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Subjective reward processing and catechol-*O*-methyltransferase Val158Met polymorphism as potential research domain criteria in addiction: A pilot study

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The Research Domain Criteria (RDoC) approach seeks to understand mental functioning in continuous valid dimensions ranging from functional to pathological. Reward processing is a transdiagnostic functioning domain of the RDoC. Due to prototypical abnormalities, addictions are especially applicable for the investigation of reward processing. Subjective reward processing is challenging to determine and differs between genotypes of the catechol-*O*-methyltransferase gene (COMT) Val158Met polymorphism for incomparable daily life experiences. Thus, we implemented the monetary incentive delay (MID) task with comparable reward cues and visual analog scales (VAS) to assess subjective reward processing in male abstinent cannabis-dependent individuals ($N = 13$) and a control group of nicotine smokers ($N = 13$). COMT Val158Met genotypes were nominally associated with differences in cigarettes smoked per day and motivation in the MID Task ($p = 0.028$; $p = 0.017$). For feedback gain, activation of the right insula was increased in controls, and activation correlated with gain expectancy and satisfaction about gain. Subjective value is not detached from reward parameters, but is modulated from expectancy and reward by the insula. The underlying neural mechanisms are a fundamental target point for treatments, interventions, and cognitive behavioral therapy.

KEYWORDS

subjective reward processing, COMT Val158Met polymorphism, research domain criteria, insula, gain expectancy

Introduction

The US National Institute of Mental Health has developed the Research Domain Criteria (RDoC) approach to explore the underlying biological causes of mental disorders (1). For this purpose, a research framework has been established to link and integrate current clinical syndromes with basic biological and behavioral components. The goal seeks to understand mental functioning in continuous valid dimensions ranging from functional to pathological. The RDoC framework consists of five predefined domains of functioning. The negative and the positive valence systems cover loss and reward constructs, such as loss, reward anticipation, reward prediction error, habit, and reward valuation including delay (2). Constructs are analyzed in a multi-dimensional approach comprising genes, circuits, observed behaviors, and self-reports (3).

Motivation is the energizing of behavior in pursuit of a goal, and obtaining goals or basic needs appropriately is rewarding (4). Thus, reward processes and motivation are closely linked. For example, reward contingency is used in current cognitive/behavioral treatments for schizophrenia and addiction to modify behavioral deficits or excess in motivation (5). Reward-related symptoms appear in the diagnostic criteria for multiple disorders and are thus transdiagnostic in nature (6). Reward processing abnormalities are a key component in the development and manifestation of a wide range of psychopathologies, including addictions (5). For instance, cannabis use is associated with amotivational syndrome (6). The effect of cannabis use on neural reward processing has been investigated intensively with the well-established reward paradigm of the monetary incentive delay (MID) task (7). The results display alterations in reward-related neural functioning (8, 9). For example, cannabis users showed hypoactivity in the left insula cortex in response to loss and loss avoidance outcomes (8). Cannabis and nicotine are often used at the same time and altered reward functioning could also be verified for nicotine use (10, 11). According to previous findings, nicotine use does not affect amotivational syndrome (6). The co-use of cannabis and nicotine and mutual reinforcement is a critical matter of prevention and public health (10).

Physical reward parameters or reward cue properties, such as magnitude, cannot precisely define subjective reward value (12). Value is an internal component of reward and is represented in subjective preferences, such as individual needs or emotional valence (5, 13). Representations are generated by brain mechanisms mediated by dopamine neurons in the substantia nigra and VTA, and phasic dopamine responses increase with the expected reward value (13). Nevertheless, the assessment of unobservable subjective value is a central challenge in reward research (5). One way to resolving this issue is to ask people how rewarding they find something and to compare choices between objectively equal rewards (5, 13).

Within the RDoC approach, genetic variants are among the units of analysis (3). The Val158Met polymorphism of

the gene for catechol-*O*-methyltransferase (COMT) leads to valine to methionine exchange at position 158 of the protein (14). Homozygotes for the 158Met allele exhibit 35–50% lower brain COMT activity than homozygotes for 158Val with higher extrasynaptic dopamine levels, while heterozygotes show an intermediate enzyme function (15). Wichers et al. investigated the effect of the Val158Met polymorphism on the ability to experience reward in daily life (16). Homozygotes for 158Met generated almost similar amounts of subjective wellbeing from a “bit pleasant” daily life experience as 158Val homozygotes did from a “very pleasant” experience. The ability to experience reward increased with the number of “Met” alleles. Despite the genetic differences in subjective reward processing, the daily life experiences in the study of Wichers et al. were neither comparable reward cues nor objectively equal rewards. Reward experience was operationalized as the effect of event appraisal on positive affect and associations between COMT genotype, event appraisal, and positive affect were examined with regression analysis. In addition, reward sensitivity was also measured with self-reports, as well as behavioral tasks (17). Increased reward sensitivity was predictive of substance use, substance use disorders, greater cravings, and positive affective responses in alcohol cue reactivity paradigms. In this context, genetic modulation of reward sensitivity *via* dopamine transmission may be of special interest to understand individual differences (17, 18).

The aim of the present pilot study is to investigate reward processing for the first time dimensionally in the context of the RDoC approach, rather than focusing on mental disorder categories only (3). Reward processing is a transdiagnostic functioning domain of the RDoC. Due to prototypical abnormalities, addictions are especially applicable for the investigation of reward processing and the focus is on the potential effects of nicotine and cannabis on amotivational syndrome and dimensions of subject reward processing, such as motivation. Subjective reward processing differs between Val158Met genotypes for incomparable daily life experiences and was assessed with delay after the experiences. In the present study, this issue was methodically resolved with visual analog scales (VAS) or self-reports, comparable reward cues, and a constant reinforcement rate. We hypothesized that homozygotes for 158Met would be more satisfied with the achieved gain in the MID Task than individuals with the other genotypes.

Materials and methods

Participants

Thirteen inpatients with cannabis dependency and 13 volunteers without cannabis abuse participated in the study. The sample size was set to a minimum of 24 participants to anticipate results for the main study (19, 20). All participants

were male subjects, right-handed smokers of European ancestry. The participants had been instructed to discontinue cigarette smoking for at least 2 h before the study and were interviewed and assessed by a clinical psychologist. The diagnosis of the cannabis-dependent inpatients was confirmed with the International Diagnostic Checklists for DSM-IV and axis I or II disorders other than nicotine dependence were excluded for all participants (21, 22). A urine drug test was conducted to control for the use of cannabis and other drugs (23). Any history of the abuse of other drugs, psychiatric, neurological or chronic diseases, head trauma, loss of consciousness, impaired vision, and the use of medication for volunteers were exclusion criteria. Nicotine dependence was assessed with the Fagerström Test (FTNA) and the participants were matched on nicotine dependence, age, verbal intelligence, and years of education (24). The groups did not differ in any dimension of the controlled variables ($P \geq 0.064$).

Cannabis consumption was assessed with self-report questionnaires and the European Addiction Severity Index questionnaire (EuropASI) (25). Only cannabis-dependent inpatients with at least 4 days of abstinence and without withdrawal symptoms were included. The mean age of initial use was 16 years ($SD = 2$), the mean cannabis use was 8 years ($SD = 6$), and 12 grams per week ($SD = 5$). The mean abstinence was 24 days ($SD = 24$).

Genotyping

DNA was extracted from the EDTA anticoagulated blood samples of all participants. A fragment containing the COMT Val158Met polymorphism (dbSNP: rs4680) was amplified with polymerase chain reaction (PCR). Sanger sequencing of this fragment was conducted commercially (26). Analyses of the sequences were performed by eye aided by the software Lasergene (27). Table 1 summarizes the genotypes of both study groups. Hardy Weinberg equilibrium was fulfilled for all study groups.

Reward reaction task and subjective reward processing

The reward reaction task was based on the monetary incentive delay (MID) task (7). Participants had to react to monetary reward cues with the push of a button (Figure 1 and Supplementary material).

The amount of gain and loss varied between 1, 2, and 3 Euros corresponding to the number of 1 Euro coins on the picture. The participants started with a credit of 5 Euros. They completed one practice run and two test runs. In the test runs, the target presentation was adapted to the individual reaction rates and an average hit rate of 50% was predefined. The participants were informed that they would be paid the higher outcome of one of

TABLE 1 COMT genotypes of individuals who smoke ($N = 13$) and are cannabis dependent ($N = 13$).

COMT genotype	Nicotine N (percent)	Cannabis N (percent)	Σ
Val/Val	7 (54)	4 (31)	11
Val/Met	4 (31)	5 (38)	9
Met/Met	2 (15)	4 (31)	6
Σ	13 (100)	13 (100)	26

the two test runs. Subjective reward processing was assessed with visual analog scales (VAS) correspondingly to the study of Wrase et al. (28). Before the reward reaction task, the participants were asked to rate their motivation and gain expectancy. After the task, they rated their effort for a gain of 3 Euros compared with 2 Euros and 1 Euro, their fear of a loss of 3 Euros compared with 2 Euros and 1 Euro, and their satisfaction with the achieved gain. Finally, the participants completed the items of the personality trait Reward Dependence of the Temperament and Character Inventory (TCI) (29).

fMRI data acquisition

fMRI data acquisition was performed on a 3 T magnetic resonance scanner (Magnetom VISION Siemens®) with a circularly polarized standard head coil (CP-Headcoil). For anatomical reference, a 3 D Magnetization Prepared Rapid Gradient Echo (MPRAGE) data set was acquired with the following parameters: TR = 9.7 ms, TE = 4 ms, flip angle 12° , matrix = 256×256 , and voxel size $1 \text{ mm} \times 1 \text{ mm} \times 1 \text{ mm}$. For functional scans, a gradient-echo echo-planar imaging (GE-EPI) sequence was conducted with the parameters TR = 1.9 s, TE = 30 ms, flip angle = 90° , matrix = 64×64 , and voxel size = $3.8 \text{ mm} \times 3.8 \text{ mm} \times 3.3 \text{ mm}$. fMRI volume acquisition was time-locked to the offset of the cues.

fMRI data analysis

fMRI data were analyzed for BOLD responses to reward anticipation and feedback. Data analysis was conducted with SPM8 (30). Voxel time series were interpolated to adjust non-simultaneous slice acquisition within each volume. Motion artifacts were corrected. Head movements were below 3 mm in translation and 3° in rotation from one volume acquisition to the next. The anatomical images were coregistered with the mean functional images. For normalization, the coregistered image was first spatially normalized to the standard template provided by the Montreal Neurological Institute (MNI-Template), and the obtained normalization parameters were then applied to all functional images. Voxel time series were smoothed with a Gaussian kernel ($FWHM = 8 \text{ mm}$).

In the first-level analysis, a statistical model with all conditions was computed for each participant according to



FIGURE 1

Cues for gain (green), neutral (black), loss (red) 250 ms, delay (cross) 2.25–2.75 s target (white square), and feedback with outcome 1650 ms.

TABLE 2 Activated brain regions through reward anticipation and feedback in individuals who smoke ($N = 13$) and are cannabis dependent ($N = 13$).

Contrast	Study group ($N = 13$)	Brain region	x	y	z	t	P
Anticipation gain vs. loss	Nicotine	R insula	39	-7	7	4.32	0.001
	Cannabis	R anterior cingulate cortex (ACC)	9	29	28	3.45	0.005
Anticipation loss vs. gain	Nicotine	L inferior frontal gyrus (IFG)	-54	17	31	4.50	0.001
	Cannabis	R primary somatosensory cortex	33	-40	58	3.76	0.001
Feedback gain	Nicotine	L postcentral gyrus	-60	-22	49	6.21	0.001
	Cannabis	R middle temporal gyrus (MTG)	57	-28	-2	7.15	0.001
		R superior temporal gyrus (STG)	60	-34	16	5.69	0.001
Feedback loss	Nicotine	R inferior frontal gyrus (IFG)	48	20	16	4.88	0.001
		R middle frontal gyrus (MFG)	24	56	-8	6.75	0.001
		R inferior frontal gyrus (IFG)	51	32	1	5.79	0.001
	Cannabis	R insula	27	23	-11	4.63	0.001
		R middle temporal gyrus (MTG)	60	-49	10	5.43	0.001
		R inferior frontal gyrus (IFG)	60	8	10	5.18	0.001
		R cingulum	12	-34	46	4.36	0.001

the general linear model approach (31, 32). Gain and loss were contrasted with the neutral conditions, e.g., “anticipation of gain” vs. “no anticipation.” Contrasts were calculated as t -statistic for each voxel.

In the second-level analysis, contrasts within the reward anticipation and feedback conditions were calculated for each group with a one-sample t -test at a significance level of $P < 0.005$ and a cluster threshold of $k > 5$. One-sample t -tests were FDR-corrected (33). Contrasts between cannabis-dependent inpatients and the control group were calculated for each condition with a two-sample t -test at a significance level of $P < 0.005$ and a cluster threshold of $k > 9$. The activated brain areas were determined on the basis of the coordinates of Hägele et al. (34). Regions of interest (ROIs; radius 5 mm) were sphere shaped and centered upon the peak voxel within each area of interest. The ROIs beta-values for each condition were extracted and converted into percent signal change using the Marseille Region of Interest Toolbox (35) software package (36).

Behavioral data analysis

With t -tests and multivariate analyses of variance, subjective reward processing, Reward dependence, and reaction times were analyzed for group differences between cannabis-dependent

inpatients and the control group. Subsequently, these variables were analyzed for Pearson's correlations with activated brain region and the number of met alleles. Bonferroni corrections were conducted to control the family-wise error rate. With Kruskal–Wallis H tests, group differences for nicotine use and subjective reward processing were analyzed between genotypes. Behavioral data analysis was calculated with the software package SPSS 22.0 (SPSS Inc., Chicago, IL, USA). Results with a p -value below 0.05 were considered statistically significant.

Results

Reward anticipation and feedback

Table 2 gives an overview of the brain regions revealing the main effects of reward anticipation and feedback. During feedback, no loss, activation of the right inferior frontal gyrus was increased in individuals who smoke ($x = 51$; $y = 32$; $z = 1$). Table 3 contains the brain regions with activation differences between the groups. Individuals who smoke showed an increased activation of the right insula for the anticipation of gain vs. loss ($x = 60$; $y = 5$; $z = 4$), and for feedback gain ($x = 21$; $y = 23$; $z = -8$; Figure 2).

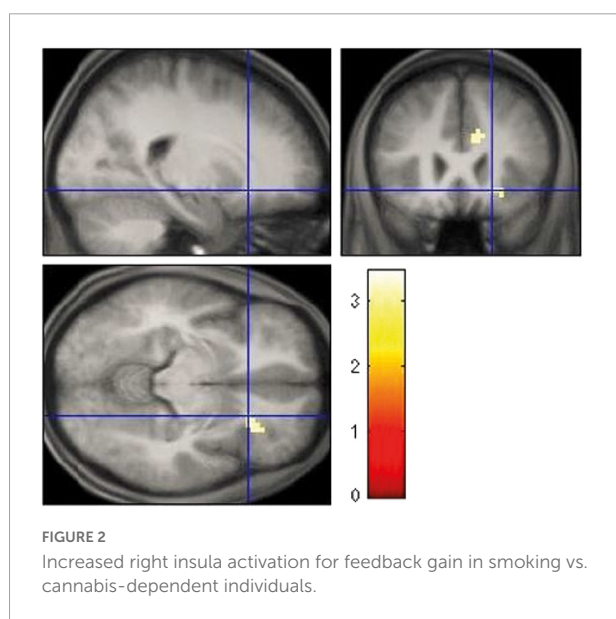
TABLE 3 Contrasts with differently activated brain regions ($P \leq 0.001$).

Contrast	Group comparison	Brain region	x	y	Z	t
Anticipation gain vs. loss	Nicotine > Cannabis	R insula	60	5	4	3.96
Anticipation loss vs. gain	Nicotine > Cannabis	L inferior frontal gyrus (IFG)	-48	50	1	3.96
Feedback gain	Nicotine > Cannabis	R insula	21	23	-8	3.46
Feedback loss	Nicotine > Cannabis	R inferior frontal gyrus (IFG)	63	11	16	4.37
		L inferior frontal gyrus (IFG)	-48	14	13	4.02
		L putamen	-30	5	4	3.91
		R insula	30	23	1	3.65

Subjective reward processing and catechol-O-methyltransferase genotype

Individuals who smoke and cannabis-dependent inpatients did not differ significantly in subjective reward processing or reward dependence (all $P > 0.28$; **Supplementary material**).

A Kruskal–Wallis H test showed nominal differences in cigarettes per day $\chi^2(2) = 7.118$, $p = 0.028$ and motivation $\chi^2(2) = 8.192$, $p = 0.017$ between COMT genotypes with a mean rank of 11.00 for Val/Val, 11.72 for Val/Met, and 20.75 for Met/Met and 11.86 for Val/Val, 19.06 for Val/Met, and 8.17 for Met/Met. There were statistically significant correlations for the anticipation of gain vs. loss in cannabis dependents, for feedback loss in smokers, and for feedback gain in individuals who smoke and cannabis-dependent inpatients (**Supplementary material**). For feedback gain, activation of the insula correlated negatively with gain expectance and positively with satisfaction with the achieved gain in individuals who smoke and cannabis-dependent inpatients ($p < 0.004$; $p < 0.014$).



Discussion

In the present pilot study, we investigated subject reward processing and the COMT Val158Met polymorphism in abstinent cannabis-dependent inpatients and individuals who smoke with the well-established reward paradigm of the MID Task and found the commonly activated brain regions for this task (Table 4; 37). The aim was to take reward processing into account dimensionally across different forms of addiction, rather than only focusing on overarching mental disorder categories. The study focused on the application of the RDoC as well as the feasibility to assess subject reward processing.

Subjective reward processing did not differ between nicotine and cannabis in any dimension. Nonetheless, the results suggest that the most likely dimensions for differences in a larger sample are effort gain and satisfaction about gain. Because of the small sample size and other methodical limitations, the claim was not to draw a statistical inference for genetic neuroimaging and the sample size was set to a minimum of 24 participants. The sample size should not have been set in terms of cannabis and nicotine use, but the COMT genotype. In the total sample, homozygotes for 158Met were not more satisfied with the achieved gain than carriers of the other genotypes, which is probably due to the sample size. However, genotypes nominally differ for cigarettes per day and motivation with the highest number of cigarettes and the lowest motivation rank for homozygotes of 158Met. Tentatively, one might assume that homozygotes for 158Met may have less negative subjective effects of nicotine and thus smoke more (38). Reduced

TABLE 4 The main findings for the MID Task, the COMT Val158Met polymorphism, and its implications.

- **For feedback gain:** Activation of the right insula was increased in controls and activation correlated with gain expectancy and satisfaction about gain.
- Subjective value is not detached from reward parameters, but is modulated from expectancy and reward by the insula
- **COMT Val158Met:** Genotypes were nominally associated with differences in cigarettes smoked per day and motivation in the MID Task.
- **Homozygotes for 158Met** may have less negative subjective effects of nicotine and thus smoke more.

motivation in homozygotes of 158Met has previously been found in men of a Swedish sample (39). For individuals who smoke, cigarettes per day and motivation correlated negatively. The profile of nicotine changes from stimulant to sedative with increasing dosage (40). The present correlation can thus be attributed to the “Nesbitt’s paradox” (38). Cannabis also affects motivation (41). Therefore, cigarettes and motivation were putatively not associated with cannabis dependence. In individuals who smoke, cigarette use correlated negatively and motivation positively with the activation of the right inferior frontal gyrus during feedback no loss. The right inferior frontal gyrus has been assigned to self-control, reward prediction errors, and sensation seeking and high sensation seekers are less sensitive to punishment (42–45). Higher cigarette use may hence imply lower self-control and response inhibition. Moreover, the present results suggest that motivation may be high precisely when predictions do not occur regardless of negative consequences.

For anticipation gain vs. loss, individuals who smoke had increased activation of the right insula compared with cannabis dependents. Acute THC increases perfusion in the insula with stronger increases for Val/Met heterozygotes (46). In anticipating rewards, insula activation is associated with motivational salience and sensitivity to reward magnitude (47, 48). Acute nicotine sensitizes and chronic smoking enhances the right insular response to gain and loss anticipation (11, 49). In contrast, activation of the left insular is reduced through acute cannabidiol and correlates with the salience of reward anticipation (48). The different contrast of anticipation gain vs. loss in the present study rather than gain vs. neutral cues in previous studies could be due to the diminished success probability of 50% (37). The current results suggest an overall desensitizing effect of the co-use of nicotine and cannabis on the insula and a decrease in salience for reward anticipation.

For feedback gain, individuals who smoke had increased activation of the right insula compared with cannabis-dependent inpatients. For reward feedback, the insula is involved in affective responding (37). The right anterior insula mediates interoceptive awareness (50). It integrates affective value with bodily states for reward-related adaptive behavior (51, 52). In the total sample, the activation of the right insula during feedback gain correlated negatively with gain expectancy and positively with satisfaction about the achieved gain. This finding indicates that the right insula interconnects gain expectancy with the evaluation of the reward-related performance and outcome independent of the substance. Additionally, the gain of the second test run correlated negatively with gain expectancy and positively with satisfaction. Higher gain expectancy at the beginning reduces reinforcement and thus motivation over the course of the MID Task, which impairs task monitoring and performance. Moderate or lower expectations have the opposite effect

and therefore increase satisfaction with the outcome. The insula and interoceptive awareness are also crucial for drug cravings (53–55). The insula elicits conscious interoceptive urges, such as drug craving in response to rewarding and drug stimuli, respectively. For cannabis-dependent inpatients, gain expectancy correlated negatively with satisfaction and abstinence. Satisfaction correlated positively with consumption. These associations suggest a sensitizing effect of cannabis on subjective reward processing with an increase in gain expectancy and satisfaction. The dose-dependent increase in satisfaction is putatively due to the acute psychoactive effect of cannabis (56). Acute subjective changes in relaxation and perception caused by cannabis are also related to its effect on the insula (46). Positive subjective drug expectancies impair decision-making of addicted patients (57). The impairment consists of an overreliance on habits at the expense of goal-directed behavior and may be particularly symptomatic for early abstinence. As subjects had no experience with the MID Task (7), rationally, they should have quoted a middle gain expectancy. The negative correlation between abstinence and gain expectancy confirms drug-associated habitual biases during early abstinence. Moreover, insular functioning is highly dependent upon dopamine transmission and aberrant activation indicates impaired cue processing (58). Higher-than-predicted rewards generate positive prediction errors and elicit brief dopamine activations, whereas lower-than-predicted rewards generate negative prediction errors and induce decreases in activity (12). Accurately predicted rewards do not change the activity. For cannabis-dependent inpatients, gain expectancy correlated negatively with the activation of the right anterior cingulate cortex during the anticipation of gain vs. loss. The anterior cingulate cortex is involved in error monitoring and is affected by nicotine and cannabis (59, 60). For error monitoring, the expectation about an action is compared with the outcome and a deviation elicits the correction of behavioral responses (61). Any expectancy such as the current gain expectancies distract *per se* from task performance. Higher gain expectancies may additionally shift and bias attention toward cues for monetary gain under the neglect of losses. In consideration of the diminished success probability of 50%, however, it is just as important to avoid losses, as it is to gain money. Increased activity of the anterior cingulate cortex may suggest that cannabis-dependent inpatients with lower gain expectancy differentiated more effectively between gain and loss cues and thus, equally focused on and endeavored to avoid losses.

At first glance, the assessment of subjective reward processing seems exceptionally challenging. Subjective reward values such as preferences, needs, or emotional valence are unobservable, highly individual, and thus indefinable by physical reward parameters (12). In the present study, this issue was methodically resolved with visual analog scales (VAS) or self-reports, comparable reward cues, and a constant

reinforcement rate. The variable gain instead of a predefined gain for all subjects was a limitation of the study. Nevertheless, the results suggest that subjective value is not completely detached from reward parameters, but a quotient between value and parameter: previous gain expectancies namely modulate the subsequent reward evaluation. Abnormalities in reward processing are prototypical for addictions and comprise a sensitizing effect of cannabis on subjective reward processing with an increase in gain expectancy and satisfaction according to the present results. Higher gain expectancies, whether cannabis-induced or not, reduce reinforcement, motivation, and satisfaction of the outcome and the insula might function as an inverse balance between expectation and satisfaction. The COMT Val158Met polymorphism might influence motivation. Any expectation distracts from task performance by affecting error monitoring and response correction of reward-related behavior negatively. Addiction research suggests that expectations or biases might be traced back to the overreliance on habits at the expense of goal-directed behavior. The same bias and overreliance on habits were reported for non-clinical obsessive-compulsive symptoms (62). Obtaining goals or basic needs in an appropriate manner is rewarding, and the pursuit of a goal defines motivation (4). Therefore, cognitive biases and the underlying neural mechanisms of error monitoring, response correction, and goal attainment might be the most fundamental target points of mental functioning and treatment.

In the RDoC framework, subjective reward processing can be assigned to the domain of the negative and positive valence systems. The COMT Val158Met polymorphism, the reward system including the insula, performance in the MID Task, and self-reports were utilized as units of analysis. Subjective value is not detached from reward parameters but is a modulated from expectancy and reward by the insula. The insula is crucial for the interconnection of expectancy and evaluation and aberrant activation might imply incongruence. Cognitive biases and the underlying neural mechanisms are thus the most fundamental target point for treatments, interventions, and cognitive behavioral therapy in further research. Examples of initial approaches are Cognitive Bias Modification and Emotional Bias Modification (63–65). These approaches are likely to be intensively extended and to be future of RDoC diagnostics and predominantly neural psychotherapy.

Data availability statement

The original contributions presented in this study are included in the article/**Supplementary material**, further inquiries can be directed to the corresponding author.

Ethics statement

This study was reviewed and approved by Ethics Committee at the University of Duisburg-Essen. The patients/participants provided their written informed consent to participate in this study.

Author contributions

NR and NS designed the study. NR was involved in the data acquisition and wrote the manuscript. AH was involved in genotyping. UB, IT, AH, and NS revised the manuscript. All authors read and approved the manuscript.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Supplementary material

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fpsy.2022.992657/full#supplementary-material>

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