The Neuronal Substrate of Risky Choice

An Insight into the Contributions of Neuroimaging to the Understanding of Theories on Decision Making under Risk

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This chapter provides an overview of studies in the field of neuroscience that investigate some of the processes and concepts of risk perception, risky choice, and decision making under risk. First, early studies in the field of neuroscience addressing the diminished decision-making abilities in lesion patients are presented. A classical task in this research field is described along with its neural implications. After this, the underlying model, its hypotheses, and neuronal implications are discussed. Different aspects within risky decision making, such as the influence of memory, inhibition, motivation, and personality, on risky choice and the respective underlying neuronal substrate are described. After this, studies of risky decision making in healthy subjects are reviewed. A selection of studies shows that theories focusing on cognitive aspects only have to be enriched in order to allow for additional aspects within risky decision making (e.g., emotion). Next, the classical economic approaches and the development of theories incorporating further aspects within economical decision making and the underlying neuronal substrate will be presented. Finally, research in the field of neuroeconomics, focusing on the role of social decision making and evaluative judgment within risky decision making, is reviewed.

Key words: **decision making; risk; fMRI; neuroeconomics**

Introduction

In recent years, growing knowledge, new technologies, diseases, environmental challenges, and progress in science have broadened existing definitions and concepts of risk. The more complex models and definitions of risk become, the more hope is put into new technologies to provide a deeper understanding. Brain imaging techniques, such as functional magnetic resonance imaging (fMRI), allow studying the neural basis of various cognitive functions by investigating changes in cerebral blood flow, which is an indirect measure of oxygen use at specific locations in the brain. fMRI provides insight into the spatial distribution and dynamics of brain activation as a function of stimulus presentation or task performance or as a consequence of disease or medication. At first glance, fMRI seems to be a straightforward, promising, and convincing tool that directly accesses cognitive function and provides a window into the brain. It offers the "extraordinary opportunity" to observe the processes of the human brain,[1] and it is seen as the "key technology of our times."[2]

Especially enthusiastic are researchers in fields that are in a stalemate concerning new ideas for further research. "The neuroscience revolution occurred when . . . scientists realized that the best hope for understanding the workings of the brain comes from an interdisciplinary approach, a combination of traditional approaches to yield a new synthesis, a new perspective"(p. 4).[1] For example, researchers in the field of neuroeconomics hope to find evidence for the neuronal substrate underlying risk perception or decision

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making under risk. By connecting these processes to behavior, they hope to gain a better understanding of the phenomenon.[3] Nonetheless, *even if* imaging techniques allow investigating neural correlates of cognitive function, certain limitations in temporal and spatial resolution restrict design and experimental manipulation and hence the research questions that can be answered with this technique.

This chapter provides a critical overview of neuroscience studies that investigate processes and concepts potentially relevant to risk perception and communication. First, early studies by Bechara and colleagues addressing the issue of risk perception in patients with lesions within the ventromedial prefrontal cortex (VMPFC) are presented.[4–12] The classical gambling task in research fields investigating risk decision making is introduced, and the neural implications are presented and critiqued. After this, the somatic marker hypothesis (SMH) and its neuronal implications are discussed. Different aspects of risk perception using such decision-under-risk paradigms are shown. In particular, slight changes in the tasks result in an altered underlying neuronal substrate. Neural changes are supposed to highlight the influence of memory, inhibition, motivation, and personality on risky choice. After this, the underlying neuronal substrate of risky and informed decision making in healthy subjects is summarized.

In the subsequent section, a selection of studies in neuroscience is introduced that show that theories focusing only on cognitive processes have to be enriched with models that further distinguish possible emotional aspects of risk decisions. In the next section, risky choices investigated with economic models are introduced. This section will start with classical approaches and lead to the development of Prospect Theory (PT) and its assumptions. The hypothesized underlying neuronal substrate as well as the problems arising will be described. Finally, the aspects in neuroeconomic studies focusing on social decision making and the role of evaluative judgment within risky decision making will be critically discussed.

The Iowa Gambling Task and Neural Implications when Administered to Lesion Patients

In the field of neuroscience, studies simulating real-life risky decision making in a laboratory setting detect the cognitive and neural mechanisms that, when damaged, are responsible for impairments. Such impairments have been observed, for example, in patients with damage to certain sectors of the VMPFC.[a] The problems that result occur especially within the personal and social functioning of the patients. Symptoms manifest themselves, for example, in obliviousness toward the future consequences of actions and the reward-related guidance of behavior.[8] The literature gives a detailed description of the symptoms these patients suffer.[13,14] Beyond difficulties with the cognitively demanding aspects of a task (e.g., estimation of prices), the ability to understand, experience, and effectively use emotions is especially impaired.[14]

Bechara *et al*.[8] introduced the "Iowa Gambling Task" (IGT), an experimental neuropsychological task in which participants search for monetary payoffs by selecting cards from several decks arrayed before them. Each deck holds a certain number of winning/rewarding and loosing/punishing cards, and the relative proportions of these cards can be learned by repeated drawing. Healthy subjects are able to make successful choices based on their intuitive estimates of which decks are risky and which decks are profitable in the long run. They show an improvement over time. They fail, however, to give an account of the net gains and losses associated with any single deck. In their initial article, Bechara and colleagues introduce E.V.R., a prototypical patient with damaged VMPFCs who behaves disadvantageously in real-life decision making (pp. 7–8).[8] To determine the specific influence of the VMPFC on decision making under risk, subsequent research with the IGT included six "E.V.R.-like subjects" and compared their performances to nine control patients with lesions in other brain regions (p. 10).[8, b] The researchers found this task detects impairments in brain-damaged patients. Such patients perform "defectively in this task, and . . . the defect is stable over time" (p. 13).[8]

Studying patient deficits in gambling tasks enable researchers to compare performance within or even between patient groups. Moreover, such studies allow the assignment of behavioral deficits to particular brain areas when lesions are shared or overlap. However, there are some constraints of fMRI techniques that limit the ability of these studies to provide an exact causal relationship between lesions and the behavior in deficit. For example, patient studies usually investigate

[a]The VMPFC, as classified by the Iowa group, includes the medial orbitofrontal cortex (OFC) and parts of the lateral OFC. Moreover, it overlaps with medial Brodmann's areas 10, 11, and 12 and lower Brodmann's areas 24, 25, and 32.[5,6] It is important to note that other authors refer to a similar region when using the term OFC.[15,16]

[b]Detailed descriptions of the patients' lesions are given in Bechara *et al*.[12]

a few patients only, which makes statistically meaningful analyses difficult. Moreover, although the lesions of patients overlap in a region of interest, such as the VMPFC, patients' lesions are not of an equal size and hence cover heterogeneous regions of the brain.

It is also important to note that in different studies the labeling of brain areas varies. The data regarding individual brain structure, once acquired, has to be spatially preprocessed and normalized via (automated) transformations in order to match a standard brain physiology template. This enables the researcher to make anatomical and physiological statements and comparisons between the brains of different patients. Functional data is likewise normalized to the template, enabling the assignment of functions to specific brain structures. These transformations and normalizations can be tricky when the brain of a patient is physically damaged. Thus, it is important to be aware of the need in patient or lesion studies to consider alternative techniques to normalize the data in order to overcome a mismatch between template, image, and functional data.[17]

The Somatic Marker Hypothesis and Its Neural Implications

The inability of patients to decide advantageously (i.e. to, learn from an experience in order to gain a reward) led to the formulation of the SMH.[14] The SMH states that, in an uncertain situation where different behavioral alternatives can be chosen, somatic markers guide behavior by excluding previously experienced emotionally charged behaviors. Somatic markers can be either "primary inducers," like unconditioned pleasurable and aversive stimuli, or "secondary inducers," such as memorable learned entities that are generated by recall. The inducers produce a somatic state, which in turn can overtly be perceived as a feeling or covertly remain as a representation at the level of the brain stem.[18,19] Somatic states reached through secondary inducers enable the re-experiencing of a feeling that is associated with something. All of these somatic states, either negative or positive, combine to produce one overall somatic state. This overall state provides important signals to the brain, including the overall emotional state (via insula, somatosensory cortex) and it suggests *overtly* or *covertly* how decisions should be biased when choosing between alternatives.[5,8,10,14,20]

Bechara and colleagues[8] state that the IGT detects brain-injured patients' impairments in a controlled setting. They assume that patients' performances reflect "their real-life inability to decide advantageously, especially in personal and social matters. . . . In life, as in the task, an exact calculation of the future outcomes is not possible and choices must be based on approximations" (p. 13).[8] Moreover, with the SMH the respective roles of primary and secondary inducers in decision making can be differentiated.[14] Conclusions can also be drawn about the underlying neuronal substrate of the inducers as these are believed to be regulated by different brain regions.

The VMPFC is the part of the brain most crucial in encoding secondary inducers via dispositional associations, i.e., it recalls the learned entities and bodily states from past experiences. The VMPFC is thought to reactivate representations of a situation or state by recruiting this information from other areas in the brain. Secondary inducers can be recalled from areas representing a change in the body, like the somatosensory cortex, insula, cingulate, basal ganglia, and brain stem nuclei.[21] For primary inducers, i.e., unconditioned emotional stimuli, the VMPFC recruits information from the amygdala.[4,9] It appears that damage to the amygdala alters performance in the IGT because of its role in linking stimuli to affective attributes. The VMPFC holds the role of a decision maker over many alternative inputs received from other areas as well.[4] Furthermore, neurotransmitter systems, like the dopaminergic system in the brain stem, are thought to bias the action of the somatic states in response selection[11,14,21].

The assumptions of the SMH were tested by the Iowa laboratory in several studies[5,8,10,12]. Results show that somatic markers, as measured by skin conductance responses (SCRs), allow people to make better choices even before conscious knowledge is accessible. In the initial study, the authors provide three different explanations for the impaired patients' behavior: the patient is extremely sensitive to reward, is insensitive to punishment, or is generally insensitive to future consequences. After varying aspects of the task, the authors concluded that patients fail to perform advantageously in the task because they are "unresponsive for future consequences,"[8] reflecting "myopia for the future."

The SMH shows how a (cognitive) model can contribute to the understanding of a phenomenon of interest. It delivers hypotheses that can be tested and falsified despite the fact that one experiment was not enough to provide satisfying evidence. Altogether, the SMH has, via the IGT, identified some brain regions involved in decision making under risk. It shows how emotion-related feedback from the body can guide performance. Nonetheless, it fails to describe how exactly the interaction between body state, emotion, and decision making is supposed to happen, especially when the biases guiding behavior are unconscious.[21,22]

The Role of Working Memory, Inhibition, Motivation, Personality, and Underlying Neuronal Substrate in Risky Choice

Since its inception, different aspects of the IGT have been varied and results have been replicated and augmented. Apart from new ideas about mechanisms and factors other than "myopia for the future" that might be responsible for the findings, these studies provide evidence for additional brain regions presumably involved in regulating task performance. This is true not only for tasks focusing on decision making under risk but also for a whole range of tasks that are likely to be influenced by the same factors (e.g., evaluative judgment, risk judgment).

The role of working memory on the IGT was questioned by studies with dual-task methodologies.[23,24] Bechara *et al*.[7] dissociated working memory from decision-making capacities by showing that working memory did not depend on intact decision making. Interestingly, decision making was affected by working memory. Performance in the IGT was worse when patients had working memory problems. A broader damage to the prefrontal cortices (PFCs), including the dorsolateral prefrontal cortex (DLPFC), leads to impairments in the IGT.[7,16] Other authors tested for different explanations of the impact of working memory in task performance.[22,25] New methods to screen the knowledge people have about the card decks reveal that a hypothesis about unconscious somatic markers may not be necessary to explain the behavior in gambles.[22] The authors conclude that people know about the advantageous strategy. Whether they apply this knowledge to guide their behavior remains another question.

Other research considers that impaired performance in the IGT may be because of an inability to inhibit responses or deficits in reversal learning. Studies investigating the impairments of patients with frontal lobe damage have found that ventral PFC and OFC (but not DLPFC) patients show an impaired reversal learning performance.[26–28] If impairments in reversal learning are responsible for poor performance in the IGT, we have to add the OFC as a candidate region in risky decision making.

Deficits in task performance from a lack of motivation or personality influences were investigated. Different patient groups were recruited and their typical behavioral patterns were analysed (e.g., increased or decreased betting). Personality changes in participants with either bilateral VMPFC lesions or with prefrontal lesions but not bilateral ventromedial involvement and persons with nonprefrontal lesions were investigated. The results suggest that diminished emotionality, impaired decision making, and psychosocial dysfunction may be related to ventromedial prefrontal dysfunction in both prefrontal lesion groups compared to nonprefrontal.[29]

Sanfey *et al*.[25] investigated patients' risk preferences independently from the accuracy of their decisions in order to detect whether they were risk seeking or avoiding. They found VMPFC patients to be both risk seeking and risk avoiding. In comparison, healthy controls, both old and young, were risk averse. Moreover, the authors could not clearly disentangle any lesion differences that could contribute to a better understanding of the findings for the patient group. The risk-taking group only *tended* to have lesions extending to the DLPFC. Hence, these studies do not suggest additional regions of interest nor do they exclude the VMPFC concerning the role it plays in risky decision making.

Risky and Informed Decision Making and the Neuronal Substrate in Healthy Subjects

So far we have discussed conclusions about candidate regions drawn from knowledge about lesion locations in impaired patients. Studies using neuroimaging also gain evidence from the investigation of healthy subjects. Hence, we can draw conclusions about the underlying neural substrate of task aspects. Such studies provide mixed support for the central role of the VMPFC and OFC. When the decision-making component of a computerized "Iowa Gambling-like-task" was isolated, decision making, per se, activated OFC, anterior cingulate cortex (ACC), medial PFC, DLPFC, insula, inferior parietal cortex, thalamus, and cerebellum. Guessing (i.e., unlearned task performance occurring within the first task administration) activated sensory-motor associative areas and the left amygdala. Informed decision making (learned risk-taking task performance during a second administration) activated the hippocampus, posterior cingulate (responsible for memory processes), striatum, and the cerebellum (regulating motor control).[30] Moreover, a study focusing on the risk anticipation period of the IGT found that during that period the medial frontal gyrus was activated exclusively. Interestingly, no orbitofrontal regions were activated.[31]

We have to bear in mind that especially fMRI data are likely to be distorted in the frontal brain and that they are relatively slow measures. Hence, subtle variations of task aspects in order to tease out even more specific regional functions might be an aim that can only be reached with the support of additional physiological measures (e.g., eye movement parameters, physiological parameters from the pupil, SCR,

reaction time measures). Further, even if participants of these studies were considered "healthy," this does not mean that their brains are exactly matching. Variations are conceivable in structure and function. Additionally, personality traits (e.g., sensation seekers versus harm avoiders) and constitution may differ, not to mention gender differences that could lead to performance discrepancies and hence should at least be controlled.

The Neural Substrate for Emotional, Social, and Cognitive Intelligence within Decision Making under Risk

Sanfey[20] sum up the advantages of the SMH in that it offers a way of integrating emotional influences into a domain that predominantly would explain behavioral strategies by traditional views within cognitive psychology. He picks up some critical points regarding existing studies. According to Sanfey,[20] the SMH fails to clearly indicate how unconscious emotional processes interact during decision making. The SMH does not offer a way to explain in detail the emotional processes (e.g., fear, anger) or the somatic markers triggered by the IGT.

To better disentangle emotional and cognitive processes in decision making, Sanfey *et al*.[25] investigated performances in another game known as the Ultimatum Game. In this game, two players have to split a sum of money. One of the participants, the offerer, proposes a split of the money and the other, the responder, can accept or reject this offer. If the offer is rejected, neither participant is rewarded. Theories based on rational choice predict that no offer should ever be rejected by the responder. However, in all studies employing the game, responders readily reject low offers.

People reject offers not because they lack knowledge about how to play the game but rather because they react angrily to an offer perceived as unfair. These studies provide evidence for the neural basis of decision making under risk, while suggesting an important role for emotions, besides cognitive processes, in risky decision making. Unfair offers elicit activity in the DLPFC and ACC which relate to cognitive processes, but, more interestingly, they also result in bilateral activity in the anterior insula, an area related to emotion processing. Moreover, activation in this emotional state processing region correlated with subsequent decisions to reject or accept an offer, showing stronger activation in subjects that rejected more unfair offers. Hence, the authors concluded that "neural representations of emotional states guide human decision-making" and that "models of decision-making cannot afford to ignore emotion as a vital and dynamic component of our decisions and choices in the real world."[25]

Further evidence that the neural substrate for emotional and social intelligence and cognitive intelligence are different is provided by Bar-On *et al*.[32] They compared patients with average cognitive intelligence and with an emotional intelligence lying below average. The experimental group included three kinds of patients with lesions in either the ventromedial cortex, the amygdala, or, alternatively, in the insula/somatosensory cortex. Control subjects had lesions in the superior and/or middle frontal gyri in the right PFC, right precentral gyrus, and/or in the right paracentral lobule with neither damage below the corpus callosum nor an extension to the frontal pole. In some subjects, the middle temporal gyrus and the occipital cortex (without insula or somatosensory cortex) were affected too. Emotional intelligence was measured with the Bar-On EQ-I, which is a self-reported measure of social and emotional behavior. It provides an estimate of underlying emotional and social intelligence.[33]

With the IGT, the researchers investigated the subjects' ability to "exercise personal judgment in decision making." Social functioning was employed with semi-structured interviews as well as rating scales that provided a "Total Social Scale" score indicating the extent of impairment (see Tranel *et al*.[34] for more details). Cognitive intelligence, perception, memory, and executive functioning were also assessed and, as intended, did not differ between the groups. Additionally, no significant correlation was found between cognitive and emotional intelligence.

Both social functioning and personal judgment in decision making differed significantly between the groups. On the one hand, the authors' assumption that emotional and social intelligence as well as the underlying neuronal system differ from cognitive intelligence and its neural substrate is supported. On the other hand, the results reconfirm the neurological substrate that mediates somatic state activation and personal judgment within decision making found in patient studies. These neural systems seem to overlap with those of social and emotional intelligence. The authors sum up that

> the processes of judgment and decision making depend on neural systems for: (i) memory, which is supported by high-order association cortices as well as the dorsolateral sector of the prefrontal cortex; (ii) emotion, which is mediated by subcortical limbic structures that trigger the emotional response; and (iii) feelings which are supported by limbic as well as closely associated regions such as the insula, surrounding parietal cortices and the cingulate

> cortex. (. . .) The VMPFC links these systems together; (. . .). Impairment of these mechanisms (. . .) can have an ill effect on one's ability to effectively cope with daily demands. Such impairment may include a decrease in one's ability to: (. . .) (v) cope flexibly with the immediate situation, make decisions and solve problems of a personal and interpersonal nature.[32]

The studies by Sanfey and Bar-On indicate that ideas regarding candidate areas can come from all types of studies that investigate behavior that is based on a variety of cognitive processes possibly intermingled in the behavior of interest. Examples are studies exclusively investigating neural networks of emotions or emotion processing[35–37] or studies focusing on task variations that only trigger cognitive processes. Overall, fMRI does not unambiguously delineate areas responsible solely for one behavior of interest. In other words, there is no area exclusively active during risk perception. Connections in the brain are complex, and hypotheses regarding locations of activity from a specific task have to be discerned with the help of cognitive models.

Ecological Decision Making

A different origin for neuroscientific approaches related to a concept of risk comes from studies interested in the neural circuits associated with ecological decision making. Neuroeconomical theories have been developed describing the neuronal substrate of behavior by integrating normative approaches of emotion and cognition with decision making. In the quickly growing field of neuroeconomics, the overall goal is to provide a better understanding of the cognitive processes of decision making and to draw conclusions for risky behavior in everyday life.

Most classical economic theories assume that humans are rational decision makers that follow the rules of probability without being influenced by emotions. Given this assumption, mathematical models and theories exist that predict and explain decision making. Such models are the most valuable tools ecologists have had so far to analyse complex problems. However, these models often fail to consider biases in human decision making. They neglect that people might use various, rather irrational, strategies (e.g., heuristic routines) to overcome uncertainties in risky decision making. As a consequence, the rational economists' models, providing a normative description of behavior, were supplemented with psychological and biological assumptions. The purpose was to account for less rational, more realistic, human behavior and to be able to draw conclusions and generate hypotheses about behavior by looking at the underlying neural processes.

In the following section, the classical economical view of peoples' behavior is introduced and it is shown how deficits of such models led to the formulation of PT. After that, the neural implications of neuroeconomic models are discussed.

From a Rational Economic View to the Development of Prospect Theory

Bernoulli, a Swiss mathematician, observed in 1738 that people do not evaluate options by their objective utilities to maximize their expected (net) wealth but rather by some kind of personal utility system.[38] He assumed that within this personal utility system one chooses the option with the highest expected utility in each possible state, constructing a weighted average. The weights are probability estimates of each state; hence, the expected utility is an expectation in terms of probability theory. The Expected Utility Theory, introduced later by Neumann and Morgenstern,[39] proved that any normal preference relation over a set of states can be written as expected utility. It is one of the most traditional models and it describes decisions under risk and uncertainty with the help of a utility function. The function assumes a concave relationship between wealth and utility. Nevertheless, this model fails to explain certain paradoxes (e.g., the Ellsberg paradox, the Allais paradox), and hence the model's descriptive value was questioned.[40]

A new theory introduced by Kahneman and Tversky,[41] PT, replaced the term "utility" with "value." PT predicts that preferences will depend on how a problem is framed. Value is defined in terms of gains and losses, i.e., people do not make decisions based on the absolute level of net wealth. Gains and losses are evaluated relative to some kind of neutral reference point, which may vary from situation to situation. Moreover, the value function for losses differs from that for gains in that it is convex and steeper with its curve lying below the horizontal axis.[41] Unlike the Expected Utility Theory, which concerns itself with how decisions under uncertainty *should be made*, PT concerns itself with how decisions *are actually made*. It is taking into account some irrational tendencies or asymmetries, reflected in so-called framing and editing procedures within human choices, by introducing a flexible reference level for the decision maker.

Neural Implications of Neuroeconomics and Prospect Theory

In economic models, findings of research focusing on "decision utility," "expected utility," and "outcome probability" (OP) can be distinguished. In PT, decision utility is central. PT attempts to overcome

shortcomings of classical economical models by defining subjective value functions with different assumptions about the curves for gains and losses. The impact of high and low probabilities is characterized by a weighting function. Additional major components of PT are the "value function," which focuses on the neuronal representations of values, the "weighting function," which focuses on the neural processes involved in the generation of such a value function, and "representation." The latter encompasses a presentation of the neural processes underlying framing effects and editing operations that help inform behavior.

Decision utility and expected utility predict neuronal representations from anticipated and experienced reward and punishment. Results from neuroimaging studies suggest that different brain mechanisms in decision making could embody both forms of utility. Signals related to reward, if experienced or expected, have been found in brain areas, such as the striatum, DLPFC, OFC, posterior parietal cortex, supplementary eye field, and ACC and posterior cingulate cortex (for a review see Ref. 42).

Regions involved in decision utility, which is an estimate or anticipation of experienced utility during decision making, should be recruited during reward anticipation or when a subjective value of future events is evaluated. The dopaminergic system is a system arising from the midbrain to various cortical and subcortical areas. The system occupies a modulatory neurotransmitter, dopamine, which seems to be the primary substrate for decision utility. Neurons within this system heighten their firing rate when unexpected rewards or stimuli that predict future rewards are being processed (see Schultz[43] for a review). Candidate brain areas are the ventral striatum, PFC, and the amygdala. The ventral striatum is one of the target regions of the dopaminergic system. It integrates signals coming from PFC, amygdala, and hippocampus, and it seems to play a crucial role in the representation of anticipated rewards. It increases activity during an exposure to a riskless stimulus (i.e., in a pure win situation).[44]

The PFC may be further divided into the DLPFC and the VMPFC, which both play a role in decision making under risk. The importance of the DLPFC and VMPFC was predominantly shown in the studies investigating the impaired behavior of lesion patients playing the IGT. Here, the DLPFC was important for manipulating and/or maintaining cognitive representations in working memory in order to better plan future behavior. The VMPFC was more associated with experienced than anticipated reward. However, as patients with lesions in that brain region fail to develop anticipatory SCRs to risky choices, a certain involvement of the VMPFC in the development of anticipatory responses was presumed as well.[12]

The amygdala is involved in emotion and learning. It is essential for the production of fear responses and crucial for the learning of associations between emotional stimuli and behavior (for review see Refs. 35–37). Again, studies by the Iowa group provide evidence that the amygdala is required to help build fearful anticipatory SCRs to losses.[4]

Regions involved in expected utility, which implies that an action has already resulted in a reward or punishment, should show differential activation during the experience of positive or negative feedback. Moreover, brain activity possibly varies depending on the magnitude and valence of the outcome. Studies show activation related to such processes in the striatum, the OFC, the VMPFC, and in the amygdala.

The striatum can be divided into its dorsal and ventral parts, and each part receives inputs from different structures. The ventral striatum is thought to get input from amygdala, hippocampus, and VMPFC, whereas the dorsal part of the striatum receives signals from the dorsal and lateral PFCs. This suggests different roles for dorsal and lateral areas in risky decision making. The ventral part, which includes nucleus accumbens and ventral putamen, is active during reward anticipation and experience. The dorsal part of the striatum, i.e., caudate nucleus and dorsal putamen, is central in the processing of experienced reward magnitude and valence.[44–47]

The VMPFC shows signal changes during reward and not so much during loss. Gambling tasks show that it plays a role in the choice between desirable stimuli. Patient studies suggest involvement in behavior of executive control rather than in representing value per se. The ACC seems sensitive to perception of negative outcomes, which is presumably influenced by the dopaminergic activity within this region.[44] The amygdala, however, seems to be specifically involved in the representation of negative outcomes, as previously shown by Bechara *et al*.[4]

In PT, the same brain process could be responsible for representing the value of gains and losses as the shape of the value function is assumed to be equal independently of the direction of the actual outcome. So far, separate systems for the representation of reward magnitude and the valence of an outcome were mostly suggested by neuroscientific data (see review by Lee[42]). There is also evidence that there are brain regions, like the striatum, that are involved in the processing of outcome valences as well as reward magnitude.[45–47]

In a situation that offers both potential gains and losses, PT assumes that the value function is steeper for losses than for gains, implying risk or loss aversion. Again, the ventral striatum may play an important role in signal integration of gain and loss representations. It has access to representations of positive and negative outcome values as it receives input from the amygdala, hippocampus, and PFC. As described in detail above, it has been shown in studies with patients suffering from lesions in the amygdala and/or in the VMPFC that these regions are essential for building up and "holding/keeping" a risk aversion. Nevertheless, it was also shown that, with task variations, the deficits are more or less present in particular patients. The role of the OFC was discussed as the region important for learning reward associations, suggesting that this, and not loss aversion, could be the mechanism responsible for the deficits found in patients.

The neural representations of OP are mostly described with the help of animal studies (see review by Trepel *et al.*[44]). When animals make choices between stimuli that promise different probable rewards, it was found that neurons in the lateral intraparietal area increase firing rates with growing probability of a positive outcome. Moreover, reward probability may also be reflected by the dopaminergic system, which was found to respond with varying firing rates in dependence of reward probabilities in human midbrains (regions central for dopamine reception) and monkeys.[48,49] Greatest dopaminergic activity was found during the conditions of highest uncertainty. This again speaks for the assumption that the dopaminergic system codes for the degree of risk associated with a decision.[44]

In accord with PT, the most important aspect of OP is that high probabilities are underweighted while low probabilities are overweighted. The result is a characteristic curve of a weighting function that stands for an overall willingness to entertain risk. The shape of the curve could emerge from diminishing sensitivity and also from emotional aspects within risky decision making. For example, underweighting high probabilities might reflect fear and overweighting low probabilities might reflect hope. Trepel *et al.*[44] hypothesize that fear could be represented by amygdala activation and hope would be reflected in activation of the ventral striatum. Additionally, they hypothesized that an impulsive acceptance of gambles, as found in some patients, would result in an increase in weighting function elevation. Hence, the individual differences in the elevation of a weighting function could be related to differences in the dopaminergic system as well as the serotonergic (5-HT) system, as both have been shown to be associated with individual levels of impulsivity.

Framing and editing operations are hypothesized by PT to be responsible for guiding decision making via shaping of representations of the prospects. In dual-process models of judgment and choice, researchers distinguish between two processing routes that can influence judgment and decision making. The one route would be spontaneous, associative, and affective and would, for example, use availability and/or affect heuristics to make choices. The other route would be rule based, cognitive, and reflected through dominance in choice and/or higher order reasoning in decision making.

Assuming a dual route within PT for the generation of judgment and choice might also contribute to the understanding of underlying brain areas. Passive framing and probability weighting may be processes belonging to the first system, which relies on limbic and basal ganglia regions. More controlled active framing and editing operations may reflect changes within the second system, incorporating regions of the lateral and dorsomedial PFCs.[50]

The neural substrate of framing effects becomes evident in studies focusing on reward perception in dependence of the reward context. It was shown that the ventral striatum and the VMPFC responded to outcomes more when the respective outcome was viewed as a gain or when it was presented in a gains-only gamble.[45] Editing processes involve controlled reformulations of prospects and hence should recruit memory and executive control processes, thereby relying on the DLPFC. Moreover, editing requires inhibition of fast automatic (and hence route one driven) responses, which is known to take place in the right lateral PFC and with the help of the dopaminergic system. Studies strengthen this assumption by showing that patients with lesions in the VMPFC fail to inhibit responses.

There is growing evidence regarding the neural representation of risky decision making. Models like the PT try to absorb the losses of pure rational economic models by introducing more flexible explanations for risk-seeking or risk-adverse behavior. Nonetheless, further work is needed to disentangle the exact contributions of the different aspects hypothesized by PT that are shaping the choices. Especially, the framing and editing effects have to be investigated in some more detail as well as the exact cognitive and affective processes that lead to the perception of an overall probability weight. So far, only assumptions or indirect knowledge gained through animal or patient studies are available. Moreover, it is unclear how people are thought to behave if the probabilities are unknown, i.e., if they are making *uncertain* decisions.

As discussed above, fMRI can only answer these questions with many subsequent studies. PT may be too complex a model to allow investigating neural representation of risky decision making in one fMRI experiment. Meaningful tasks have to be found that allow such designs. Those tasks have to be repeated in a block-wise manner to account for shorter cognitive events than those lasting 2–8 s. Risky decisions are among such cognitive events. As a consequence, we need to average the brain signal over many trials. In such studies, outcome depends on the rate with which stimuli are presented because a certain amount of one stimulus type is required to get a sensitive measure of one condition. If neural activity underlying certain stimuli is compared, the same should only differ in the exact matter of interest (as we build differential contrasts for comparisons), i.e., stimuli need to be matched. Without a control task (i.e., comparing activation to a resting state), fMRI data are even harder to interpret.

It is probably most challenging to find a suitable control condition that exclusively differs from the experimental task in one single aspect, i.e., the effect of interest. Unfortunately, the models to date are not constructed in that way. They usually describe a phenomenon and not how to control for it in an experiment.

Neuroeconomical Studies Focusing on Social Decision Making and Evaluative Judgment

Decisions made during social interaction, or when the outcome depends on the behavior of another person, are different from decisions made in isolation. Brain areas important in individual decision making are relevant in interactive decision making, too. Signal changes from interactive decision making in a trust game were found in the nucleus caudate.[51] Here, activation changed systematically depending on whether the player perceived an opponent's response as malevolent, neutral, or benevolent. As described in more detail above,[25] the activation in the anterior insula changes when an opponent's offer in the Ultimate Game is perceived as unfair. Brain activation in the anterior insula, ACC, and striatum also changes depending on whether or not subjects believe they are playing with a real partner or a computer. This indicates that aspects, other than reward, can bias decisions.[25,52]

Other decision-making strategies are learned through past experiences. People, when recruiting experiences to generate an evaluation of choice alternatives in a game, may update their own value functions or even their beliefs about other players' behavior.[51,53,54] There is little literature providing evidence for the neural substrate of interactive reinforcement learning (in the sense of revising utilities of certain choices of one self and others in order to perform better). Bhatt[55] investigated players' beliefs about their own and other players' beliefs and choices. Earnings from choices and accurate beliefs correlated negatively with activity in the insula and positively with ventral striatum activity. The same brain mechanisms could also contribute to judging risk, as people can come to different conclusions about the hazard of a certain risk resulting from learned experiences and beliefs. So far, there are not many studies investigating interactive risky decision making.

Evaluative judgment (when the decision maker has to evaluate alternatives before making a choice) plays a central role within everyday decision making. However, judgment and decision making depend on the same neural systems. Evaluative judgment can and should be investigated separately because judgments, for example in connection with attitude expression, are not restricted to decision making.

The neuronal basis for evaluative judgment was explicitly investigated by Zysset *et al*.[56] They define judgment as "the assessment of an external or internal stimulus on an internal scale." Thus, evaluative judgments represent a special type of judgment. Like memory retrieval tasks, evaluative judgments also depend on previously acquired knowledge and experienced events and these are closely related to a person's value system. Which internal scale an event is related to is, therefore, self referential.

In the study by Zysset and co-workers,[56] the evaluative process was isolated by comparing brain activation during evaluative judgment with brain activation during episodic and semantic memory retrieval. Anterior frontomedian cortex, inferior precuneus, and left inferior PFC were found as the underlying substrate for evaluative judgments. The anterior frontomedian cortex activation reflects metacontrol processes, and the activation of the inferior precuneus reflects episodic memory retrieval during evaluative judgments. The inferior frontal gyrus shows signal increase when people are selecting information among alternatives during an evaluative judgment. Furthermore, the results suggest a functional interdependence of the anterior frontomedian and the median parietal cortex, with the former being more activated by processes incorporating self-referential processes as well as self-initiation of subsequent cognitive processes. The latter (inferior precuneus) is thought to be more activated by processes incorporating episodic retrieval.

That aspects within evaluative judgment, for example if it is performed explicitly or implicitly, involve different neural networks is highlighted in a study by

Cunningham *et al.*[57] The authors found that, dependent on whether the task is explicit or implicit, different brain regions are important. For example, right OFC and temporal pole were associated with emotional intensity only during explicit evaluation. Inferior frontal/insular cortex activation correlated significantly with attitude valence not with emotion intensity. Nevertheless, activity in some areas was found to be task independent, suggesting some sort of automatic implicit processing by these areas. Activity in the amygdala correlated with emotional intensity, and activity in the right insula and the ACC correlated with valence in both tasks. A study by Vorhold *et al.*[58] provides evidence that, during an explicit risk rating task, the medial PFC, the inferior frontal gyrus, the cerebellum, and the amygdala are all activated.

Altogether, it is important to note that evaluative judgment is, like decision making, not a single process. It varies, for example, in the degree to which it is implicit (automatic, unconscious) or explicit (controlled, conscious). As a consequence, different brain mechanisms should also subserve different aspects within an evaluation judgment like emotional intensity, valence, memory recruitment, and the self-initiated processes to change and/or control an initial behavior. Usually, judgment features are not modelled or defined separately but are rather subsumed under decision-making properties.

Northoff *et al.*[59] try to disentangle the contributions of cognitive and affective components within decision making. Paradigmatically, these components are believed to be reflected in affective judgment consisting of a reaction to emotional stimulation and an evaluation requiring rational categorizations. Subsequently, affective judgment may influence decision making. It was found in healthy subjects that the brain signal in the VMPFC when performing affective judgments (with and without a preceding expectancy period) correlated with the subsequent performance in the IGT. This can be seen as evidence that VMPFC activity during affective judgment is crucial for beneficial decision making. Maybe the VMPFC holds the key role in the complex interplay between affective judgment and successful decision making.

Further, ideas for an explanation of the exact relationship between judgment and decision making are discussed.[59] Judging risk is an ability needed and used frequently within everyday life. Judgments occur even without direct decision making. It seems indispensable to consider the neural substrates for all of the displayed mechanisms when generating ideas about candidate regions active in risk perception.

Taken together, theories assuming solely rational behavior cannot account for complex real-life decision making. It is becoming clearer that affect plays an important role in risky choice.[60] Models of emotion-based choices can help to enrich these rational theories. Joining different ideas, models, or even disciplines together helps to draw conclusions about underlying neuronal processes in more complex real-life behavior. Risk perception, risky decision making, and risk judgments are such complex behaviors. fMRI can contribute to the understanding of the underlying neuronal substrate of risky choice. Despite this, some important restrictions have been outlined above that have to be considered when using fMRI to interpret these phenomena.

Conflicts of Interest

The author declares no conflicts of interest.

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