking

Neurocognitive models of adolescent brain development have suggested that adolescent risk taking and decision making is driven by heightened activation of the neural reward system during adolescence, especially focusing on the ventral striatum. Risky behavior may come with substantial individual and societal costs, yet in some cases greater risk taking could be an adaptive feature, which may be particularly prominent in adolescence (Crone and Dahl, 2012). For instance, when exploring new environments, taking an unknown risk may entail several benefits. Here, we describe findings on adolescents’ neural reward-sensitivity in relation to maladaptive and adaptive behavioral risk-taking.

4.1. Reward-sensitivity and risk taking

One way to test neural reward-sensitivity and its behavioral outcomes in adolescence is by using decision-making tasks where choices can result in rewards or losses. For example to test the developmental pattern of neural responses to rewards in an adolescent sample, we conducted a two wave longitudinal study including 254 adolescents between the ages of 8 and 27 years to examine neural responses to winning and losing money in a gambling game (Braams et al., 2015). Results showed strong activation in the ventral striatum, and specifically the nucleus accumbens (NAcc), when winning compared to losing across all ages. Moreover, a quadratic trend of reward-related neural activation in the NAcc, peaking in late-adolescence, described the data best. These results extend findings from the meta-analysis comparing adolescents and adults (Silverman et al., 2015) by showing stronger reward activity in the ventral striatum in adolescents compared to adults and children (see also Galván et al., 2006).
To further test the functional relevance of these age-related findings, we also investigated the relationship between neural responses to rewards and pubertal developmental, hormone levels, behavioral risk taking on the balloon analogue risk task (BART), and self-reported reward-sensitivity. The latter was measured with the behavioral inhibition and behavioral activation scale (BIS/BAS), a well-validated questionnaire in which subscales measure different aspects of reward-related behavior. Results showed positive associations between reward activation in NAcc and self-reported reward drive. That is, those participants who indicated they were willing to exert more effort for a reward also showed greater NAcc responses to rewards (Braams et al., 2015; see also van Duijvenvoorde et al., 2014; Op de Macks et al., 2011). Another two-year longitudinal study observed an increase and peak in reward sensitivity in late adolescence (as indicated by BAS reward-responsiveness). Grey matter volume of the NAcc explained part of the developmental changes in reward-responsiveness (Urošević et al., 2012).

Several studies have tested whether neural activation in reward areas is related to real-life risk taking behavior. These studies showed that reward-related neural activation is indeed positively related to a variety of risky real life behaviors including risky sexual behavior, illicit drug use and binge drinking (Bjork and Pardini, 2015; Braams et al., 2016; Galván et al., 2007; see Fig. 3). Also structural developmental studies observed relations with real-life substance use, indicating that adolescents with smaller NAcc volumes were more likely to initiate substance use in a 2-year follow-up period (Urošević et al., 2015). Finally, functional coupling between subcortical limbic structures and the OFC has been related to risky real-life behavior. That is, self-reported rule-breaking behavior has been related to a functional coupling of the striatum and OFC (Qu et al., 2015). Moreover, alcohol use in adolescent boys was associated with lower connectivity between the amygdala and OFC (Peters et al., 2015). Together, this set of studies suggests that functional activity, connectivity, and structural development within a subcortical reward-network may be a marker for the propensity to display behaviors possibly detrimental to individual health.

On the other hand, adolescents’ increased response towards rewards may also drive adaptive risk taking, a link that has yet been tested in few studies. For instance, a recent study showed that for adolescent girls (ages 8–25), a higher secretion of testosterone was related to increased risk taking, but this risk taking led to higher financial outcomes (Peper et al., 2013). Moreover, OFC morphology mediated the relation between testosterone and increased the level of this adaptive risk-taking (gaining money by taking risks). This relation was not observed in boys, who instead demonstrated increased maladaptive risk-taking with higher testosterone levels. These findings suggest that there may be gender differences in how pubertal hormones affect brain development, and possibly, this may result in different outcomes of risk-taking in boys and girls. Consistent with these findings, a recent study demonstrated that adolescent girls (11–13-years) who showed more reward exploration (a risky, but possibly adaptive choice) in an explore-exploit paradigm had stronger resting-state connectivity between the insula and lateral prefrontal cortex than girls that explored less (Kayser et al., 2016). Until now, only a few studies addressed sex differences and pubertal influence in (mal)adaptive risk taking. However, there is accumulating evidence that pubertal hormones may differentially influence boy’s and girl’s brain and behavior (see Gur and Gur for an excellent review, in press). Including such effects in future studies may be highly informative to understand individual differences in adolescent’s adaptive and maladaptive risk taking.

4.2. The influence of social context on reward-sensitivity
Adolescence is a time of social reorientation in which a shift occurs from a focus on parents to a focus on peers (Rubin et al., 2008) and social contexts may influence neural response to rewards in adolescence. In the next section, we discuss the influence of social context on adolescents’ reward-sensitivity across three social domains: peer presence, peer influence, and vicarious rewards (i.e., gaining for other).

4.2.1. Peer presence
In a computerized risky driving task that was played either alone or in the presence of a peer (Chein et al., 2011), adolescents took more risky crossings and experienced a higher number of crashes (ages 14–18) when peers were present. Young adults did not demonstrate this peer effect. The authors observed enhanced activation in the ventral striatum and OFC when peers were present, but only for the adolescent age group, and specifically for the anticipatory decision phase. A similar effect was found when adolescents (14–19 years) and adults (25–35 years) played a gambling task where they had to guess whether a next playing card would be higher or lower than the current card (Smith et al., 2015). When playing the game alone, no differences were observed between adolescents and adults, but when playing with peers present, adolescents showed stronger activity in the ventral striatum. Speculatively, one mechanism that may underlie the influence of peer presence is a heightened sensitivity to peer evaluation (Somerville et al., 2013). A recent study showed that while seemingly being viewed by a peer in a live video feed, adolescents’ self-report indicated rising embarrassment. Adolescents also showed greater physiological arousal than children and adults, which was mimicked by emergent recruitment of the medial PFC, most prominently increasing up to mid-adolescence. Adolescent-emergent engagement of the medial PFC has been suggested to reflect, or perhaps result in, a high degree of salience, emotional arousal, and self-relevance in social-evaluative situations. Interestingly, in the driving task neural activations in the ventral striatum were stronger for adolescents who reported less resistance to peer influence (Chein et al., 2011), further strengthening the hypothesis that adolescents are more sensitive to social evaluations.

Apparently just the mere presence of peers has a pronounced effect on adolescent behavior and neural activation, more so than in adults. This seems consistent with real-life scenarios in which risk-taking behaviors in adolescents occur more readily when they are among their peers (see Steinberg, 2008). It may be that the presence or potential evaluation of peers, triggers risky behavior by increasing salient contextual cues, such as the potential for rewards (or losses). To investigate whether adolescent’s reward-sensitivity can also be reduced by social context, a recent study tested whether the presence of mothers modulated neural reward responses and observed risky choices in adolescents (Telzer et al., 2015). Adolescent risky choice was reduced in the presence of mothers compared to when being alone, as well ventral striatum activation during risky choice. In contrast, during safe decisions, there was increased recruitment of the lateral PFC and greater functional connectivity between lateral PFC and ventral striatum in the presence of mothers. These findings fit well with suggestions that connectivity between the medial and lateral PFC and the striatum are crucial for integrating signals relevant to social contexts (Somerville et al., 2013).

4.2.2. Influence of others’ behavior on risky choice
An intriguing question is the extent to which peers can alter risk taking towards riskier versus safe directions (see also Alberts et al., 2013). Behavioral studies showed that adolescents’ risk perceptions and prosocial behavior can increase or decrease based
on peer-expressed norms (Knoll et al., 2015; van Hoorn et al., 2016; Blankenstein et al., 2016). A recent imaging study tested whether neural responses may be predictive of the influence of peer-expressed norms of driving behavior (Cascio et al., 2014). First, adolescents’ (ages 16–17) neural responses were measured during a response inhibition task. In a second behavioral session, adolescents were paired with a peer expressing either risky or safe driving norms. Results showed that adolescents displayed riskier behavior in presence of the risky compared to the safe peer. Moreover, activation in the ventral striatum and prefrontal cortex during the response inhibition task predicted the influence of the safe peer on adolescents’ driving behavior. Thus, higher activation in these brain regions was associated with engaging in fewer risks when paired with cautious peers (Cascio et al., 2014). These findings demonstrate that stronger inhibition activity in a cognitive control task can predict how much adolescents are influenced by the social norms of a safety-promoting peer.

4.2.3. Vicarious rewards: winning for someone else

The previous sections focused on the extent to which peers enhance behaviors with potential negative outcomes, such as risk taking while driving. However, sharing and vicarious rewards may also be highly rewarding for adolescents. This has been tested in social decision-making paradigms in which money can be gained for different beneficiaries. A study by Fareri et al. (2012) with adult participants showed that ventral striatum responses are stronger when sharing money with a friend compared to a computer or a disliked other. With a model-based approach it was found that striatal and medial PFC responses predicted a social value reward signal that arose from reciprocation and depended on the closeness of the relationship with the interaction partners (Fareri et al., 2015a).

Similar results were obtained in a study with adolescent participants in which money could be won for best friends and disliked others. Ventral striatum responses were higher when winning for a friend than losing for a friend, whereas this was not found when winning money for a disliked other. Interestingly, this effect was modulated by the strength of the social bond. Girls who showed the highest responses to winning money for their best friend also reported higher friendship quality with this same friend (Braams et al., 2014). Striatum responses in this study may be related to empathic feelings towards friends, which makes reward of others feel as rewarding as rewards for self. Given the stronger focus on peer networks in adolescence, this suggests that reward sensitivity may be an important factor in forming social bonds.

An interesting new direction of research has linked the elevated neural response to rewards in adolescence to prosocial motivations. In a longitudinal study in adolescents, Telzer et al. (2014) investigated neural responses to monetary gains in a risk-taking task that resulted in either gains for the self (hedonic rewards: self-pleasure) and a donating task that resulted in gains for family members (eudaimonic rewards: pleasure for others). They reported that those adolescents who showed stronger ventral striatum activity to hedonic rewards reported an increase in problem behavior one year later, but adolescents who showed stronger ventral striatum reactivity to eudaimonic rewards showed a decrease in problem behavior over time.

These findings suggest that changes in adolescents’ reward-related responses pose opportunities for positive prosocial
development, such as increases in cooperation, sharing and helping, that can be influenced by the social context that we find ourselves in. These findings also provide important starting points for research in adolescents with social developmental problems such as autism, social anxiety and antisocial behavior. Very little is yet known about how exaggerated neural reward responses to risk and reward-seeking behaviors affects adolescents with problem behaviors and how the environment plays a role in guiding or shaping developmental pathways (Schriber and Geyer 2015).

5. Influence of adolescent reward sensitivity on cognitive control and learning

Compared to the number of studies that have focused on the influence of reward-sensitivity on risk taking in adolescence, it seems that less is known about how enhanced reward-sensitivity affects cognitive functioning, which may show equally adaptive and maladaptive features. The driving force behind general cognitive development is thought to be cognitive control, defined as the ability to control thoughts and actions. Cognitive control abilities start to emerge in early childhood, gradually improve over childhood and through adolescence, and consist of multiple constructs such as working memory, inhibition, and flexibility as well as different higher level functions such as reasoning, performance-monitoring, and planning (Diamond, 2013). Cognitive functioning may be greatly influenced by reward-sensitivity, as it may influence the level of motivation for applying cognitive control. For instance, cognitive control may be hindered in a social context, such as through decreased concentration when peers are present, but it is also possible that (social) rewards lead to increases in cognitive performance, due to increased motivation.

5.1. Extrinsic rewards and cognitive control functions

A set of studies has used a combined behavioral and imaging approach to test whether extrinsic rewards and higher stakes help or hinder adolescents' control (e.g. by upregulating motivation and control or choking under pressure respectively). That is, studies have examined the effect of adding incentives to performance and neural activity during response inhibition tasks such as the anti-saccade task (Luna et al., 2013). In this type of tasks, it is generally observed that children and adolescents commit more errors (pointing to immute response inhibition) than adults. In a first study (Geier et al., 2010) adolescents (13–17 years), but not adults (18–30 years) showed an increase in performance when monetary incentives were offered. Relative to adults, adolescents additionally demonstrated reduced ventral striatum activity during cue assessment, but enhanced activity while preparing an action for rewarded compared to neutral trials. Similar findings were reported in a second study (Padmanabhan et al., 2011): when monetary rewards were offered for correct performance, adult (18–25 years) performance remained high and improved in children (8–13 years) and adolescents (13–17 years) compared to neutral trials. Moreover, it was observed that only adolescents showed increased responses in the parietal cortex and striatum when comparing reward trials with neutral trials. These findings suggest that the upregulation of activation in these neural systems may be a mechanism for how extrinsic rewards may improve behavioral control particularly in adolescence.

A cross-sectional study using a different type of cognitive task (Teslovich et al., 2014) also found evidence for a facilitating effect of rewards specifically for adolescents' cognitive control. In this task adults (21–30 years) and adolescents (11–21 years) were instructed to detect the direction of movement of a cloud of moving dots. Correct trials were rewarded with either a small or large reward (one or five 'points'), whereas incorrect trials resulted in no points. Response times were longer in adolescents when large rewards were at stake, suggesting an upregulation of control in a high-stakes setting. Additionally, frontal and parietal areas—brain regions typically associated with executing cognitive control—were activated more in adolescents compared to adults for high rewards. Also, these regions showed increased functional connectivity with the ventral striatum when large compared to small rewards were at stake. An interesting next step would be to relate this frontal-parietal activation to the specific inhibitory decision process of waiting for a larger reward.

Finally, a recent longitudinal study using a similar design (10–22 years) observed that the effect of incentives on performance was susceptible to large individual differences: for some individuals, incentives (both rewards and losses) improved performance, whereas for others, it decreased performance, which may have leveled out the general effects of incentives on performance (Paulsen et al., 2015). In the absence of incentives (neutral condition) the authors observed that ventral striatal activation was associated with better response inhibition in younger participants, which was interpreted as an increased activation of reward-related circuitry to intrinsic rewards, which in turn may enhance cognitive control. Interestingly, this relation reversed (i.e., hindered response inhibition) in adulthood.

5.2. Intrinsic rewards

Brain regions involved in processing of extrinsic rewards such as money are also consistently activated during more intrinsic rewards (e.g., 'being right'), and to responses to correct or incorrect performance feedback in cognitive rule-learning tasks (Aron et al., 2004; Daniel and Pollmann, 2010). A study of Satterthwaite et al. (2012) tested whether core reward-regions were activated for correct responses even in the absence of any feedback. In a standard n-back working-memory task (N = 304; ages 8–22 years) ventral striatum activity was greater after correct compared to incorrect responses, scaled with task-difficulty, and was correlated with individual's task performance. Interestingly, the magnitude of ventral striatum activation peaked in mid-adolescents, which fits well with studies of neural activation to monetary rewards that used a similar age range (Braams et al., 2015) and may suggest that adolescence is a period of not only heightened external, but also heightened intrinsic reward-sensitivity (i.e., being right, or the inner drive to perform well). Possibly, the effect of reward responsiveness may follow an inverted U pattern, such that some level of external or internal reward may boost motivation towards better cognitive performance (Satterthwaite et al., 2012), whereas too much focus on reward may result in performance decrements (choking), thereby hindering performance.

5.3. Social influence on cognitive performance

Several studies have focused on the effect of social context on cognitive performance in adolescence. A positive social context is rewarding for humans (Steinberg, 2008), but this may be especially so in adolescence (Blakemore and Mills, 2014). An interesting question to address is how social context influences cognitive performance and learning. One way to investigate the influence of peer-presence on adolescents’ cognitive performance and learning is by studying the so-called audience effect, i.e. the change in cognitive performance when an observer is present. In adults, most studies have shown an increase in performance with an observer for simple tasks (a helping effect) but a decrease in performance for complex tasks (for a meta-analysis, see Bond and Titus, 1983). A study in children (Kim et al., 2005) tested the effects of an observer (a friend) during an inhibitory go-nogo
6. Towards a model-based approach

The reviewed studies suggest an important link between neural reward-sensitivity and behavioral outcomes such as risk taking and cognitive control. These findings fit well with neuropsychological models, in which motivationally-salient events such as rewards or social contexts may increase activation or connectivity of the affective-motivational system. However, typically these models do not further specify the mechanisms underlying the observed change in neural responses, and predict how this would link to specific behavioral processes. Applying quantitative, model-based analyses has proven to be highly useful in delineating more precise mechanisms underlying developmental shifts in learning and decision-making. Not only does this help in theorizing and interpretation of the changes in reward processing across adolescence, it also moves away from purely descriptive differences between age groups. For illustration, we describe two prominent model-based examples that specify processes of value-based decision making and outcome-based learning.

First, expectation models are classes of models that focus on the process of decision-making, and what computations may underlie the evaluation of certain choice options (e.g., Pachur et al., 2013). A first step in these models is the translation of objective choice attributes (probabilities, gain amounts, loss amounts) into subjective representations. Then, after computing the subjective value of available options, the option with the highest subjective value is chosen. Applying such a model-based decomposition of decision parameters to adolescents’ motivated decision-making could help us to understand why we observe heightened risk taking in some adolescents. Studies have started to use this method in developmental studies, with promising results. For instance, a recent study tested to what extent advice from an adult expert influences risky decision making in early adolescents (ages 12–14), late adolescents (ages 15–17), and adults (ages 18–45) by use of a formal model-based approach (Engelmann et al., 2012). Results showed that advice influenced particularly the weighting of probabilities in adolescence. Also, risk-averse advice increased the correlation strength between activity in the lateral PFC and valuation of safe choices, but only in adolescents. Other studies have decomposed adolescents’ choice behavior on objective expected values (Barkley-Levenson and Galván, 2015; van Duijvenvoorde et al., 2015) and risk (van Duijvenvoorde et al., 2015). The latter study found that activation in the insular and prefrontal cortex tracked trial-to-trial variations in risk, which was exaggerated in adolescents compared to children and adults. These studies are examples of how such models may be used to decompose and test developmental changes in adolescents’ decision-making.

Second, a widely used class of models focuses on learning, and particularly how outcome evaluation influences our subsequent behavior and predictions (e.g., reinforcement-learning models). That is, when decision outcomes do not match expectations formed on the basis of previous experience (e.g. trials), they trigger a learning signal that is referred to as a prediction error. Rewards that are better than predicted generate positive prediction errors and lead to response acquisition, whereas worse rewards generate negative prediction errors and lead to extinction of behavior. When the prediction error becomes zero, no further learning occurs and the prediction remains stable. The extent to which a prediction error alters subsequent subjective valuation of choice options depends on one’s learning rate. High learning rates give heavy weighting to recent outcome, whereas lower learning rates lead to more integration over a longer feedback history.

Adolescents have shown heightened prediction error signals compared to adults to both positive (Cohen et al., 2010) and negative outcomes (Hauser et al., 2015) in a reward network including the striatum and insula. More recent studies suggest that developmental differences in response to rewards may not be due to changes in learning signals, but how these learning signals drive subsequent behavior. That is, learning-rates were related to changes in functional connectivity between the striatum and medial PFC (van den Bos et al., 2012). It has been suggested that these findings indicate a greater influence of positive and negative outcomes in adolescents’ motivated behavior that are dependent on the interaction between subcortical and cortical neural systems (Hartley and Somerville, 2015).

Taken together, the use of such models in cognitive neuroscience moves imaging research away from testing what changes, towards how these changes occur. Although it is important to critically evaluate the use of computational models within and across different age groups (e.g., Nassar and Frank, 2016), such approaches may have several advantages. First, as stated, they allow decomposing behaviors into its underlying processes, thereby increasing our understanding what drives adolescents behavior (e.g., van Duijvenvoorde et al., in press). Second, they may better explain adolescents’ behavior on task and context variations. Third, the prediction of individual differences will be increased if we can include the underlying mechanisms of the observed processes (Pachur et al., 2013). Based on the potential of such computational models we expect that these methods will be increasingly applied in future developmental studies.

7. Strengths and weaknesses of neuroscience research

We started out by describing the long-standing interest in motivational changes in adolescence, and how neuroscience methods provided us with new perspectives on long-debated questions. What have we learned from neuroscience that may help to refine developmental theories of adolescent motivation? One of the great advantages of neuroscience studies is the level of detail of the measurement tools. By examining structural and functional changes in...
Fig. 4. An overview of the definition of childhood, adolescence and adulthood, in the studies that are included in the meta-analysis of Silverman et al. (2015; exceptions are made for studies that are included in the meta-analysis, but do not report age ranges), and studies that are discussed in this review. Light blue represents children (as defined in the studies). Blue represents adolescence (as defined in the studies), and dark blue represents adults (as defined in the studies). Adult age groups are represented up to age 30 years for illustrative purposes. Studies that have tested age as a continuous measure are displayed in black. The graph shows that there are large differences between studies in how adolescence is defined in ages in years. Moreover, there is overlap between studies in the ages that refer to as children and adolescents. What can be concluded from this figure is that there is a great need for more detailed measurement of adolescence in terms of age in years, but also in terms of pubertal development.
brain networks, it is possible to examine growth trajectories not only at the level of behavior, but also at the level of underlying neural architecture that supports behavior. As we highlighted, one exciting opportunity is to further integrate behavioral models and neural measures. That is, models of decision-making and learning can be combined with neuroscience measures to detect and interpret specific neural signals. As such, these levels of analyses provide new insights in high-stake questions, such as what drives sensation seeking and risk-taking in adolescence.

However, the progress in understanding adolescent changes in motivation lies not only at the level of combining neuroscience and behavioral methods. One of the ways neuroscience studies can be improved is by having a better conceptualization of what adolescence is, and by making use of predictions based on developmental theory. There is a surprising lack of consistency in the neuroscience literature in the selection of age ranges that are believed to encompass adolescence (see Fig. 4), with many being unable to test adolescent-specific developmental trajectories (e.g. including pre- and post-adolescent ages). This may result in an inability to test a transitional framework, focusing on changes in and out of adolescence, and may result in inconsistent findings (e.g. Bjork and Pardini, 2015). In addition, the focus has been solely on defining (calendar) age with surprisingly little measurements of maturity. For example, Cohn and Westenberg (2004) suggested that age by itself is a poor proxy of development. These authors argue that defining adolescence as stages of development and recognizing individual differences in timing of transitions between these stages, is much a more fruitful way of understanding adolescence. There is some progress in the neuroscience literature in terms of defining biological maturity, with a focus on pubertal development in several recent studies (Braams et al., 2015; Op de Macks et al., 2011; Peper and Dahl, 2013; van Duijvenvoorde et al., 2014), which we also highlighted in the current review, but there is no emphasis yet on defining social maturity. Thus, this is a level of analysis where neuroscience can benefit from developmental approaches.

A further step forward that is established in developmental psychology, but not yet in neuroscience literature, is the value of longitudinal designs. These designs, with preferably more than three time points when interested in U-shaped developmental patterns (Crone and Elzinga, 2014), have the advantage of allowing for the fit of growth models within individuals, and allow for a testing of changes over time (for recent examples, see Braams et al., 2015; Ordaz et al., 2013; Peters et al., 2016a). In addition, longitudinal designs allow for prediction models, which make it possible to test whether one process can predict change in another process over time. For example, we recently showed that brain connectivity was predictive of changes in impulsivity control (Achterberg et al., 2016) and alcohol use (Peters et al., in press) two years later, while controlling for first time point measurements. Another important benefit of longitudinal methods is that it allows for a more detailed test of when developmental goes astray. This is especially important in adolescence, given that most of the psychiatric disorders related to mood and motivation (such as depression, social anxiety and substance abuse) emerge for the first time in adolescence (Giedd et al., 2008; Lee et al., 2014).

8. Conclusion

In this review we aimed to integrate current findings on adolescents’ motivated behavior, and specifically adolescents' reward-sensitivity by using a brain-behavioral approach. The reviewed studies generally support a heightened reward response in adolescence, predominantly observed in key reward-regions such as the ventral striatum and OFC, which most likely rely on an interconnected network of subcortical and cortical reward-related regions, including the amygdala, insula and lateral PFC. Studying subcortical and subcortical-cortical connectivity is an increasingly used tool and has the potential to study the interactive development of these networks and the relation to motivated behavior.

Findings indicate that adolescents’ reward-sensitivity may lead to increased sensation seeking, risk taking, and hindering effects on cognitive performance. In other contexts, however, heightened reward-sensitivity may boost cognitive control resources, and promote instrumental or prosocial behavior (see also Telzer, 2016). Thus, an important question for future research is which of these environmental contexts are more likely to trigger which behavioral outcome, and for which adolescents these influences are most prominent. For instance, one could hypothesize that individual differences in sensitivity to environmental influences, such as a reactive temperament, may be particularly important at the onset of adolescence during which affective-motivational changes create a window that amplifies behavior that may lead to negative or positive developmental trajectories. Multiple encounters with health-endangering behaviors, or negative peer-influences, may propel some adolescents more easily towards risky behavior with possible negative individual and societal outcomes.

A similar developmental trajectory might be proposed for positive developmental outcomes. That is, if certain experiences, such as cooperation, sharing and helping, are experienced as relatively more rewarding for some adolescents (adolescents with a reactive temperament), then multiple encounters of these experiences may set the stage for a trajectory in which adolescents feel more committed to these prosocial goals also when developing into adulthood. This hypothesis predicts that those adolescents who show highest emotional reactivity to rewards in early adolescence also show the largest benefit of prosocial experiences (Crone and Dahl, 2012).

The heightened reward-sensitivity in adolescence—and its influence on motivated behavior—may be highly relevant for research in clinical populations, such as children that have behavioral problems, antisocial development or attention-deficit hyperactivity disorder (ADHD). A recent meta-analysis showed that reinforcement potentials normalize inhibitory control in children and adolescents with ADHD to baseline levels of healthy controls (Ma et al., 2016). Further research on the behavioral effects of reward-sensitivity should therefore take into account individual differences and subgroups (e.g., Fair et al., 2012).

In this review, we demonstrated that advancements in cognitive neuroscience methods allow for an additional level of explanation to understand motivational changes in adolescence. Nonetheless, further breakthroughs arise when research is more specific in terms of combining paradigms with behavioral models. Specificity is also warranted in the definition of adolescence, including understanding transition periods in adolescence, such as the onset of puberty and the changes in social maturity. An integrative bio-psychological perspective has great potential for bridging levels of explanation from basic neural measures to behavioral outcomes, to applications in the classroom and clinical interventions. The described trajectories propose adolescence as a crucial period for exploration and social learning, and underline that this is a high-stake period of interventions. Ultimately, these insights will lead to a better understanding towards the needs of youth and fostering opportunities for the new generation.

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