MOTIVATION AND COGNITIVE CONTROL

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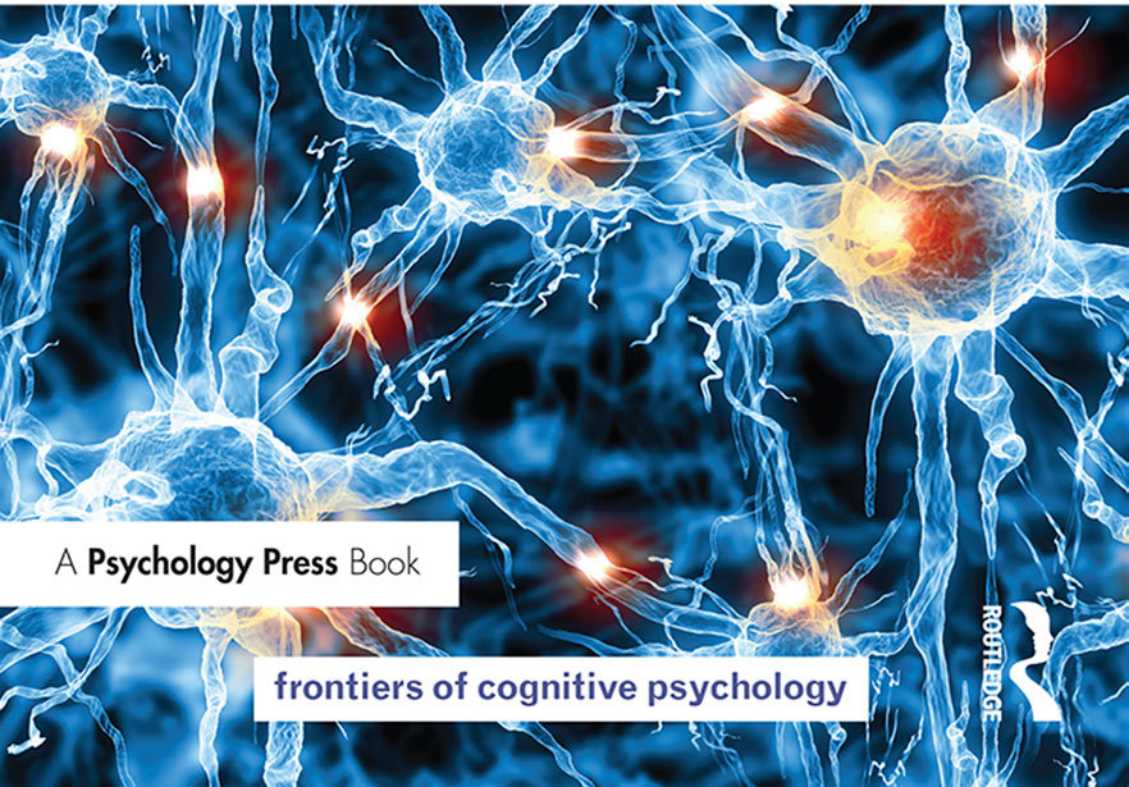

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LIFESPAN DEVELOPMENT OF ADAPTIVE NEUROCOGNITIVE REPRESENTATIONS

Reciprocal Interactions Between Cognition and Motivation

Shu-Chen Li and Ben Eppinger

Introduction

An individual's development across the life span entails co-constructive interactions between environmental, cultural, and socioemotional influences from the developmental context and the individual's neurobiological inheritance. Crucially, individuals are not just mere passive recipients of their ecological, cultural, and neurobiological endowments; rather, they are active agents who make decisions and take actions to shape their own development. In order to better capture interactive dynamics between (a) the resources for and constraints on individual ontogeny that arise from the development context, (b) the mechanisms of brain maturation and senescence, and (c) the individual's self-regulatory behavior, a synergistic conceptualization of development is to view an individual's development across the life span as the development of self-regulated adaptive neurocognitive dynamics (Li, 2003, 2013) that are "embodied" in motor, sensory, and perceptual processes and "situated" in social and environmental contexts (cf. Clark, 2001; Robbins & Aydede, 2008). Viewed through the lens of self-regulated developmental adaptations of neurocognitive processes, brain circuitries and mechanisms that afford seamless interactions between motivational regulation and cognitive control are main themes for lifespan developmental neuroscience research.

Earlier behavioral research has tackled the interactions between motivation, self-regulation, and cognition in the contexts of achievement motivation (e.g., Bandura, 1977; Brandtstädter, 1989; Gollwitzer & Moskowitz, 1996; Weiner, 1985; White, 1959; Wigfield & Eccles, 2000) and successful aging (Baltes, 1997; Baltes & Baltes, 1990; Carstensen, Isaacowitz, & Charles, 1999; Freund, 2008; Heckhausen, Wrosch, & Schulz, 2010; Hess, 2014). To zoom in on neurocognitive processes of self-regulated developmental adaptations, the current review focuses specifically on the relations between neuromodulation of frontal-hippocampal-striatal circuitry and interactions between cognition and motivation across the life span.

Reciprocal Cognition–Motivation Interactions via Dopamine Modulation of the Frontal-Striatal-Hippocampal Systems

Being self-organizing systems, brain processes dynamically adapt during the course of mental operations and behavioral actions to optimize the levels of matches and mismatches between environmental states, action outcomes, and internal states (e.g., see a theoretical account in Friston & Kiebel, 2009). One of the striking features of the brain is that neurons contain and release a large number of neurotransmitters, which play important roles in regulating signal transmissions between neurons (see Vizi & Lajtha, 2008, for overviews). Several transmitter systems, such as the catecholamines (dopamine, serotonin, and norepinephrine), broadly innervate various neural circuitries throughout the brain. Neuromodulatory systems have been considered as neural substrates for regulating adaptive, value-dependent selection in the brain (Friston, Tononi, Reeke, Sporns, & Edelman, 1994). Depending on situational demands and the integrity of brain functions, neurotransmitters modulate task-relevant brain circuitries, so that individuals can flexibly adapt their thoughts and actions.

Through widespread projections, neurotransmitters have pervasive effects in regulating brain dynamics in different networks. Figure 14.1 illustrates the major pathways of the dopaminergic systems. The vast majority of dopaminergic neurons are found in the midbrain (mesencephalon), particularly in the substantial nigra pars compacta (SNc) and the ventral tegmental area (VTA). Originating from the SNc and VTA, the DA neurons widely innervate the frontal-striatal-hippocampal circuitries through three main pathways: (1) the nigrostriatal pathway with fibers of DA neurons projecting from the SNc to the caudate and putamen in the dorsal striatum, (2) the mesolimbic pathway projecting from the VTA primarily into the nucleus accumbens (NAcc) in the ventral striatum but also to the hippocampus and amygdala, and (3) the mesocortical pathway projecting from the VTA to the frontal, cingulate, and perirhinal cortex (see Figure 14.1; cf. Bäckman & Farde, 2005; Chinta & Andersen, 2005; Sánchez-González, García-Cabezas, Rico, & Cavada, 2005).

Through these different pathways dopamine has been shown to affect cognitive control as well as motivational functions. On the one hand, pharmacologically manipulating the efficacy of dopamine signaling (e.g., Nagano-Saito et al., 2008; Wallace, Vytlacil, Nomura, Gibbs, & D'Esposito, 2011) as well as individual differences in the integrity of the fronto-striatal-thalamic white matter tracts have been shown to affect the functional connectivity between the prefrontal cortex (PFC) and the striatum during tasks that require cognitive control. On the other hand, the roles of midbrain dopamine neurons in modulating and adapting motivation relevant processes (e.g., reward anticipation, signaling prediction-outcome discrepancy or novelty, establishing associations between rewards, as well as the extent of effort invested in obtaining rewards) have been established in animal and human research (Düzel, Bunzeck, Guitart-Masip, & Düzel, 2010; McClure, Daw, & Montague, 2003; Montague, Hyman, & Cohen, 2004; Niv, Daw, Joel, & Dayan, 2007; Schultz, Dayan, & Montague, 1997; Tobler, Fiorillo, & Schultz, 2005; see Kurniawan, Guitart-Masip, & Dolan, 2011; Schultz, 2013, for recent reviews). At a more general

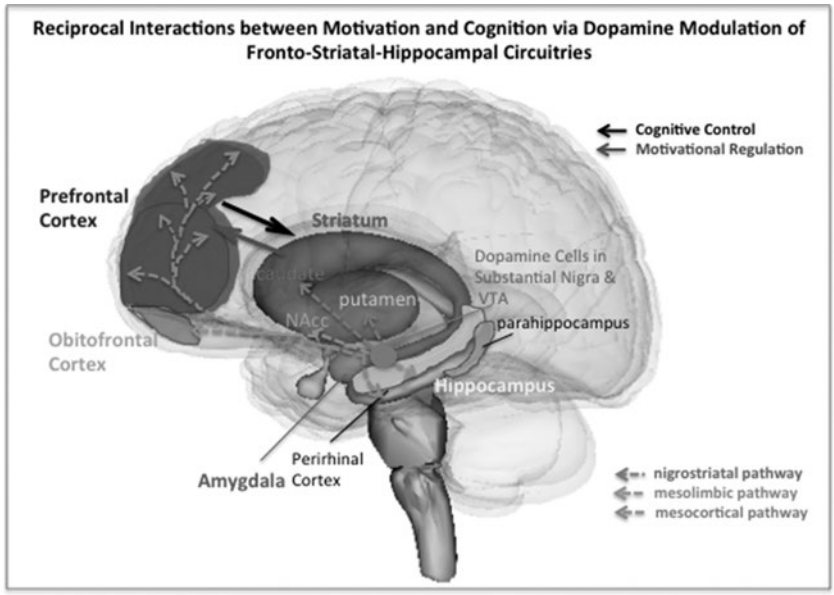

FIGURE 14.1 Schematic diagram of major dopamine pathways in the fronto-striatal-hippocampal circuitries modulating reciprocal interactions between cognitive control and motivational regulation. Image modified after an anatomography of the striatum (http://sv.wikipedia.org/wiki/Striatum#mediaviewer/Fil:Striatum.png) generated by Life Science Database (licensed under CC-BY-SA-2.1-jp).

level, a recent study showed that dopamine also signals unrewarded stimuli if these were presented in previously rewarded contexts, suggesting that midbrain dopamine is sensitive to higher-order context generalization (Kobayashi & Schultz, 2014). Taken together, through the three key pathways the dopamine modulation is at the interface for modulating (i) cortical cognitive processes, such as working memory, attention, and performance monitoring (e.g., Cools, Clark, & Robbins, 2004; D'Ardenne et al., 2012; Hämmerer et al., 2013; Ito, Stuphorn, Brown, & Schall, 2003; Jocham & Ullsperger, 2009; Krämer et al., 2007; Landau, Lal, O'Neil, Baker, & Jagust, 2009; Li et al., 2013; McNab et al., 2009; Montague et al., 2004), (ii) subcortical motivational processes mediated by reward or affective states (for reviews see Berridge & Robinson, 2003; Schultz, 2013; Shohamy & Adcock, 2010; Volkow, Wang, & Baler, 2011), and (iii) the reciprocal interactions between cognition and motivation (for review see Aarts, van Holstein, & Cools, 2011).

Maturation and Senescence of Dopaminergic Modulation

Evidence for the maturation of the different dopamine pathways during child and adolescent development is still scarce, due to practical limitations of applying invasive methods, such as positron emission tomography (PET) receptor imaging, in

these age groups. Nonetheless, comparisons of dopamine functions across the life span that are based on results from animal studies or from human postmortem studies indicate substantial maturational and senescence-related changes in dopamine modulation. For instance, the evidence from a postmortem study shows that the activity of an enzyme that regulates extracellular dopamine levels in the prefrontal cortex (i.e., the catechol-*o*-methyltransferase enzyme) increases about twofold from neonate to adulthood, and declines slightly afterwards (Tunbridge et al., 2007; see Figure 14.2A). Evidence from animal studies also suggests that the efficacies of both the subcortical and cortical dopamine systems (e.g., the density of different receptor types) increase continuously and steadily during the postnatal period. Furthermore, the subcortical dopamine system reaches its peak already in peri-adolescence, whereas the development of the cortical system is slower and reaches its peak level only in early adulthood. Specifically, Tarazi and Baldessarini (2000) found that the density of dopamine D1, D2, and D4 receptors in the caudate and nucleus accumbens of rats increased to a peak at about postnatal day 28 (approximately equivalent to late childhood and peri-adolescence in humans), but then declined significantly (from postnatal 35 to 60 days) to adult levels. Interestingly, the density of these three receptor types in the prefrontal cortex, however, rose steadily throughout the childhood and adolescence and reached a maximum of the adult level only around 60 days postnatally. A recent PET study with a small sample of adolescents and younger adults provided rare human in vivo data and showed that dopamine D1 binding potential in the prefrontal cortex continued to show age-dependent differences until adulthood, whereas no such age-dependent differences were observed in the dorsal or ventral striatum (Jucaite, Forssberg, Karlsson, Halldin, & Farde, 2010). The more protracted maturation of the cortical dopamine system has been linked to the protracted development of attention and other frontal executive functions during childhood and adolescence (e.g., Diamond, Briand, Fossella, & Gehlbach, 2004; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005). Moreover, the lead-lagged pattern in the maturational trajectories of the subcortical and cortical dopamine systems in adolescence (Figure 14.2D) parallels other evidence for more protracted cortical relative to subcortical brain maturation in terms of structures (e.g., Giedd et al., 1996; Sowell et al., 2004) and functions (e.g., Crone & van der Molen, 2004; Galván, Hare, Voss, Glover, & Casey, 2007; Somerville & Casey, 2010). Together the sensitivity of the subcortical systems to motivational influences and the still developing cortical control and regulatory functions may render the adolescence period particularly malleable by positive or negative motivational influences (Andersen, 2003; Casey, Jones, & Hare, 2008; Crews, He, & Hodge, 2007; Crone & Dahl, 2012; Li, 2013; Luciana, Wahlstrom, Porter, & Collins, 2012; Sommerville & Casey, 2010).

As for senescence of dopaminergic modulation, there is the consensus that various aspects of the dopamine systems decline during the course of typical aging. For instance, in vivo PET receptor imaging studies in healthy older adults show extensive evidence for gradual but pervasive declines in the binding potential of presynaptic striatal dopamine transporter (e.g., Erixon-Lindroth et al., 2005; see

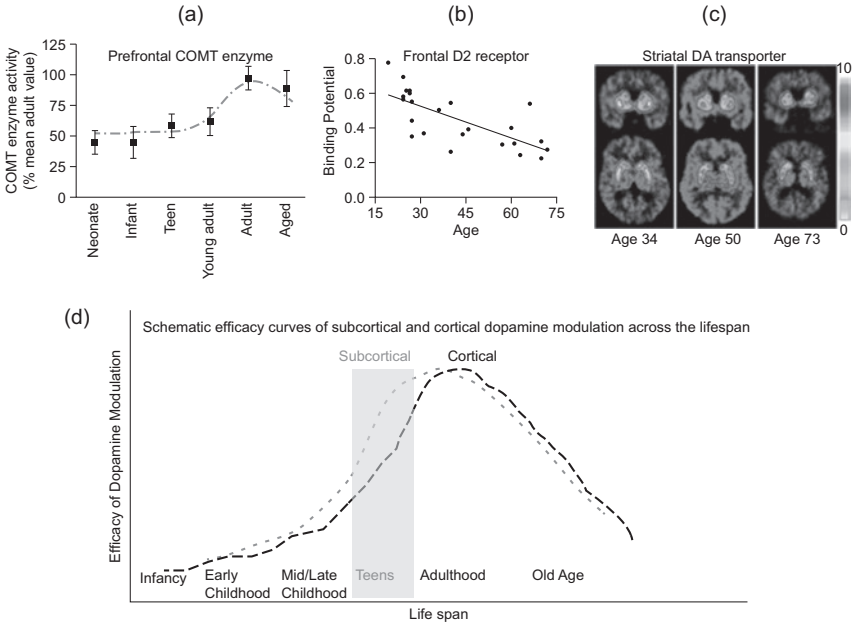

FIGURE 14.2 Efficacy of different aspects of the dopamine systems as a function of age. (A) COMT enzyme activity across the life span assessed in postmortem tissues. Adapted from "Catechol-o-Methyltransferase Enzyme Activity and Protein Expression in Human Prefrontal Cortex across the Postnatal Lifespan," by E. M. Tunbridge et al., 2007, *Cerebral Cortex, 17*, pp. 1206–1212. Copyright 2007 by *Cerebral Cortex*. (B) Adult age differences in frontal dopamine D2 receptor binding potential. Adapted from "Age-Related Dopamine D2/D3 Receptor Loss in Extrastriatal Regions of the Human Brain," by V. Kaasinen et al., 2000, *Neurobiology of Aging*, *21,* pp. 683–688. Copyright 2000 by Elsevier. (C) Adult age differences in striatal dopamine transporter binding potential. (D) Schematic age gradients for the levels of subcortical and cortical dopamine functions across the lifespan. Adapted from "The Role of the Striatal Dopamine Transporter in Cognitive Aging," by N. Erixon-Lindroth et al., 2005, *Psychiatry Research Neuroimaging*, *138*, pp. 1–12. Copyright 2005 by Elsevier.

Figure 14.2C) and frontal postsynaptic dopamine D2 receptor (e.g., Kaasinen et al., 2000; see Figure 14.2B). Cross-sectional estimates from different studies showed that in various extrastriatal and striatal regions the estimated densities of dopamine receptors decline about 10% per decade, starting around the beginning of the third decade of life (e.g., Inoue et al., 2001; Kaasinen et al., 2000; for reviews see Bäckman, Nyberg, Lindenberger, Li, & Farde, 2006; Li & Rieckmann, 2014). Computational studies have related aging-related declines in dopamine modulation to aging-related deficits in increased neuronal noise and reaction time fluctuations as well as attenuated episodic memory, working memory, and cognitive control (Li, Lindenberger, & Sikström, 2001; Li, Naveh-Benjamin, & Lindenberger, 2005; Li & Sikström, 2002; see Li & Rieckmann, 2014 for a recent review).

Since dopamine dynamically modulates the frontal-striatal-hippocampal circuitry through the three major dopaminergic pathways (Figure 14.1), the maturation and senescence of its modulatory efficacy are likely to have direct implications for the development of cognition, motivation, and their interactions. In the following three sections, we specifically highlight recent developmental and aging studies on (a) self-regulatory control of reward processing, (b) reward modulation of attention and memory, and (c) lifespan age differences in habitual and goal-directed regulation of actions in tasks that require more complex learning or decision processes.

Lifespan Development of Performance Monitoring Mechanisms

The development (e.g., Bunge & Wright, 2007; Munakata, Snyder, & Chatham, 2012) and aging (e.g., Buckner, 2004; Sander, Lindenberger, & Werkle-Bergner, 2012; Störmer, Passow, Biesenack, & Li, 2012; Yuan & Raz, 2014) of frontal processes underlying working memory and cognitive control have been the key themes of developmental and aging cognitive neuroscience. The focus of this section is on lifespan development of performance monitoring mechanisms that engage striatal dopamine modulation of the fronto-striatal interactions (for earlier theories of error and conflict monitoring see Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004).

On the one hand, recent evidence indicates that via the meso-cortical pathway striatal dopamine is involved in modulating prefrontal cognitive control functions (see Cools, 2011, for recent review), such as context updating (e.g., D'Ardenne et al., 2012), working memory (e.g., Landau et al., 2009), and outcome monitoring (e.g., McClure et al., 2003). On the other hand, the prefrontal circuitry also regulates striatal inputs, as shown in studies applying transcranial magnetic stimulation (TMS) over the frontal cortex to affect task-related striatal activity (e.g., van Schouwenburg, O'Shea, Mars, Rushworth, & Cools, 2012) and dopamine release in the caudate nucleus (Strafella, Paus, Barrett, & Dagher, 2001).

Age Differences in Psychophysiological Markers of Feedback-Related Processing

In psychophysiological studies of probabilistic reinforcement learning or stimulus-response conflict monitoring, the amplitude of the feedback-related negativity (FRN)—a negative deflection in event-related potentials following feedbacks—is usually larger after negative than positive feedback, such as monetary gains or losses and performance feedbacks (e.g., M. Cohen, Wilmes, & van der Vijver, 2011, for review). Studies using simultaneous EEG with functional brain imaging recordings (e.g., Hauser et al., 2015) and other source localization methods usually indicate that the medial part of the frontal cortex, particularly the anterior cingulate cortex, is the source of the FRN (Gehring & Willoughby, 2002; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). A recent neurophysiological study with younger adults showed that the differentiability of FRN after monetary gains and losses was larger

in individuals carrying the 9-repeat allele of the dopamine transporter gene (DAT) and in individuals carrying the short allele (s) of the serotonin transporter gene (5HTTLPR), who presumably are associated with higher striatal dopamine and serotonin levels (Heitland et al., 2012). There is also evidence that manipulating dopamine pharmacologically affects the amplitude of FRN (Santesso et al., 2009). Of note is a recent study investigating the effects of a genotype relevant for striatal dopamine receptor functions on FRN in a lifespan sample (Hämmerer et al., 2013). The results showed that individuals carrying the genotype associated with higher dopamine receptor efficacy (i.e., A allele homozygotes of a single nucleotide polymorphism [rs907094] of the *PPP1R1B* gene) showed larger feedback-related brain evoked potentials. Moreover, this effect was more pronounced in children and older adults in comparison to adolescents and younger adults (see Figure 14.3). This

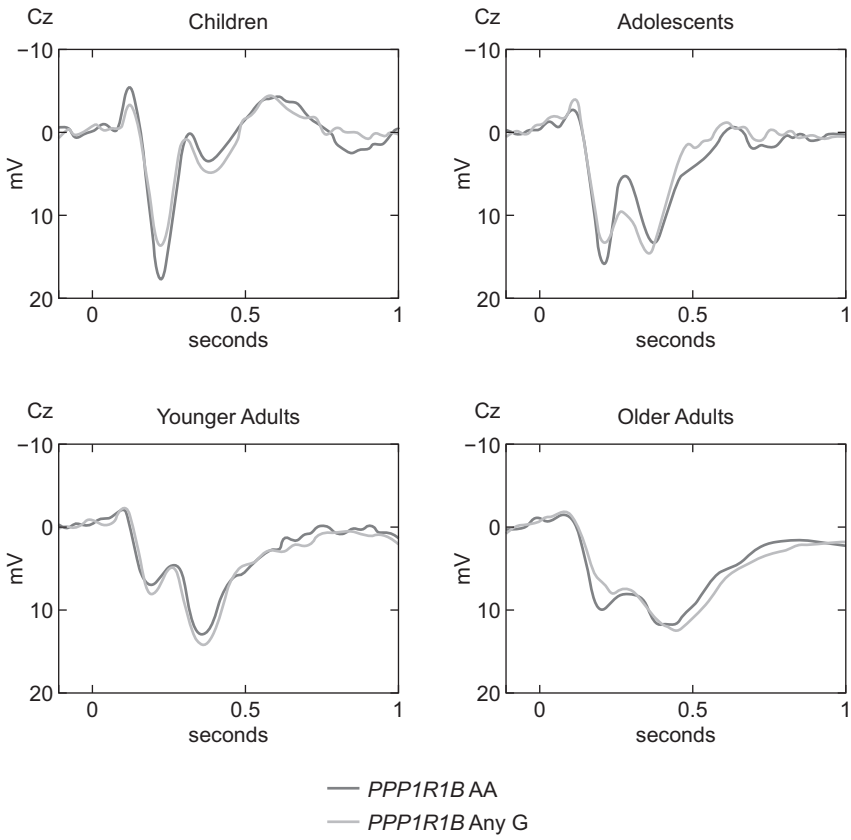

FIGURE 14.3 Lifespan age differences in genotype (PPP1R1B, also known as DARPP-32) effects on feedback-related negativity after losses during probabilistic reinforcement learning. Adapted from "Effects of *PPP1R1B* (*DARPP-32*) Polymorphism on Feedback-Related Brain Potentials across the Lifespan," by D. Hämmerer et al., 2013, *Frontiers in Psychology, 4,* 1–8. Copyright 2013 by Frontiers in Psychology.

finding, together with earlier results of interactions between age and genotype effects, indicates that genotype effects on neurocognitive phenotypes may vary as a function of brain maturation and aging (Li et al., 2013; Lindenberger et al., 2008; Papenberg et al., 2014).

Lifespan Development of Motivational Regulation of Attention and Memory

Besides frontal processes of monitoring action outcomes, studies on dopamine modulation of reward processing have also generated new research on motivational regulation of memory and attention. Through the mesolimbic and mesocortical pathways, reward-elicited dopamine release may enhance hippocampal memory (see Shohamy & Adcock, 2010, for review), frontal-parietal attentional (e.g., Krebs, Boehler, Appelbaum, & Woldorff, 2013) and working memory processes (Kennerley & Wallis, 2009; Krawczyk, Gazzaley, & D'Esposito, 2007).

Dopamine neurons in the VTA also innervate the hippocampus and the surrounding medial temporal lobe regions. Animal studies indicate that dopamine modulates long-term potentiation of hippocampal neurons, which is important for learning and memory (Lisman, Grace, & Duzel, 2011). In young adults, brain activity in the mesolimbic circuitry during reward anticipation has been shown to be related to hippocampal activity and enhance episodic memory (Adcock, Thangavel, Whitfield-Gabrieli, Knutson, & Gabrieli, 2006; Shohamy & Wagner, 2008; Wittmann et al., 2005; Wittmann, Schiltz, Boehler, & Düzel, 2008; Wolosin, Zeithamova, & Preston, 2012).

Lifespan Differences in Reward Modulation of Memory

Thus far, lifespan developmental studies on reward enhancement of memory have mostly focused on younger and older adult samples. Studies using the value-directed remembering paradigm (i.e., to-be-remembered memory items are associated with specific values indicating the points that could be earned by remembering the items) showed that both younger and older adults allocated more study items for high-value items, with older adults showing a stronger tendency in this regard such that aging-related impairments in delayed memory recall were reduced for high-value items (Castel, Murayama, Friedman, McGillivray, & Link, 2013). Extant behavioral evidence seems to indicate that, declined mesolimbic dopaminergic modulation notwithstanding, episodic memory in older adults can still benefit from motivational regulation. In terms of recognition confidence, however, higher values of the to-be-remembered items did not enhance older adults' confidence for correctly recalled targets as it did for the younger adults (Spaniol, Schain, & Bowen, 2013). One possibility for the lack of reward effects on recognition confidence may be related to the high confidence errors that older

adults typically exhibit in recognition memory paradigms (e.g., Dodson, Bawa, & Krueger, 2007; Shing, Werkle-Bergner, Li, & Lindenberger, 2009). Older adults' reduced sensitivity of memory confidence to reward as observed in the value-directed remember paradigm (Spaniol et al., 2013) might reflect less distinctive memory representations, which have been attributed to aging-related dopaminergic decline (Li et al., 2005). Indeed, a recent pharmacological imaging study showed that a pharmacological dopamine agonist (levodopa) enhanced episodic memory and brain activation in older adults (Chowdhury, Guitart-Masip, Bunzeck, Dolan, & Düzel, 2012).

Of note, age differences in value-directed remembering across the life span have been investigated in a cross-sectional study covering the age range from 5 to 96 years. Across the life span, an inverted U-function captures memory performance as well as selectivity in recalling higher versus lower value memory items. Younger adults show highest value-based selectivity effects, followed by older adults, whereas adolescents and children showed less value-dependent selectivity relative to the two adult groups (Castel et al., 2011; see Figure 14.4A).

Lifespan Differences in Reward Modulation of Attention

Through the mesocortical pathway midbrain dopamine modulation of reward processing may modulate frontal-parietal working memory and attention functions. For instance, in response conflict tasks, associating stimuli with rewards was found to enhance cognitive control of conflict processing (e.g., Krebs et al., 2013; Padmala & Pessoa, 2011). There is also evidence suggesting that motivational influences can modulate attention during early visual selection in an "automatic" manner: The presence of task-irrelevant stimuli that were previously associated with high rewards slows down visual search (Anderson, Laurent, & Yantis, 2011). A recent EEG study also showed that P3 responses to targets and contingent negative variation (CNV) during rapid visual search (Hughes, Mathan, & Yeung, 2013) were both sensitive to reward manipulations. Developmental or aging studies on motivational modulation of perception or early visual attention are still scarce, with only rare exceptions. The perceptual efficacy during perceptual discrimination, as indexed by drift rates estimated by diffusion models (e.g., Ratcliff, 2002), has been shown to be sensitive to stimulus valence (both positive and negative). Moreover, negative adult age differences in perceptual efficacy were reduced for valent stimuli (Spaniol, Voss, Bowen, & Grady, 2011).

A recent lifespan study from our lab included children, adolescents, and younger and older adults (ranging from 6 to 75 years) to explore the effects of reward on visual attention; our results showed that reward speeds up visual search in all four age groups (Störmer, Eppinger, & Li, 2014). Age interacted with the effect of reward magnitude, with younger adults showing a large effect size relative to the other age groups (Figure 14.4B). Together these results are consistent with lifespan age differences in mesocortical dopaminergic modulation.

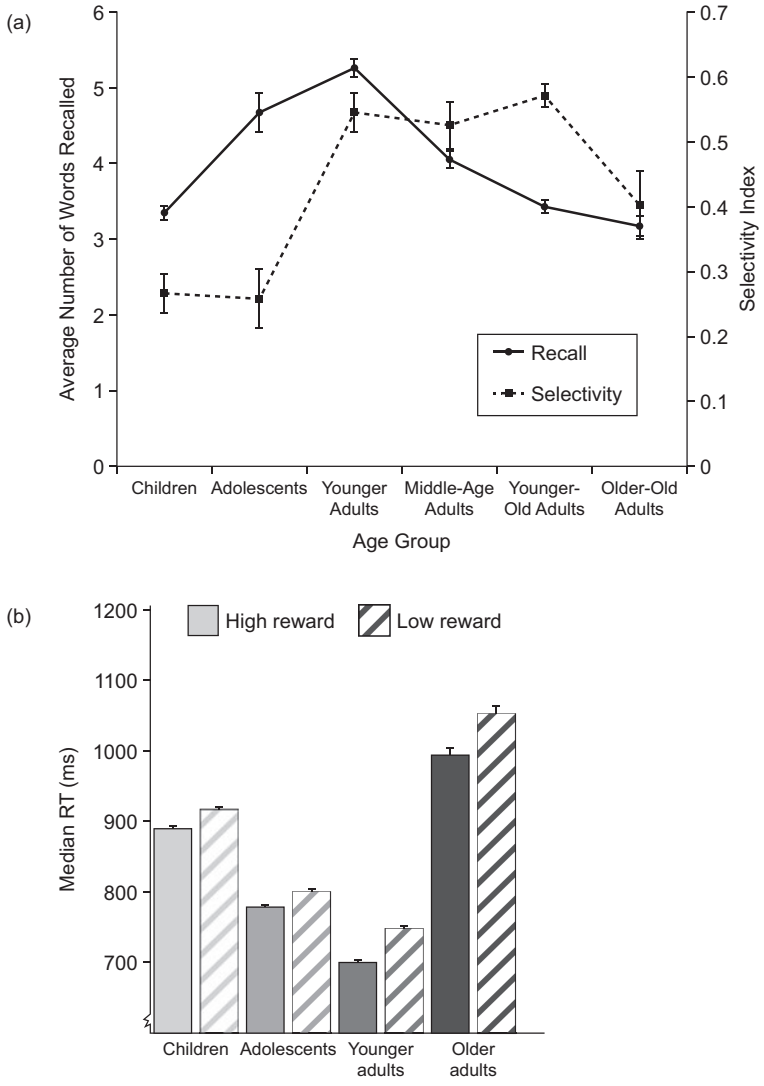

FIGURE 14.4 Lifespan age differences in reward modulation of memory and visual attention. (A) Younger adults are most selective in remembering high-value information relative to older adults, adolescents, and children. Adapted from "Selecting Valuable Information to Remember: Age-Related Differences and Similarities in Self-Regulated Learning," by A. D. Castel, K. Murayama, M. C. Friedman, S. McGillivray, and I. Link, 2013, *Psychology and Aging, 28*, pp. 232–242. Copyright 2012 by the American Psychological Association. (B) Reward speeds up visual attention across the life span, but with a larger effect in young adulthood. Adapted from "Reward Speeds Up and Increases Consistency of Visual Selective Attention: A Lifespan Comparison," by V. S. Störmer, B. Eppinger, and S.-C. Li, 2014, *Cognitive, Affective & Behavioral Neuroscience, 14*, pp. 659–671. Copyright 2014 by Psychonomics Society Inc.

Lifespan Development of Habitual and Strategic, Goal-Directed Learning and Decision Making

Other than the more basic mechanisms of attention, memory, and reward processing reviewed in the proceeding sections, goal-directed learning and decision making are more complex behaviors that entail dynamic interactions between cognition and motivation. In this part of the chapter we will primarily focus on the interplay of habitual and goal-directed decision mechanisms during childhood development and aging.

Children show a high degree of sensitivity to regularities in their environment, which allows them to build up routines or habits (Munakata, Snyder, & Chatham, 2012). The underlying experience-based learning mechanisms are a powerful and computationally efficient way of acquiring behavior (Marcovitch & Zelazo, 2009). However, as many parents (and sometimes the children themselves) painfully realize, these habits come at the cost of being very persistent and difficult to overcome when a given habit is no longer optimal in a new environment or situation. For example, children at the age of 3 years may already be able to verbally indicate a change in the sorting rule of a card-sorting task. However, they are unable to use this knowledge and perseverate in continuously using the old rule for card sorting (Marcovitch, Zelazo, & Schmuckler, 2002; Zelazo, Frye, & Rapus, 1996). With increasing age the ability to adjust behavior to rule changes improves, reaching adult levels around early adolescence (Crone, Zanolie, Van Leijenhorst, Westenberg, & Rombouts, 2008; Somsen, 2007). Thus, childhood development is characterized by an increasing flexibility in the use of internal goal-directed representations, or models of the world, to overcome habitual responding (Casey, Thomas, Davidson, Kunz, & Franzen, 2002; Marcovitch & Zelazo, 2009; Munakata et al., 2012; Snyder & Munakata, 2010).

Interestingly, as reviewed earlier, older adults show very similar performance as children deficits on tasks that require adaptive learning and decision-making processes (Hämmerer & Eppinger, 2012). Results from studies on reinforcement learning show age-related performance deficits during learning from probabilistic compared to deterministic reward (Eppinger, Kray, Mock, & Mecklinger, 2008; Hämmerer, Li, Mueller, & Lindenberger, 2011; Pietschmann, Endrass, Czerwon, & Kathmann, 2011). These deficits get even more pronounced when older adults have to flexibly adapt to changes in learned contingencies during reversal learning (Eppinger & Kray, 2011; Mell et al., 2005; Weiler, Bellebaum, & Daum, 2008). More recent findings suggest that aging may be associated with a shift from strategic, goal-directed action control to habitual learning and decision-making processes (de Wit, van de Vijver, & Ridderinkhof, 2014; Eppinger, Walter, Heekeren, & Li, 2013; Worthy, Byrne, Gorlick, & Maddox, 2014). Taken together, these findings indicate that the interplay of habitual and strategic, goal-directed learning and decision-making mechanisms changes over the life span.

In the following sections we will review and discuss these findings in relation to current neurocomputational accounts that try to capture the dynamic interactions between motivational and cognitive control processes during learning and decision making.

Habitual and Goal-Directed Processes

The dissociation between habitual and goal-directed mechanisms is at the core of many current theories of learning and decision making (Balleine & O'Doherty, 2010; Daw, Niv, & Dayan, 2005). Habitual or model-free learning reflects the acquisition of behavior based on experience (Thorndike, 1911). It can be modeled using reinforcement learning algorithms, using so-called updating rules, in which the expected value of a state (or an action) is updated by the difference (commonly termed "reward prediction error") between the expected and the received reward (Sutton & Barto, 1998). Reward prediction errors during habitual reinforcement learning have been associated with phasic signals from dopamine neurons in the ventral tegmental area and the substantia nigra (D'Ardenne, McClure, Nystrom, & Cohen, 2008; Niv, Edlund, Dayan, & O'Doherty, 2012; Schultz, Dayan, & Montague, 1997; Waelti, Dickinson, & Schultz, 2001). These teaching signals are projected to limbic and para-limbic areas, such as the ventral striatum and ventromedial PFC, where they are used to update reward predictions during learning (Jocham, Hunt, Near, & Behrens, 2012; Rudebeck, Saunders, Prescott, Chau, & Murray, 2013; Sul, Kim, Huh, Lee, & Jung, 2010). Goal-directed or model-based learning and decision making reflect choices that are guided by internal goal representations or maps (Miller & Cohen, 2001; Tolman, 1948). In contrast to model-free learning, model-based decision mechanisms are more effortful, because they involve a complete representation of the state space of a task—for instance, all the contingencies between subsequent states, actions, and rewards of a decision task (Daw, Gershman, Seymour, Dayan, & Dolan, 2011; Gershman, Markman, & Otto, 2013; Otto, Gershman, Markman, & Daw, 2013; Wilson, Takahashi, Schoenbaum, & Niv, 2014; Wunderlich, Smittenaar, & Dolan, 2012). The learning of model representations has been associated with activity in cortical structures, primarily the lateral prefrontal and parietal cortex (Gläscher, Daw, Dayan, & O'Doherty, 2010; Smittenaar, FitzGerald, Romei, Wright, & Dolan, 2013). Recent findings suggest that in addition to these two decision systems there is a set of areas involving the anterior PFC and the lateral inferior PFC that may be involved in controlling which decision system will predominantly guide behavior (Lee, Shimojo, & O'Doherty, 2014). The theoretical idea here is that the anterior PFC and lateral inferior PFC monitor the uncertainty of the predictions that are generated by the model-based and model-free decision system and use these uncertainty estimates to dynamically allocate control in favor of the more optimal, reliable course of actions (Lee et al., 2014; Yoshida & Seymour, 2014).

Lifespan Developmental Differences in Model-Free Learning and Decision Making

Current findings from research in developmental cognitive neuroscience indicate that model-free and model-based mechanisms as well as the underlying neural systems show different developmental trajectories (Marcovitch & Zelazo, 2009; Munakata et al., 2012; Somerville & Casey, 2010). Model-free (habitual) mechanisms develop comparatively early in life and are involved in the acquisition of many basic aspects of behavior, ranging from conditioning to attachment, (social) play, and food preferences (Millar, 1990; Trezza, Baarendse, & Vanderschuren, 2010; Ventura & Worobey, 2013). As reviewed in an earlier section, current evidence points to the view that the subcortical dopamine system may already be developed early in life (e.g., Haycock et al., 2003). Furthermore, there are several findings that point to nonlinearities in the development of dopaminergically innervated striatal areas involved in reward processes. Studies on reward-based learning and decision making in adolescents suggest that sensitivity reward as well as striatal BOLD (blood oxygen level–dependent) responses to prediction errors during reinforcement learning may be elevated in teenagers (age 14–19 years) compared to children (age 8–12 years) and adults (Christakou et al., 2013; J. Cohen et al., 2010; Urošević, Collins, Muetzel, Lim, & Luciana, 2012). This suggests that adolescence may represent a unique developmental period that is characterized by a hypersensitivity of the reward system in combination with a still developing prefrontal cognitive control system (Somerville & Casey, 2010; Steinberg, 2008). The neurophysiological mechanisms behind this hypersensitivity are not yet established. Findings in rodents suggest that dopamine-induced changes in the connectivity of mesocorticolimibic circuits (particularly projections to the medial PFC) may play an important role (Manitt et al., 2011, 2013). Whether these effects are primarily the consequence of hormonal changes during puberty or whether they arise through interaction with changes in the motivational and social states of individuals during adolescence is still unclear (Arnsten & Shansky, 2004).

Changes in reward processing during learning and decision making are not confined to childhood development (Eppinger, Nystrom, & Cohen, 2012). Findings from a recent meta-analytic study suggest that age-related changes in experiential decision-making tasks may result from underlying deficits in learning (Mata, Josef, Samanez-Larkin, & Hertwig, 2011; Samanez-Larkin, 2013). Consistent with this idea, recent findings from age-comparative studies on reinforcement learning show that deficits in learning from reward are associated with a reduced sensitivity of the ventral striatum and ventromedial prefrontal cortex to reward prediction errors (Eppinger, Schuck, Nystrom, & Cohen, 2013; Samanez-Larkin, Worthy, Mata, McClure, & Knutson, in press). Moreover, a pharmacological fMRI study in older adults showed that these effects can be restored using l-DOPA (a precursor of DA), indicating a direct link between compromised DA levels and the representation of prediction errors in the ventral striatum in older adults (Chowdury et al.,

2013). Therefore, the current literature suggests that age-related impairments in habitual learning (learning from experience) may be mediated by deficits in dopaminergic prediction error signaling in subcortical areas.

Lifespan Developmental Differences in Model-Based Learning and Decision Making

In contrast to the nonlinear developmental trajectories of the habit system, model-based mechanisms, which are involved in making adaptive, flexible decisions based on internal goal representations, can be expected to show a linear developmental trajectory across childhood and adolescence (Marcovitch & Zelazo, 2009; Munakata et al., 2012). Evidence from structural MRI studies indicates that the cortical volume and thickness of the lateral and orbitofrontal PFC as well as the parietal cortex continue to develop into early adulthood (Gogtay et al., 2004; Shaw et al., 2006; Sowell et al., 2004). This is consistent with results of several functional imaging studies that suggest that developmental differences in the ability to use internal goal representations to guide behavior, particularly in challenging situations, can be attributed to a protracted maturation of the PFC (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Crone, Donohue, Honomichl, Wendelken, & Bunge, 2006; Velanova, Wheeler, & Luna, 2008). Moreover, recent theoretical approaches link the development of more abstract goal representations to a hierarchical maturation of areas within the PFC (e.g., Munakata et al., 2012). Such a hierarchical view on PFC development would predict that the ability to dynamically allocate control to the two decision systems, depending on the uncertainty of the value estimates they generate, may operate efficiently only very late during development. This is because the anterior prefrontal cortex that such a process relies on is one of the latest to be fully matured in humans (Badre, 2008; Koechlin & Hyafil, 2007; Koechlin, Ody, & Kouneiher, 2003).

The prefrontal cortex is also one of the areas that are most vulnerable to effects of aging. Longitudinal estimates from structural MRI studies point to an age-related decline of approximately 1.05 cm^3 in grey matter volume and 1.07 cm^3 in white matter volume in the prefrontal cortex per year (Raz et al., 2005; Resnick, Pham, Kraut, Zonderman, & Davatzikos, 2003). Similarly, longitudinal functional MRI studies show evidence for an age-related under-recruitment of the prefrontal cortex (Nyberg et al., 2010). This deficit in PFC recruitment seems to be particularly pronounced in tasks with high demands on strategic goal-directed learning and decision making (Eppinger, Heekeren, & Li, 2015; Mell et al., 2009).

To summarize, current findings suggest that development across childhood is characterized by an increasing ability to internally represent task structures (state spaces) and to apply the internalized knowledge of the world in a goal-directed (model-based) manner. The development of strategic goal-directed decision processes is tightly coupled to the maturation and functional integrity of the prefrontal cortex (PFC). Thus, age-related impairments in PFC function should be

reflected in a decline of model-based decision processes with age. Based on the current literature it could be expected that the ventral striatal system, which is involved in model-free learning, shows a different nonlinear developmental trajectory. In light of the findings reviewed earlier, we expect that during childhood development the ventral striatal system is functionally more mature than the prefrontal system. During adolescence the imbalance between the two decision systems is further elevated due to a hypersensitivity of the ventral striatal system. This imbalance may lead to decisions biases (e.g., risky behavior, a greater focus on immediate reward) as well as protracted development of model-based behavior. During aging, one can expect functional declines in both systems—the ventral striatal habit learning system, as well as the prefrontal system, which is engaged in strategic, goal-directed decisions. The prediction regarding age-related decline in the prefrontal system is relatively straightforward. Prefrontal deficits should lead to a shift away from a model-based decision strategy to a model-free strategy. Regarding age-related decline in the striatal system there are two possible scenarios: On the hand one could predict that age-related deficits in striatal reward prediction error signaling should lead to impairments in model-free reinforcement learning (a reduced reward effect). However, it could also be that decline in the striatal system impairs the ability to keep track of the reward histories of the different choice options. As a consequence older adults may converge on a model-free strategy that focuses only on the most recent outcome (in terms of reinforcement learning, a model-free strategy with a learning rate close to 1).

Lifespan Development of Interactions between Habitual and Strategic Goal-Directed Learning and Decision Making

So far, most of the work on age differences in learning and decision making has focused on habitual or goal-directed learning and decision-making mechanisms in separation. The dynamic interplay between these processes in different age groups and questions about the factors that influence these interactions have not been examined directly. Moreover, many of the current behavioral approaches suffer from the fact that they cannot precisely disentangle the contributions of habitual and strategic goal-directed decision processes in different age groups. To address this question we will refer to a two-stage Markov decision task (see Figure 14.5) that has been used in recent studies to dissociate habitual and goal-directed contributions to choice behavior using a computational approach (Daw et al., 2011; Wunderlich et al., 2012). We will then formulate predictions regarding the lifespan developmental trajectories of these processes (see Figure 14.6A) to simulate choice patterns in the two-stage Markov decision task for the different age groups (see Figure 14.6B). Finally we will validate the simulation results using empirical findings from a recent behavioral study comparing younger and older adults with this paradigm (Eppinger, Walter, et al., 2013).

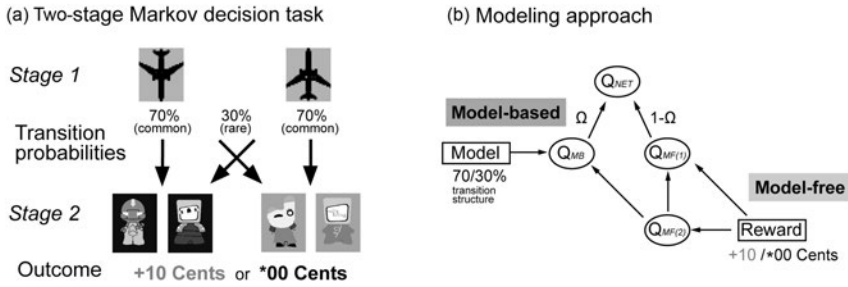

FIGURE 14.5 (A) Schematic picture of the two-stage Markov decision task. On the first stage of this task participants have to make a goal-directed decision that integrates knowledge of the transition structure with knowledge of the currently best option on the second stage. (B) A hybrid RL algorithm is used to model choice behavior in the task. The model provides an estimate of the relative contribution of model-free and model-based decision mechanisms to behavior (see **Ω**-parameter in the figure). Adapted from "Of Goals and Habits: Age-Related and Individual Differences in Goal-Directed Decision-Making," by B. Eppinger, M. Walter, H. R. Heekeren, and S.-C. Li, 2013, *Frontiers in Neuroscience,* 7, p. 3. Copyright 2013 by Frontiers in Neuroscience.

Two-stage Markov decision task. The idea of the two-stage Markov decision task is that in order to reach a preferred (rewarded) state on the second stage of the task participants have to engage in a strategic decision on the first stage (see Figure 14.5A). That is, they have to integrate model-free information about the reward probabilities on the second stage with a model-based representation of the transition structure on the first stage (see Figure 14.5A). Intuitively this means that at the second stage of the task participants have to continuously learn which is currently the best option (model-free learning). However, in order to get to the currently preferred stimulus at the first stage, they have to make a model-based decision that incorporates the transition probabilities into their decision. For example, in order to get to the lower right figure with the lighter background in Figure 14.5A most reliably (in 70% of the cases), one has to choose the upper right option. However, given the probabilistic nature of the transition structure, from time to time one will also end up at the other two states (figures with the darker background in Figure 14.5A). The critical dependent variable in the task is the choice behavior on the first stage as a function of the reward participants received on the previous trial (reward, no reward) and as a function of the transition that participants had on the previous trial (common or rare). Model-free behavior on the first stage is characterized by a main effect of reward (greater stay behavior after rewarded than unrewarded trials). Model-based behavior is reflected in an interaction between transition type and reward. That is, the participant takes the reward and the transition type ("How do I get to the reward?") into account.

Computational model and simulations. To simulate lifespan developmental difference in the relative contributions of model-free and model-based decision processes and to analyze the choice data in Eppinger, Walter, et al. (2013) we used a hybrid reinforcement learning model (Daw et al., 2011; Eppinger, Walter, et al., 2013; Wunderlich et al., 2012; for a schematic depiction see Figure 14.5B). This algorithm assumes that choices on the first stage of the task are driven by a weighted combination of model-based reinforcement learning, which accounts for the transition structure, and model-free SARSA (λ) TD learning, which accounts for the reward effects. The weighting of model-based versus model-free decision mechanisms is determined by the free parameter omega, ω. If ω approaches 0 behavior is model-free, which is reflected in a main effect of reward. In contrast, an omega close to 1 indicates model-based choice behavior, which is reflected in an interaction between transition structure and reward on the previous trial. For the purpose of the simulations we used the parameters from the younger adults sample in Eppinger, Walter, et al. (2013) and manipulated the ω-parameter according to our predictions outlined in Figure 14.6A. It should be noted that the model is not an explanatory computational model because it is agnostic about the mechanisms that lead to the changes in the model-basedness

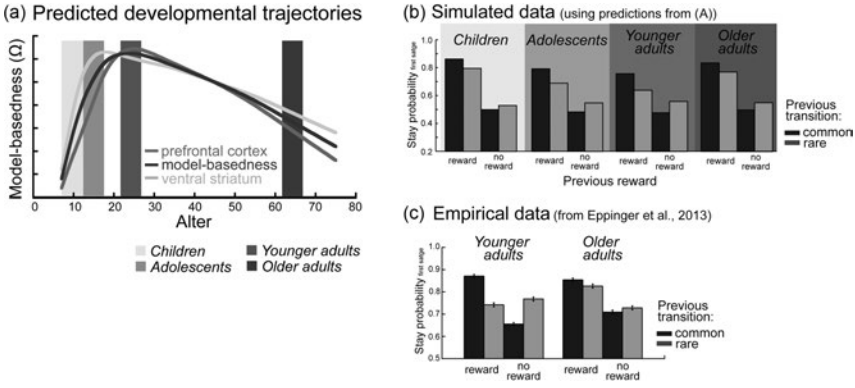

FIGURE 14.6 (A) Predicted developmental trajectories for the ventral striatal system (light grey), the prefrontal (grey), and model-based behavior (black). (B) The developmental trajectories for model-based behavior from (A) were used to simulate choice data in the two-stage Markov task using the hybrid reinforcement learning model (see Figure 14.5B). The results show a protracted development of model-based behavior during adolescence as well a shift from model-based to model-free decision making in older age. (C) The empirical results from Eppinger, Walter, et al. (2013) confirm the model predictions of an age-related shift from model-based to model-free behavior. Adapted from "Of Goals and Habits: Age-Related and Individual Differences in Goal-Directed Decision-Making," by B. Eppinger, M. Walter, H. R. Heekeren, and S.-C. Li, 2013, *Frontiers in Neuroscience,* 7, p. 3. Copyright 2013 by Frontiers in Neuroscience.

parameter ω. We think that the added value of the model is that it provides us with very specific predictions regarding the developmental trajectories of model-based and model-free decision making across the human life span.

As expected, the results of the simulations show an increase of model-based behavior with development. With age children are more and more able to overcome their tendency for habitual responding. That is, they integrate model-free information about the expected value of the decision options on the second stage with the transition probability structure of the task (as reflected in an interaction between transition on the previous trial and reward on the previous trial). As shown in the simulation results in Figure 14.6B, aging is associated with a shift from model-based to model-free behavior, indicating that older adults have difficulties in integrating the two types of information. This pattern of results is nicely consistent with empirical findings from a recent age-comparative study using the same task (Eppinger, Walter, et al., 2013). Results of this study showed a strong reduction of model-based behavior in older compared to younger adults (Figure 14.6C). Interestingly (and not predicted from the model simulation), this effect was particularly pronounced in task conditions in which unexpected reward on the second stage indicated that the decision strategy on the first stage had to be adjusted (trials in which a rare transition resulted in reward). In these task conditions older adults perseverated choosing the suboptimal option, whereas younger adults engaged in a strategic exploration of the decision space using their knowledge of the task transition structure. Moreover, the results of the Eppinger, Walter, et al. (2013) study suggest that older adults don't integrate the reward history as much into the choice behavior as younger adults do. This is reflected in an enhanced reward influence (λ-) parameter for older than younger adults, which suggests that they may focus only on the most recent outcome. Taken together, the current findings are consistent with the idea that PFC deficits in older adults lead to a shift from model-based to model-free decision making. The fact that older adults show a reward effect (a "model-free" choice pattern on the first stage) does not necessarily mean that model-free TD learning is intact in older adults. Rather it seems that older adults converge on a simplifying strategy that focuses only on the most recent outcome (in terms of reinforcement learning, a model-free strategy with a learning rate close to 1).

So far, the developmental predictions of the simulation have not been tested empirically. Given the findings in older adults it would be interesting and important to see (a) whether the empirical findings in children and adolescents match our simulations, which predict a protracted development of model-based behavior during adolescence, and (b) whether children and adolescents show a similar degree of perseverative behavior as older adults. Moreover, the neurophysiological mechanisms that lead to these age-related changes in model-based behavior across the life span are not yet clear. In particular, it would be important to know whether decision-making deficits in children and older adults are due to deficits in similar underlying neural mechanisms or whether age differences in the underlying neural

systems can be dissociated. Given our assumptions shown in Figure 14.6A, we would expect that deficits in model-based behavior in children are primarily due to their still developing prefrontal cortex, whereas in older adults deficits in ventral striatal as well prefrontal function may contribute to the observed shift from model-based to model-free behavior.

Outlooks and Concluding Remarks

Complex behavior, such as goal-directed learning and decision making, requires self-regulated action and behavioral control, for which smooth operations are implemented through fluid interactions between cortical monitoring, hippocampal memory, and subcortical motivational processes. As reviewed in the preceding sections, through its nigrostriatal, mesolimbic, and mesocortical pathways the dopaminergic system is at the interface for modulating the interactions of cortical cognitive processes as well as subcortical reward-associated motivational processes. The maturation and senescence of the dopaminergic systems across the life span thus may play important roles in self-regulated developmental adaptions. Specifically, age-related differences in dopaminergic modulation may affect cognitive mechanisms of monitoring behavior and choice outcomes, effects of reward on memory and attention, and the relative balance in habitual and flexible goal-directed processes. As such, maturation or senescence-related effects on cognition and motivation not only are important for understanding the development and aging of these processes, but also serve as important models for understanding how relative balances between the efficacies of frontal-hippocampal-striatal circuitries may contribute to individual differences in the reciprocal interactions between cognition and motivation.

The past decade has witnessed more and more studies using differential developmental or aging trajectories of different brain circuitries to understand the relative contributions and sub-processes of various neurocognitive functions (e.g., Crone et al., 2008; Passow et al., 2013; Posner, Rothbart, Sheese, & Voekler, 2012; Schuck et al., 2013; Shing et al., 2009). For instance, the relative maturation rates of the cortical and subcortical systems in adolescence have attracted much attention (e.g., Casey et al., 2008; for reviews see Crone & Dahl, 2012; Sommerville & Casey, 2010). However, it should be noted that some of the underlying assumptions of these accounts have recently been questioned. For example, findings from a longitudinal structural MRI study on developmental changes in subcortical structures show that the volumes of subcortical structures, such as the striatum and pallidum, show a protracted development when compared to cortical developmental trajectories (Raznahan et al., 2014). These new data question a simplistic maturation lead-lag model of subcortical functions leading cortical functions and call for more in-depth investigations of the relations between the development of the fronto-striatal network at the anatomical and neuromodulatory levels. Also, there are results of several previous studies that speak against the idea that adolescents

show a generally enhanced sensitivity to reward (Bjork et al., 2004; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010). For example, in our own work we found no significant differences in the effects of implicit reward learning on visual attention in adolescents compared to children or adults (Störmer et al., 2014). This may suggest that enhanced limbic reward responses and associated decision biases may be confined to task conditions in which individuals have to explicitly consider different decision options.

Given that currently there is more convergent evidence relating lifespan differences in the efficacy of dopaminergic modulation, this review focuses only on the contributions of dopamine to lifespan development of cognitive and motivational self-regulations as well as their interactions. However, the effects of other transmitter systems and how they interact with the dopamine system in modulating cognition and motivation need to be investigated more systematically. For instance, it has been proposed that the development of different cognitive control networks (e.g., alerting, orienting, executive attention) may be modulated by different neurotransmitter systems, with the orienting attention being modulated by the cholinergic system and executive attention being modulated by the dopamine system (Posner et al., 2012; Störmer et al., 2012). It has also been proposed that serotonin may interact with dopamine in regulating approach- and avoidance-associated motivational influences on actions (see Guitart-Masip, Duzel, Dolan, & Dayan, 2014, for recent review). Although the serotonin system may be even harder to study than the DA system due to its widespread and complicated projections, more work on the interactions of these systems is necessary to develop a mechanistic understanding of age differences in cognition–motivation interactions. Method-wise, recent studies that applied TMS over the frontal cortex to affect task-related striatal activity (e.g., van Schouwenburg et al., 2012) and dopamine release in the caudate nucleus (Strafella et al., 2001) suggest that applying noninvasive brain stimulations to regulate the frontal-striatal circuitry might potentially be another mean for investigating lifespan development of the interplays between cognition and motivation.

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