

Research article

Right thalamic volume mediates impact of the dopamine beta-hydroxylase gene on the endowment effect

Tao Wang^{a,1}, Jianmin Zeng^{a,*}, Weijie Huang^{b,c}, Xiong Xiong^c, Li Su^{c,d,**}

^a Sino-Britain Centre for Cognition and Ageing Research, Faculty of Psychology, Southwest University, Beibei District, Chongqing 400715, China

^b State Key Laboratory of Cognitive Neuroscience and Learning, Beijing Normal University, Beijing 100875, China

^c Department of Neuroscience, Neuroscience Institute, Insigneo Institute for in silico Medicine, University of Sheffield, Sheffield S10 2HQ, United Kingdom

^d Department of Psychiatry, School of Clinical Medicine, University of Cambridge, Cambridge CB2 0SZ, United Kingdom

ARTICLE INFO

Keywords:

Decision-making
Behavioral economics
Neuroeconomics
Decision neuroscience
Genetic neural imaging

ABSTRACT

The endowment effect is a tendency that individuals overvalue items belonging to them relative to those items that do not. Previous studies showed a strong relation between the dopamine beta-hydroxylase (DBH) gene and the endowment effect (EE), and a link between EE and task-based *functional* MRI activation in multiple brain regions. However, the role of brain structure on EE remains unclear. In this study, we have explored whether regional brain volume mediate the effect of the DBH gene on EE. Results showed that rs1611115, single-nucleotide polymorphisms (SNPs) at DBH loci, were significantly associated with right thalamus volume and the endowment effect in males but not in female participants. Specifically, male DBH rs1611115 T-carriers had larger right thalamus volume compared to carriers of CC genotype and exhibited a greater endowment effect. Importantly, we found that right thalamus volume mediated the effect of rs1611115 on the endowment effect in male participants. This study demonstrated how thalamic volume plays an important mediating role between genetics and decision-making in humans.

1. Introduction

The endowment effect, a term coined by Thaler [1], refers to a tendency that individuals overvalue items that belong to themselves relative to items that do not. The effect is not confined to personal possessions, demonstrating in various other situations, such as intellectual property, time, public land, and environmental, health, and safety regulations [2–4]. It also has been found in some animal behaviors [5,6], suggesting that it could partly originate from fundamental organization of the brain. Several theories have been proposed to explain this effect, such as loss aversion [7,8], psychological ownership [9], biased information processing [10], and so on [11].

Individual behaviors are to some extent influenced by the genes and the brain. These factors might also affect inherent human biases, such as the endowment effect. Previous neuroimaging studies have demonstrated that the endowment effect is associated with activity in a neural network that includes the amygdala [12], dorsal striatum [13], medial

prefrontal cortex [14–16], insula [14,15], nucleus accumbens [14], and inferior frontal gyrus [17,18], many of which are components of the reward network. However, there is currently no research exploring the relationship between the endowment effect and brain structure using Voxel-based morphometry. Brain structures, particularly cortical thickness and subcortical structure volume, are closely related to cognitive functions and emotional regulation [19,20]. In brain regions involved in value-based decision-making, such as the prefrontal cortex and the amygdala, structural differences have been found to significantly correlate with decision-making behaviors [21]. Therefore, the structural features of these specific brain regions may regulate the endowment effect.

The DBH gene, which encodes dopamine β -hydroxylase, is a key catalyst in the conversion from dopamine to norepinephrine, extensively influencing neurotransmission and behavior [22]. Both DA and NE play a crucial role in the brain's reward system [23–25]. Research has shown that single nucleotide polymorphism (SNPs), specifically rs1611115,

* Corresponding author.

** Corresponding author at: Department of Neuroscience, Neuroscience Institute, Insigneo Institute for in silico Medicine, University of Sheffield, Sheffield S10 2HQ, United Kingdom.

E-mail addresses: james_psych2@yeah.net (J. Zeng), l.su@sheffield.ac.uk (L. Su).

¹ These authors contributed equally and thus are co-first authors.

influence the transcription of the DBH gene, accounting for up to 50 % of the total variation in DBH activity [26]. Besides, This gene is associated with the brain's reward system [27] and the endowment effect [28]. Neuroimaging studies have shown that variations in gene expression can affect brain structure, thereby relating to cognitive and emotional functions [29]. Therefore, we further hypothesize that the DBH gene may regulate the endowment effect by influencing the structural features of specific brain regions.

Up to now, it remains unclear whether the brain structure plays a role of mediation in the influence of DBH gene (rs1611115) on the endowment effect. This study tried to address this issue. Based on previous research, we propose the following hypotheses: a) The DBH gene is associated with the endowment effect; b) The cortical thickness or subcortical structure volume of specific brain regions is related to the endowment effect; c) The structural features of these brain regions may mediate the impact of the DBH gene on the endowment effect.

2. Methods and materials

2.1. Subjects

We recruited 120 healthy undergraduate students (73 females; Mean age = 20.63 (SD = 0.91)) from the Southwest University in Chongqing, China. This was a part of a gene-brain-cognition project. All participants provided written informed consent, and were compensated for their time. The study was approved by the Ethics Committee of Psychological Research in SWU.

2.2. Materials and procedure

This research comprises three primary components: genotype identification, behavioral experiments, and magnetic resonance imaging (MRI). Initially, saliva samples are collected from participants and sent to a genetic testing company for genotype determination. Subsequently, behavioral experiments are conducted, during which participants' behavioral data is collected to compute relevant metrics, such as the endowment effect. Finally, participants undergo resting-state MRI scans, after which data is collected and used to calculate cortical thickness and

subcortical tissue volumes. The research workflow is illustrated in Fig. 1.

2.3. Genotyping

The genotypes of rs1611115 were determined by the Mass Array system (Agena iPLEX assay, San Diego, United States). First, approximately 10–20 ng of genomic DNA was isolated from saliva samples. The polymerize chain reaction (PCR) primers used in the study were: ACGTTGGATGAAGCAGAATGTCCTGAAGGC and ACGTTGGATGTCAGTCTCACCACGGCACCT. The sample DNA was amplified by a multiplex PCR reaction, then the obtained products were used for locus-specific single-base extension reaction. Unextended primers used in the study were GTACTCCTGTCCTCTCCC. At last, the resulting products were desalted and transferred to a 384 element SpectroCHIP array. The alleles were discriminated by mass spectrometry (Agena, San Diego, United States). rs1611115 genotype was coded as a categorical variable (C/C, C/T and T/T) for the subsequent analysis.

2.4. Experiment task

The behavioral task measuring the endowment effect consisted of 3 stages: instruction, practice, and formal experiment. Participants could repeat the instruction and practice as many times as they wish until they fully understood the task before entering the formal experiment.

The formal experiment had the following design: 2 roles (buyer vs seller) \times 11 probabilities (1 %, 10 %, 20 %, 30 %, 40 %, 50 %, 60 %, 70 %, 80 %, 90 %, 99 %) of winning 1000 yuan (approximately US\$150 at the time of the test). The role variable was our focus. Totally, we got 22 trials. The order of these 22 trials was randomized for each participant respectively.

Each trial involved 6 questions about the same lottery but different prices, which is automatically generated by the computer according to predefined rules. Here is an example of the buyer condition. Each participant needed to indicate whether he would like to buy a lottery of "50 % to get 1000 yuan" at a price of X. The first price was set as the expected value of the lottery in that trial (500 yuan in this example). If the participant rejected this price of 500 yuan (did not want to buy it at that price), then the next price was calculated as the average of the 500

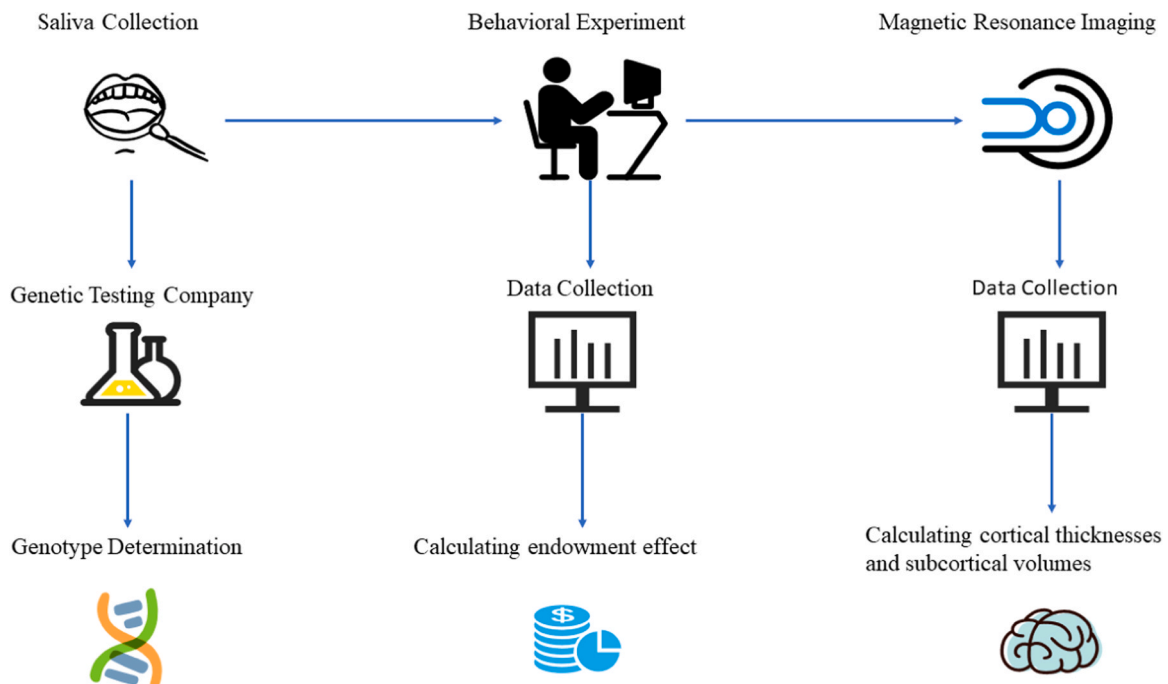


Fig. 1. The workflow of the current research.

yuan and 0 yuan, that is 250 yuan. If the participant accepted this price of 500 yuan (wanted to buy it at that price), then the next price was the average of 500 yuan and 1000 yuan, that is 750 yuan. This probing process would iterate 6 times, that is, the participants needed to indicate whether he would like to buy it at 6 different prices respectively. The next price was always the average of the updated highest accepted price and lowest rejected price. The initial accepted price and rejected price were set as 0 and 1000 yuan for buying trials and 1000 and 0 yuan for selling trials, respectively. The non-integer amounts were always rounded into integers. After these 6 decisions, we got his final highest accepted price (say 250 yuan) and lowest rejected price (say 266 yuan). We averaged these two prices to get the equivalent price (say 258 yuan) of this lottery for this participant in the buying condition.

Then, we averaged the equivalent prices of all lotteries in buying condition and selling condition respectively to get averaged buying price (willingness to pay, WTP) and averaged selling price (willingness to accept, WTA) for each participant. The WTA – WTP gap was used to measure the endowment effect.

2.5. Image acquisition and processing

After completing the behavioural experiment, on a separate visit, participants undergone T1 weighted structural MR imaging in South-west University using a Siemens Verio 3 T MRI scanner (MPRAGE, 160 slices, voxel size 1.0mm³, TR=2300 ms, TE=2.98 ms, FA=9°).

To calculate cortical thickness and subcortical volume, structural MRI images were processed with Freesurfer v5.3 (<http://surfer.nmr.mgh.harvard.edu/>). Specifically, this processing included motion correction and average [30] of multiple volumetric T1 weighted images, removing non-brain tissue with a hybrid watershed/surface deformation procedure [31], automated Talairach transformation, segmentation of the subcortical white matter and deep gray matter volumetric structures (including the hippocampus, amygdala, caudate, putamen, ventricles) [32,33] intensity normalization [34], tessellation of the gray/white matter boundary, automated topology correction [35,36] and surface deformation following intensity gradients to optimally place the gray/white and gray/cerebrospinal fluid borders at the location where the greatest shift in intensity defines the transition to the other tissue class [37–39].

Once the cortical models were completed, we parcellated the whole-brain MRI into 34 regions per hemisphere according to the Desikan-Killiany atlas [40]. Thickness was measured by the average distances in a region between the white matter and pial surfaces. In the end, we got 7 subcortical structures and 34 cortical thicknesses per hemisphere for the following analysis.

2.6. Statistics

Firstly, a paired t-test with role (buyer vs seller) as independent variable and the averaged equivalent price as dependent variable, was used to test the endowment effect for males and females respectively, in consideration of the gender difference in the endowment effect [41].

Then, analysis of covariance (ANCOVA) with EE as dependent variable, genotype of SNP rs1611115 as independent variable and age as covariate, was used to explore the effect of rs1611115 on EE for males and females respectively, in consideration of the gender difference in the impact of DBH gene on the cognitive performance [42,43]. The Pearson's correlation, with the false discovery rate (FDR) correction, was used to explore the relation between EE and brain structures (cortical thickness and subcortical structure volume) for males and females respectively. The brain regions that passed FDR correction were used for the subsequent analysis including ANCOVA to test the impact of rs1611115 on these brain regions.

For mediation analysis, we applied the mediation analysis (model = 4, bootstrap = 5000) with the dependent variable being EE, the independent variable being SNP rs1611115, and the mediator being the

brain regions surviving the FDR correction while controlling for age.

All statistical analysis were conducted in the R environment.

3. Results

3.1. Genetic distributions

Among 120 participants, 80 (34 males and 46 females) were C allele homozygotes (CC), and 40 (13 males and 27 females) were heterozygotes (TC). There were no homozygotes of the T allele (TT) in the current cohort.

3.2. Behavioral endowment effect

Using paired t-test, we found that the average equivalent price for selling was significantly higher than that for buying in males ($t(46) = 3.245$, $p < 0.01$, Cohen's $d = 0.473$), as well as in females ($t(72) = 5.976$, $p < 0.001$, Cohen's $d = 0.699$). See Fig. 2 for details.

3.3. The effect of rs1611115 on the endowment effect

To test the effect of rs1611115 on the endowment effect in males and females, we performed an analysis of covariance (ANCOVA) with the dependent variable being the endowment effect, the independent variable being SNP rs1611115, and the covariate being age for males and females respectively. We found a significant difference in the endowment effect between CC and TC only in males: $F(1,46) = 6.096$, $p = 0.018$, $\eta^2 = 0.122$; male T-carriers exhibited a greater endowment effect than males of the CC genotype. There were no significant results in females. For details, see Fig. 3A.

3.4. The effect of brain structures on the endowment effect

To explore the relationship between the endowment and brain structures in males and females. We separately calculated the Pearson's correlation between the endowment effect and cortical thickness, and the correlation between the endowment effect and subcortical volumes normalized by the total intracranial volume. Correction for multiple comparisons were conducted using the false discovery rate (FDR) correction.

All results for cortical thickness did not survive correction for multiple comparisons in either males or females. For subcortical structures, only the right thalamus volume in males, but not in females, was significantly associated with the endowment effect after FDR correction ($r = 0.4266$, FDR-corrected p value = 0.039) as shown in Table 1.

3.4.1. The effect of SNP rs1611115 on right thalamus volume

As shown in the analysis above, only right thalamus volume was significantly correlated with the endowment effect in males. So, we focused on exploring the effect of SNP rs1611115 on right thalamus volume in males (and females for completeness) and showed the results in Fig. 3B. We then performed ANCOVA analysis with the dependent variable being right thalamus volume and the independent variable being SNP rs1611115 and controlling for age, for males and females separately. Results showed that the volume of the right thalamus in T-carriers was significantly larger than C-homozygotes in males ($F(1,46) = 9.271$, $p = 0.004$, $\eta^2 = 0.172$). There was no significant effect in females.

3.4.2. Mediation analysis

To explore the three-way relation among DBH gene, brain structures, and the endowment effect in males and females, we performed the mediation analysis (model = 4, bootstrap = 5000) with the dependent variable being the endowment effect, the independent variable being SNP rs1611115, and the mediator being right thalamic volume while controlling for age. In males, a significant indirect mediation effect of

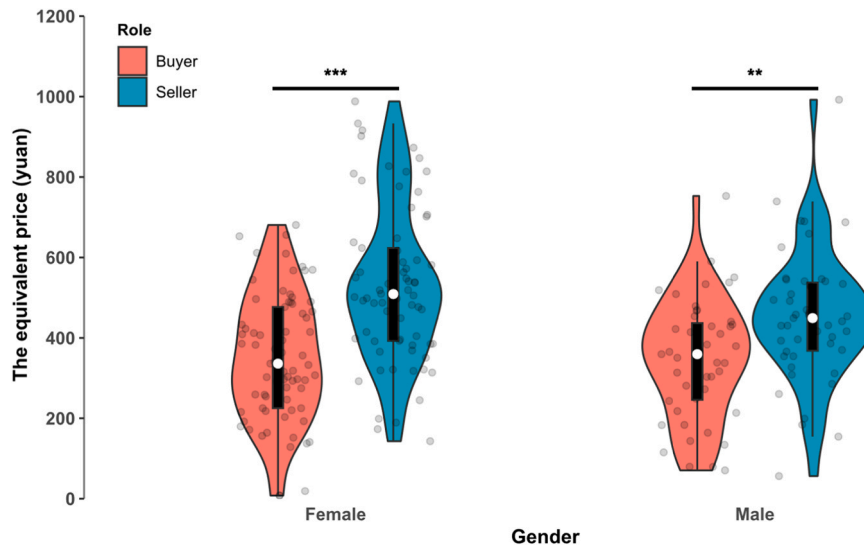


Fig. 2. The average equivalent price for each role. The violin plot shows the data distribution. The box plot illustrates the position parameters of the data (i.e., medians, quartiles). The jitter plot shows the location of each data point. ** represents $p < 0.01$; *** represents $p < 0.001$.

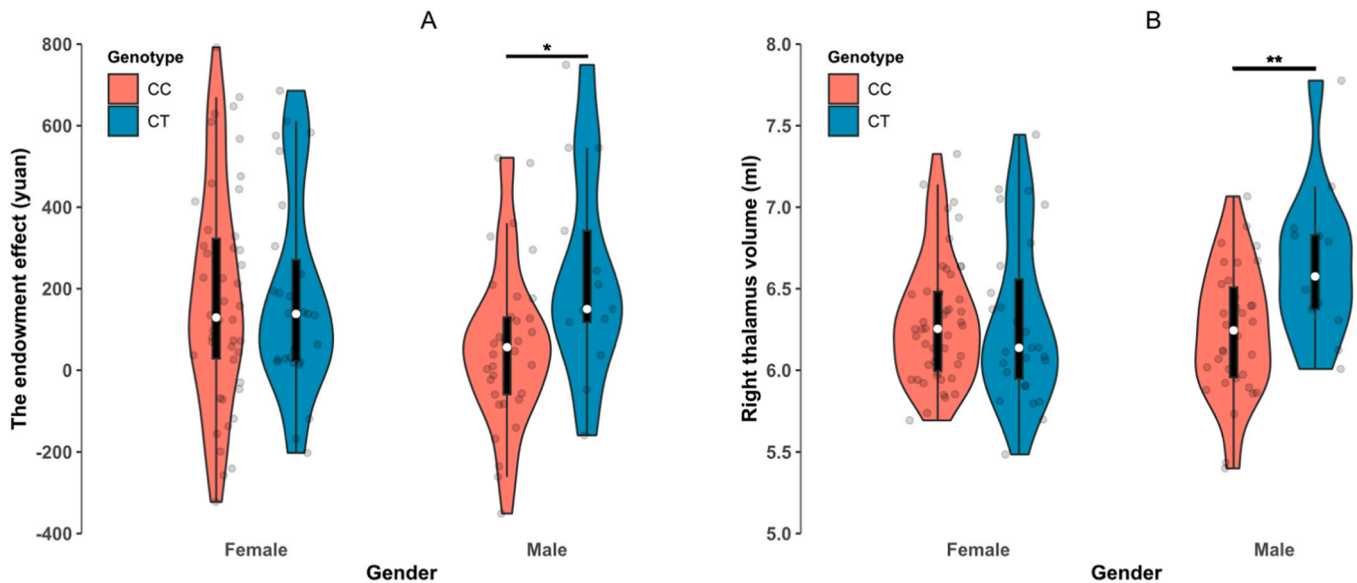


Fig. 3. The impact of genotype of rs161115 on the endowment effect (3 A) and right thalamus volume (3B). The violin plot shows the data distribution. The box plot illustrates the position parameters of the data (i.e., median, quartiles). The jitter plot shows the location of each data point. * represents $p < 0.05$; ** represents $p < 0.01$.

rs161115 was observed on the endowment effect mediated by the right thalamus volume ($\beta = 71.463$, 95 % confidence interval (CI): [12.355, 150.079], $p = .044$), but the direct effect was not significant, indicating a complete mediation effect. In addition, the mediator - right thalamus volume, could account for approximately 41.16 % of the total effect of rs161115 on the endowment effect (Fig. 4). In females, we did not find a significant mediation effect for rs161115.

4. Discussion

Consistent with previous research, we found a significant endowment effect for both males and females in our behavioral task, that is, selling prices (WTA) were higher than buying prices (WTP) for the same items. We found that male T-carriers had larger right thalamic volume compared to males with CC genotype, and they also exhibited a stronger endowment effect than non-carriers. Moreover, we found that right

thalamus volume mediates the effect of SNP rs161115 on the endowment effect in males but not in females.

4.1. The DBH gene and the endowment effect

Research has found that the DBH gene, namely SNPs rs161115, is associated with the endowment effect, with T-carriers exhibiting a stronger endowment effect than non-carriers [28]. In this study, both males and females also exhibited the endowment effect. Nevertheless, male T-carriers showed a stronger endowment effect than the other males, while this phenomenon was not observed in females. This gender difference may be related to estrogen level. Studies have shown that estrogen increases the expression of the DBH gene, accelerating the conversion of DA to NE [44]. Both DA and NE play important roles in reward-related cognitive activities [23,24,45]. Cognitive function performance is usually related to the conversion rate and level of

Table 1
Correlations between the subcortical volume and the endowment effect.

| | correlation | Standard error | t value | p value | FDR |
|-------------------|-------------|----------------|---------|---------|---------------|
| Left Thalamus | 0.0705 | 0.1487 | 0.4738 | 0.6379 | 0.8119 |
| Left Caudate | -0.1739 | 0.1468 | -1.1843 | 0.2425 | 0.6790 |
| Left Putamen | -0.0856 | 0.1485 | -0.5765 | 0.5672 | 0.7940 |
| Left Pallidum | 0.0259 | 0.1490 | 0.1736 | 0.8630 | 0.9242 |
| Left Hippocampus | 0.2386 | 0.1448 | 1.6485 | 0.1062 | 0.3718 |
| Left Amygdala | 0.1399 | 0.1476 | 0.9478 | 0.3483 | 0.6956 |
| Left Accumbens | -0.1226 | 0.1479 | -0.8288 | 0.4116 | 0.6956 |
| Right Thalamus | 0.4266 | 0.1348 | 3.1643 | 0.0028 | 0.0390 |
| Right Caudate | -0.2490 | 0.1444 | -1.7249 | 0.0914 | 0.3718 |
| Right Putamen | 0.0288 | 0.1490 | 0.1931 | 0.8478 | 0.9242 |
| Right Pallidum | -0.0143 | 0.1491 | -0.0957 | 0.9242 | 0.9242 |
| Right Hippocampus | 0.3430 | 0.1400 | 2.4493 | 0.0183 | 0.1279 |
| Right Amygdala | 0.1136 | 0.1481 | 0.7669 | 0.4472 | 0.6956 |
| Right Accumbens | -0.1181 | 0.1480 | -0.7975 | 0.4294 | 0.6956 |

neurotransmitters in the body. The conversion rate of neurotransmitters is sometimes regulated by genes [46], such as dopamine. Certain SNPs are associated with more effective DA transmission and consequently better motor and cognitive performance, while other SNPs are associated with depressed DA functioning and resulting impairments in motor and cognitive behaviors [47–49]. The gender differences of the polymorphism of the DA gene on cognitive and behavioral performance have been observed [50–52]. Regarding the SNPs rs1611115, some studies have also pointed out gender differences in cognition [51] and behavior [43]. Previous studies have found significant gender differences in the behavioral studies of the endowment effect, where males are willing to pay higher prices and exhibit lower risk aversion [53]. We hypothesize that estrogen, by enhancing the expression of the DBH gene, increases the conversion rate of dopamine (DA) to norepinephrine (NE) to a similar ceiling level for the two genotypes, thereby resulting in comparable levels of DA across different genotypes in females. Consequently, the endowment effects in female T-carriers are similar to those in females with the CC genotype. In males, however, the expression of the DBH gene in T-carriers is lower than in those with the CC genotype, resulting in higher levels of DA in T-carriers, and thereby their endowment effects surpass those of the CC genotype males. Nevertheless, this hypothesis still requires validation through future research.

4.2. Thalamus and the endowment effect

The thalamus is a multifunctional structure, often regarded as a relay station or hub, responsible for transmitting information between subcortical regions and the cerebral cortex [54]. The current research revealed a significant correlation between the right thalamic volume and the endowment effect in males. Specifically, male T-carriers have a larger right thalamic volume and exhibit a stronger endowment effect. Studies have shown that the thalamic volume is related to cognitive performance [55–57], as well as loss aversion [58], the latter of which is commonly used to account for the endowment effect [7,8]. The thalamus in primates is a crucial target for brain dopamine and is part of a novel dopaminergic system that specifically targets the primate thalamus [59]. This thalamic dopaminergic system, independent of the traditional mesocortical and mesolimbic dopaminergic systems, may play a significant role in higher brain functions and conditions like schizophrenia [59,60]. Additionally, the thalamus is rich in norepinephrine (NE), displaying a pronounced lateral distribution [61–63]. Similar to dopamine, NE is also associated with the reward system [64, 65] and loss aversion [66,67]. Although there are no significant differences in thalamic volumes between males and female [68], notable gender differences have been observed in the microstructure of the thalamus [69] and in its functional connectivity density [70]. The aforementioned findings shed light on the gender differences and lateralization phenomena in the relationship between the thalamus and the endowment effect, yet deeper underlying mechanisms warrant further exploration.

The endowment effect, prevalent in daily life, often leads to irrational decision-making. Therefore, interventions targeting the right thalamus to improve decision-making behaviors warrant attention. Several interventions can be taken to enhance individuals' decision-making abilities. Firstly, cognitive training programs can enhance individuals' ability to process reward information. For instance, Balleine and O'Doherty [71] showed that reward information processing is closely related to the activity of specific brain regions, including the thalamus. Targeted cognitive training can help individuals evaluate rewards more rationally, influencing the activity of the right thalamus and reducing the endowment effect on decision-making. Additionally, neuro-modulation techniques like transcranial direct current stimulation (tDCS) and transcranial magnetic stimulation (TMS) can directly modulate brain region activity. Knoch, Gianotti [72] found that applying TMS to modulate prefrontal cortex activity can alter individuals' risk decision-making behavior. Similarly, Fecteau, Knoch

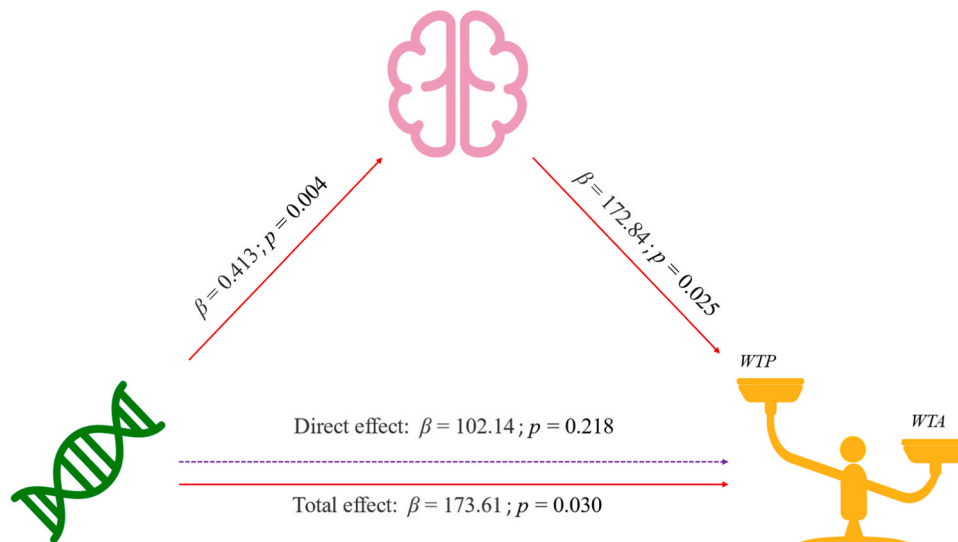


Fig. 4. Results of the mediation analysis. β : regression coefficient; p : p-value of a regression coefficient.

[73] showed that using tDCS to modulate prefrontal cortex activity can reduce individuals' risk-taking behavior. These studies suggest that directly modulating decision-making brain regions, like the right thalamus, can effectively rationalize decision-making behavior. It is important to note that the long-term effects and safety of these interventions require further research and validation. Furthermore, the design and implementation of interventions should take into account individual differences and specific circumstances.

4.3. The DBH gene, thalamus, and the endowment effect

The currently study observed that the right thalamic volume mediates the impact of SNP rs1611115 on the endowment effect in males, suggesting that an individual's valuation of goods is influenced not only by genetic factors but also by brain structural characteristics.

SNP rs1611115 is located on the DBH gene, affecting the level of dopamine. Dopamine is an important neurotransmitter associated with various psychological processes such as reward, motivation, and cognition [74]. Therefore, different alleles of rs1611115 may influence the endowment effect by affecting dopamine levels. Moreover, the right thalamus plays a significant role in processing spatial, emotional, and reward information [75]. Our results suggest that differences in the volume of the right thalamus may modulate the impact of dopamine on the endowment effect. This finding is consistent with previous research, such as the studies by Camerer [76] and Pessiglione, Seymour [77], which indicate that variations in the dopamine system are related to the endowment effect. Furthermore, neuroimaging studies have also found that the thalamus plays an important role in reward processing and value evaluation [78–80].

The results of this study provide new insights into the neurobiological basis of the endowment effect and offer a new perspective on exploring the role of individual differences in economic decision-making.

5. Limitations

The limitations of this study are as follows: 1) this study identified gender differences in the manifestation of the endowment effect among different genotypes of the DBH gene (rs1611115), but it did not empirically investigate the reasons behind these gender differences. Future research could further explore this topic to understand the underlying factors contributing to gender disparities; 2) the study also found that the volume of the right thalamus in males influences the endowment effect and mediates the impact of the DBH gene on this effect. However, there is still a lack of exploration into the underlying mechanisms involved in this mediation. Further research is needed to gain a deeper understanding of these mechanisms; 3) the number of individuals carrying the T allele in males was relatively small, which is related to the genotype distribution of this gene locus in the population. Future studies should verify these findings by utilizing larger sample sizes to obtain more reliable conclusions.

In summary, we discovered that right thalamus volume mediates the effect of SNP rs1611115 on the endowment effect in males.

Significance statement

Adaptive behavior in human through our live course is a direct result of the brain, but emerging evidence suggests that genes also introduce bias in behavior. Here, we showed that right thalamus volume mediated the effect of a dopamine-related genetic locus on a decision tendency named endowment effect in males. This discovery reveals a novel mechanism for decision-making, aiding our understanding of the complex relations among genes, the brain, and behavior. This research underscores the significant role of genetic factors in shaping individual behaviors and traits, providing a fresh perspective for the fields of psychology and neuroscience.

Funding source

JZ is funded by China Ministry of Education's Humanity and Social Sciences Project (23YJA190001). LS is funded by Alzheimer's Research UK Senior Research Fellowship (ARUK-SRF2017B-1) and NIHR Sheffield Biomedical Research Centre.

CRediT authorship contribution statement

Tao Wang: Software, Data analysis, Writing – original draft, Writing – review & editing. **Jianmin Zeng:** Conceptualization, Experimental programming, Data collection, Writing – review & editing, Supervision, Funding acquisition. **Wei jie Huang:** Data analysis. **Xiong Xiong:** Data analysis. **Li Su:** Conceptualization, Writing – review & editing, Supervision, Funding acquisition.

Conflict of interest

There are no interests or activities for myself or my coauthors that might be seen as influencing the research.

Data availability

Data will be made available on request.

Acknowledgements

Gratitude is extended to all collaborators for their sincere support and contributions.

References

- [1] R. Thaler, Toward a positive theory of consumer choice, *J. Econ. Behav. Organ.* 1 (1980) 39–60, [https://doi.org/10.1016/0167-2681\(80\)90051-7](https://doi.org/10.1016/0167-2681(80)90051-7).
- [2] K.H. John, E.M. Kenneth, A review of WTA/WTP studies, *J. Environ. Econ. Manag.* 44 (2002) 426–447, <https://doi.org/10.1006/jeem.2001.1215>.
- [3] C. Buccafusco, C. Sprigman, Valuing intellectual property: an experiment, *Cornell Law Rev.* 96 (2010) 1–45.
- [4] R.C. Bishop, T.A. Heberlein, Measuring values of extramarket goods: are indirect measures biased? *Am. J. Agric. Econ.* 61 (1979) 926–930, <https://doi.org/10.2307/3180348>.
- [5] S.F. Brosnan, et al., Endowment effects in chimpanzees, *Curr. Biol.* 17 (2007) 1704–1707, <https://doi.org/10.1016/j.cub.2007.08.059>.
- [6] V. Lakshminarayanan, M.K. Chen, L.R. Santos, Endowment effect in capuchin monkeys, *Philos. Trans. R. Soc. B-Biol. Sci.* 363 (2008) 3837–3844, <https://doi.org/10.1098/rstb.2008.0149>.
- [7] D. Kahneman, A. Tversky, Prospect theory: an analysis of decision under risk, *Econometrica* 47 (1979) 263–291, <https://doi.org/10.2307/1914185>.
- [8] D. Kahneman, J.L. Knetsch, R.H. Thaler, Anomalies - the endowment effect, loss aversion, and status-quo bias, *J. Econ. Perspect.* 5 (1991) 193–206, <https://doi.org/10.1257/jep.5.1.193>.
- [9] J. Reb, T. Connolly, Possession, feelings of ownership and the endowment effect, *Judgm. Decis. Mak. J.* 2 (2007) 107–114.
- [10] N.J.S. Ashby, S. Dickert, A. Gloeckner, Focusing on what you own: biased information uptake due to ownership, *Judgm. Decis. Mak.* 7 (2012) 254–267.
- [11] C.K. Morewedge, C.E. Giblin, Explanations of the endowment effect: an integrative review, *Trends Cogn. Sci.* 19 (2015) 339–348, <https://doi.org/10.1016/j.tics.2015.04.004>.
- [12] B. Weber, et al., Neural evidence for Reference-dependence in real-market-transactions, *Neuroimage* 35 (2007) 441–447, <https://doi.org/10.1016/j.neuroimage.2006.11.034>.
- [13] B. De Martino, et al., The neurobiology of reference-dependent value computation, *J. Neurosci.* 29 (2009) 3833–3842, <https://doi.org/10.1523/jneurosci.4832-08.2009>.
- [14] B. Knutson, et al., Neural antecedents of the endowment effect, *Neuron* 58 (2008) 814–822, <https://doi.org/10.1016/j.neuron.2008.05.018>.
- [15] T. Feng, W. Zhao, G.F. Donnan, The endowment effect can extend from self to mother: evidence from an fMRI study, *Behav. Brain Res.* 248 (2013) 74–79, <https://doi.org/10.1016/j.bbr.2013.04.005>.
- [16] W. Guo, et al., Modulating the activity of MPFC With tDCS alters endowment effect, *Front Behav. Neurosci.* 13 (2019) 211, <https://doi.org/10.3389/fnbeh.2019.00211>.
- [17] M. Votinov, et al., The neural correlates of endowment effect without economic transaction, *Neurosci. Res.* 68 (2010) 59–65, <https://doi.org/10.1016/j.neures.2010.05.006>.

- [18] M. Votinov, et al., Transcranial direct current stimulation changes human endowment effect, *Neurosci. Res.* 76 (2013) 251–256, <https://doi.org/10.1016/j.neures.2013.05.007>.
- [19] A.M. Fjell, et al., High-expanding cortical regions in human development and evolution are related to higher intellectual abilities, *Cereb. Cortex* 25 (2015) 26–34, <https://doi.org/10.1093/cercor/bht201>.
- [20] R. Kanai, G. Rees, OPINION The structural basis of inter-individual differences in human behaviour and cognition, *Nat. Rev. Neurosci.* 12 (2011) 231–242, <https://doi.org/10.1038/nrn3000>.
- [21] L. Clark, et al., Differential effects of insular and ventromedial prefrontal cortex lesions on risky decision-making, *Brain* 131 (2008) 1311–1322, <https://doi.org/10.1093/brain/awn066>.
- [22] J.F. Cubells, C.P. Zabetian, Human genetics of plasma dopamine β -hydroxylase activity: applications to research in psychiatry and neurology, *Psychopharmacology* 174 (2004) 463–476, <https://doi.org/10.1007/s00213-004-1840-8>.
- [23] R.A. Wise, P.P. Rompre, Brain dopamine and reward, *Annu. Rev. Psychol.* 40 (1989) 191–225, <https://doi.org/10.1146/annurev.ps.40.020189.001203>.
- [24] B. Poschel, F. Ninteman, Norepinephrine: a possible excitatory neurohormone of the reward system, *Life Sci.* 2 (1963) 782–788.
- [25] L. STEIN, Self-stimulation of the brain and the central stimulant action of amphetamine, *Federation proceedings* (1964).
- [26] C.P. Zabetian, et al., A quantitative-trait analysis of human plasma-dopamine beta-hydroxylase activity: evidence for a major functional polymorphism at the DBH locus, *Am. J. Hum. Genet.* 68 (2001) 515–522, <https://doi.org/10.1086/318198>.
- [27] T. Plieger, et al., Association between a functional polymorphism on the dopamine- β -hydroxylase gene and reward dependence in two independent samples, *Personal. Individ. Differ.* 121 (2018) 218–222, <https://doi.org/10.1016/j.paid.2017.05.050>.
- [28] X.R. Hou, et al., The endowment effect in the genes: an exploratory study, *Judgm. Decis. Mak.* 14 (2019) 293–298.
- [29] L. Pezawas, et al., 5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: a genetic susceptibility mechanism for depression, *Nat. Neurosci.* 8 (2005) 828–834, <https://doi.org/10.1038/nm1463>.
- [30] M. Reuter, H.D. Rosas, B. Fischl, Highly accurate inverse consistent registration: a robust approach, *Neuroimage* 53 (2010) 1181–1196, <https://doi.org/10.1016/j.neuroimage.2010.07.020>.
- [31] F. Ségonne, et al., A hybrid approach to the skull stripping problem in MRI, *Neuroimage* 22 (2004) 1060–1075, <https://doi.org/10.1016/j.neuroimage.2004.03.032>.
- [32] B. Fischl, et al., Whole brain segmentation: automated labeling of neuroanatomical structures in the human brain, *Neuron* 33 (2002) 341–355, [https://doi.org/10.1016/s0896-6273\(02\)00569-x](https://doi.org/10.1016/s0896-6273(02)00569-x).
- [33] B. Fischl, et al., Sequence-independent segmentation of magnetic resonance images, *Neuroimage* 23 (Suppl 1) (2004) S69–S84, <https://doi.org/10.1016/j.neuroimage.2004.07.016>.
- [34] J.G. Sled, A.P. Zijdenbos, A.C. Evans, A nonparametric method for automatic correction of intensity nonuniformity in MRI data, *IEEE Trans. Med Imaging* 17 (1998) 87–97, <https://doi.org/10.1109/42.668698>.
- [35] B. Fischl, A. Liu, A.M. Dale, Automated manifold surgery: constructing geometrically accurate and topologically correct models of the human cerebral cortex, *IEEE Trans. Med Imaging* 20 (2001) 70–80, <https://doi.org/10.1109/42.906426>.
- [36] F. Ségonne, J. Pacheco, B. Fischl, Geometrically accurate topology-correction of cortical surfaces using nonseparating loops, *IEEE Trans. Med Imaging* 26 (2007) 518–529, <https://doi.org/10.1109/tmi.2006.887364>.
- [37] A.M. Dale, B. Fischl, M.I. Sereno, Cortical surface-based analysis. I. Segmentation and surface reconstruction, *Neuroimage* 9 (1999) 179–194, <https://doi.org/10.1006/nimg.1998.0395>.
- [38] A.M. Dale, M.I. Sereno, Improved localization of cortical activity by combining EEG and MEG with MRI cortical surface reconstruction: a linear approach, *J. Cogn. Neurosci.* 5 (1993) 162–176, <https://doi.org/10.1162/jocn.1993.5.2.162>.
- [39] B. Fischl, A.M. Dale, Measuring the thickness of the human cerebral cortex from magnetic resonance images, *Proc. Natl. Acad. Sci. USA* 97 (2000) 11050–11055, <https://doi.org/10.1073/pnas.200033797>.
- [40] B. Alexander, et al., Desikan-Killiany-Tourville Atlas compatible version of M-CRIB neonatal parcellated whole brain atlas: the M-CRIB 2.0, *Front. Neurosci.* 13 (2019) 34, <https://doi.org/10.3389/fnins.2019.00034>.
- [41] A. Wieland, et al., Gender differences in the endowment effect: women pay less, but won't accept less, *Judgm. Decis. Mak.* 9 (2014) 558–571, <https://doi.org/10.1017/S1930297500006422>.
- [42] K.E. Hupfeld, D.E. Vaillancourt, R.D. Seidler, Genetic markers of dopaminergic transmission predict performance for older males but not females, *Neurobiol. Aging* 66 (2018) 180.e11–180.e21, <https://doi.org/10.1016/j.neurobiolaging.2018.02.005>.
- [43] M. Kamata, et al., Association study between the -1021C/T polymorphism of the dopamine-beta-hydroxylase gene promoter and personality traits in healthy subjects, *Neurosci. Lett.* 462 (2009) 54–57, <https://doi.org/10.1016/j.neulet.2009.06.077>.
- [44] E.L. Sabban, Catecholamines in stress: molecular mechanisms of gene expression, *Endocr. Regul.* 41 (2007) 61–73.
- [45] W. Schultz, Getting formal with dopamine and reward, *Neuron* 36 (2002) 241–263, [https://doi.org/10.1016/s0896-6273\(02\)00967-4](https://doi.org/10.1016/s0896-6273(02)00967-4).
- [46] W.R. Clark, M. Grunstein. *Are We Hardwired?: The Role of Genes in Human Behavior*, Oxford University Press, 2004.
- [47] I.E. Nagel, et al., Human aging magnifies genetic effects on executive functioning and working memory, *Front. Hum. Neurosci.* 2 (2008), <https://doi.org/10.3389/neuro.09.001.2008>.
- [48] F. Noohi, et al., Association of COMT val158met and DRD2 G>T genetic polymorphisms with individual differences in motor learning and performance in female young adults, *J. Neurophysiol.* 111 (2014) 628–640, <https://doi.org/10.1152/jn.00457.2013>.
- [49] F. Noohi, et al., Interactive effects of age and multi-gene profile on motor learning and sensorimotor adaptation, *Neuropsychologia* 84 (2016) 222–234, <https://doi.org/10.1016/j.neuropsychologia.2016.02.021>.
- [50] R. Holtzer, et al., Differential effects of COMT on gait and executive control in aging, *Neurobiol. Aging* 31 (2010) 523–531, <https://doi.org/10.1016/j.neurobiolaging.2008.05.011>.
- [51] K.E. Hupfeld, D.E. Vaillancourt, R.D. Seidler, Genetic markers of dopaminergic transmission predict performance for older males but not females, *Neurobiol. Aging* 66 (2018), 180. e11–180. e21.
- [52] M.G. Soeiro-De-Souza, et al., Gender effects of the COMT Val158Met genotype on verbal fluency in healthy adults, *Mol. Med. Rep.* 8 (2013) 837–844.
- [53] A. Wieland, et al., Gender differences in the endowment effect: Women pay less, but won't accept less, *Judgm. Decis. Mak.* 9 (2014) 558–571.
- [54] Gazzaniga, et al., *Cognitive Neuroscience - The Biology of The Mind*, W.W. Norton, New York, 2014, p. 45.
- [55] M. Amin, D. Ontaneda, Thalamic injury and cognition in multiple sclerosis, *Front. Neurol.* 11 (2021), <https://doi.org/10.3389/fneur.2020.623914>.
- [56] R. Fama, E.V. Sullivan, Thalamic structures and associated cognitive functions: Relations with age and aging, *Neurosci. Biobehav. Rev.* 54 (2015) 29–37, <https://doi.org/10.1016/j.neubiorev.2015.03.008>.
- [57] Y.D. Van Der Werf, et al., Thalamic volume predicts performance on tests of cognitive speed and decreases in healthy aging. A magnetic resonance imaging-based volumetric analysis, *Brain Res. Cogn. Brain Res.* 11 (2001) 377–385, [https://doi.org/10.1016/s0926-6410\(01\)00010-6](https://doi.org/10.1016/s0926-6410(01)00010-6).
- [58] N. Canessa, et al., The functional and structural neural basis of individual differences in loss aversion, *J. Neurosci.* 33 (2013) 14307, <https://doi.org/10.1523/jneurosci.0497-13.2013>.
- [59] M.A. Sánchez-González, et al., The primate thalamus is a key target for brain dopamine, *J. Neurosci.* 25 (2005) 6076–6083, <https://doi.org/10.1523/jneurosci.0968-05.2005>.
- [60] B. Moghaddam, Dopamine in the thalamus: a hotbed for psychosis? *Biol. Psychiatry* 68 (2010) 3–4, <https://doi.org/10.1016/j.biopsych.2010.05.014>.
- [61] A.F. Oke, et al., Three-dimensional mapping of norepinephrine and serotonin in human thalamus, *Brain Res.* 763 (1997) 69–78, [https://doi.org/10.1016/s0006-8993\(97\)00404-6](https://doi.org/10.1016/s0006-8993(97)00404-6).
- [62] A. Oke, et al., Lateralization of norepinephrine in human thalamus, *Science* 200 (1978) 1411–1413, <https://doi.org/10.1126/science.663623>.
- [63] I. Pérez-Santos, et al., Distribution of the noradrenaline innervation and adrenoceptors in the macaque monkey thalamus, *Cereb. Cortex* 31 (2021) 4115–4139, <https://doi.org/10.1093/cercor/bhab073>.
- [64] S.A. Flavin, D.G. Winder, Noradrenergic control of the bed nucleus of the stria terminalis in stress and reward, *Neuropharmacology* 70 (2013) 324–330, <https://doi.org/10.1016/j.neuropharm.2013.02.013>.
- [65] D. Weinschenker, J.P. Schroeder, There and back again: a tale of norepinephrine and drug addiction, *Neuropsychopharmacology* 32 (2007) 1433–1451, <https://doi.org/10.1038/sj.npp.1301263>.
- [66] P. Sokol-Hessner, R.B. Rutledge, The psychological and neural basis of loss aversion, *Curr. Dir. Psychol. Sci.* 28 (2019) 20–27, <https://doi.org/10.1177/0963721418806510>.
- [67] H. Takahashi, et al., Norepinephrine in the brain is associated with aversion to financial loss, *Mol. Psychiatry* 18 (2013) 3–4, <https://doi.org/10.1038/mp.2012.7>.
- [68] E.V. Sullivan, et al., Effects of age and sex on volumes of the thalamus, pons, and cortex, *Neurobiol. Aging* 25 (2004) 185–192, [https://doi.org/10.1016/s0197-4580\(03\)00044-7](https://doi.org/10.1016/s0197-4580(03)00044-7).
- [69] K. Menzler, et al., Men and women are different: diffusion tensor imaging reveals sexual dimorphism in the microstructure of the thalamus, corpus callosum and cingulum, *Neuroimage* 54 (2011) 2557–2562, <https://doi.org/10.1016/j.neuroimage.2010.11.029>.
- [70] D. Tomasi, N.D. Volkow, Gender differences in brain functional connectivity density, *Hum. Brain Mapp.* 33 (2012) 849–860, <https://doi.org/10.1002/hbm.21252>.
- [71] B.W. Balleine, J.P. O'Doherty, Human and rodent homologies in action control: corticostriatal determinants of goal-directed and habitual action, *Neuropsychopharmacology* 35 (2010) 48–69, <https://doi.org/10.1038/npp.2009.131>.
- [72] D. Knoch, et al., Disruption of right prefrontal cortex by low-frequency repetitive transcranial magnetic stimulation induces risk-taking behavior, *J. Neurosci.* 26 (2006) 6469–6472, <https://doi.org/10.1523/jneurosci.0804-06.2006>.
- [73] S. Fecteau, et al., Diminishing risk-taking Behavior by modulating activity in the prefrontal cortex: a direct current stimulation study, *J. Neurosci.* 27 (2007) 12500–12505, <https://doi.org/10.1523/jneurosci.3283-07.2007>.
- [74] R.A. Wise, Dopamine, learning and motivation, *Nat. Rev. Neurosci.* 5 (2004) 483–494, <https://doi.org/10.1038/nrn1406>.
- [75] M.L. Kringelbach, E.T. Rolls, The functional neuroanatomy of the human orbitofrontal cortex: evidence from neuroimaging and neuropsychology, *Prog. Neurobiol.* 72 (2004) 341–372, <https://doi.org/10.1016/j.pneurobio.2004.03.006>.
- [76] Camerer, C.F., *Prospect Theory in the Wild: Evidence from the Field*. *Advances in Behavioral Economics*, ed. C.F. Camerer, G. Loewenstein, and M. Rabin. 2004. 148–161.

- [77] M. Pessiglione, et al., Dopamine-dependent prediction errors underpin reward-seeking behaviour in humans, *Nature* 442 (2006) 1042–1045, <https://doi.org/10.1038/nature05051>.
- [78] G. Sescousse, et al., Processing of primary and secondary rewards: a quantitative meta-analysis and review of human functional neuroimaging studies, *Neurosci. Biobehav. Rev.* 37 (2013) 681–696, <https://doi.org/10.1016/j.neubiorev.2013.02.002>.
- [79] X. Liu, et al., Common and distinct networks underlying reward valence and processing stages: a meta-analysis of functional neuroimaging studies, *Neurosci. Biobehav. Rev.* 35 (2011) 1219–1236, <https://doi.org/10.1016/j.neubiorev.2010.12.012>.
- [80] S.N. Haber, B. Knutson, The reward circuit: linking primate anatomy and human imaging, *Neuropsychopharmacology* 35 (2010) 4–26, <https://doi.org/10.1038/npp.2009.129>.