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Application of the Research Domain Criteria (RDoC) Framework to Eating Disorders: Emerging Concepts and Research

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Abstract The Research Domain Criteria (RDoC) project was initiated by the National Institute of Mental Health as a heuristic for addressing the limitations of categorical, symptom-based psychiatric diagnoses. RDoC is conceptualized as a matrix, with the rows representing dimensional constructs or domains implicated in the expression of psychiatric symptoms and the columns representing units of analysis that can be used to assess dimensional constructs (i.e., genes, molecules, cells, circuits, physiology, behavior, and self-reports). Few studies in eating disorders have adopted an RDoC framework, but accumulating data provide support for the relevance of RDoC dimensions to eating disorder symptoms. Herein, we review findings from RDoC-informed studies across the five domains of functioning included in the RDoC matrix—negative valence systems, positive valence systems, cognitive systems, systems for social processes, and arousal and regulatory systems—and describe directions for future research utilizing RDoC to enhance study design and treatment development in eating disorders.

Keywords Eating disorders · Classification · Measurement · Dimensions · RDoC

This article is part of the Topical Collection on *Eating Disorders*

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Introduction

Eating disorders are serious psychiatric illnesses characterized by aberrant eating or behaviors intended to control weight, which lead to impairment in physical health or psychosocial functioning [1]. Eating disorders are common [2–4] and associated with serious medical complications [5], high rates of psychiatric comorbidity [6], and elevated all-cause and suicide mortality [7, 8]. Nevertheless, research on the etiology and treatment of eating disorders remains incipient. Etiologic models of disordered eating are complex and emphasize myriad biological, psychological, and social factors that may vary in their salience for individual patients [9]. Likewise, even the most effective treatments for eating disorders have response rates of about 50 % [10], and relapse is high following inpatient and outpatient interventions [11, 12].

One factor that complicates efforts to advance eating disorders research is the lack of a well-validated system for classifying eating-related psychopathology. The two leading models of psychiatric classification, the Diagnostic and Statistical Manual of Mental Disorders—fifth edition (DSM-5) [13]—and the International Classification of Diseases—10th edition (ICD-10) [14]—characterize eating disorders and other psychiatric conditions exclusively on the basis of observable signs and symptoms such as body weight, eating and weight control behaviors, and associated cognitive features. As reviewed elsewhere [15, 16•, 17, 18], these descriptive nosologies have notable shortcomings that pose problems for research including high rates of co-occurrence among disease categories, substantial within-diagnosis variability in symptom presentation, and a failure to reflect accurately the psychopathology of many patients, as evidenced by high rates of "not otherwise specified" diagnoses. Furthermore, because observable symptoms often are multi-determined, diagnoses based on presenting complaints are likely to be heterogeneous in terms

of underlying mechanisms, and efforts to link pathophysiological processes and associated treatments to particular psychiatric diagnoses have been disappointing [19].

In response to these limitations, the US National Institute of Mental Health (NIMH) established the Research Domain Criteria (RDoC) project as a framework for integrating research in psychopathology with findings from modern neuroscience [15, 16•, 18–21]. RDoC encourages scientists to move beyond categorical, symptom-based approaches to conceptualizing mental illness and focus on dimensions of behavior and neurobiology that may cut across current disease categories and explain within-group variability among individuals with the same descriptive diagnosis. The ultimate goal of RDoC is to facilitate "precision medicine for psychiatry" (p. 396) through the development of a new, pathophysiology-based approach to psychiatric classification [19].

Below, we provide an overview of the RDoC framework followed by a review of research supporting the relevance of RDoC concepts to eating disorders. We conclude with future directions in the application of the RDoC framework to eating disorders and clinical implications of RDoC-informed research.

The RDoC Framework

In 2008, the NIMH published a strategic plan that included as an objective "Develop, for research purposes, new ways of classifying mental disorders based on observable behavior and neurobiological measures" (http://www.nimh.nih.gov/about/strategic-planning-reports/index.shtml) [22]. RDoC is the tool for implementing this objective. The immediate goals of RDoC include identifying broad domains of functioning and their constituent dimensional constructs, developing reliable and valid measures of each construct across a range of units of analysis, and funding research to characterize domain functions across the full spectrum of clinical and nonclinical presentations [23]. At this stage, RDoC is not intended to serve as a clinical tool or as a replacement for the DSM and ICD nosologies [16•]. However, in the long-term, it is hoped that RDoC-informed research will facilitate the establishment of a psychiatric nosology grounded in the neuroscience of human behavior [19].

As shown in Table 1, the RDoC framework is conceptualized as a two-dimensional matrix with the rows representing five higher-order domains of function and their component constructs, and the columns representing various units of analysis that can be used to assess these constructs [20, 23]. The cells at the intersections of the rows and columns are populated by research findings [18]. Several recent articles have described the background and structure of the RDoC framework [15, 16•, 18, 20, 23], but a few key points are worth noting herein.

First, the RDoC domains are not intended to capture the full range of psychopathology included in the DSM and ICD nosologies. Rather, the NIMH elected to focus initially on dimensions for which there is solid evidence to support ongoing research, with the expectation that the RDoC matrix will be updated regularly to reflect current science [16•].

Second, the constructs listed in Table 1, and their associated subconstructs detailed on the NIMH website (http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml#toc_matrix), were generated during a series of workshops attended by scientists with expertise relevant to the domain under consideration (e.g., negative valence systems) whose methodologies spanned the proposed units of analysis. In order to be included in the RDoC matrix, a proposed construct had to meet two criteria: (1) There had to be evidence for the validity of the construct as a behavioral function, and (2) there had to be evidence for a neural circuit or system that is implicated in generating the behavior associated with the construct [16•, 17].

Third, RDoC places equal weight on the seven units of analysis proposed to evaluate the constructs included in the matrix, i.e., genes, molecules, cells, circuits, physiology, behavior, and self-reports. Genes, molecules, and cells are self-explanatory [23]. Circuits refer to measurements of particular brain circuits using neuroimaging techniques or assessments validated by animal models (e.g., emotion-modulated startle paradigm) [20]. Physiology refers to well-established measures that have been validated in assessing certain constructs but do not measure circuit activity directly (e.g., heart rate). Behavior comprises both behavioral tasks (e.g., working memory tasks) and observations of behavior, and self-reports include interviews, questionnaires, and other self-report instruments. There also is a separate "paradigms" column to denote tasks that are especially useful for the study of a particular construct [23].

Finally, although not depicted graphically in the RDoC matrix, developmental processes and environmental factors are included in the RDoC framework as dimensions orthogonal to the domains and constructs [18] and studies that adopt the RDoC approach are expected to incorporate these crucial determinants of human behavior [20].

It is important to note that RDoC is a relatively new and untested model of studying psychopathology. Critics have argued that it places too much emphasis on neural circuits and measures without acknowledging the limitations of these units of analysis, pays inadequate attention to social and environmental context, and fails to incorporate insights gained from clinical observation and treatment [24–26]. Thus, although RDoC is a promising framework for investigating eating disorders and other psychopathology, its utility remains to be verified.

The RDoC Framework and Eating Disorders

RDoC-informed studies differ from traditional research in psychiatry in several important ways that have relevance to eating disorders. First, rather than recruiting participants on the basis of DSM-5 or ICD-10 criteria, investigators utilizing an RDoC approach are encouraged to adopt a broader sampling frame that might include individuals who do not meet full criteria for a categorical diagnosis, patients with more than one categorical diagnosis, or individuals representing varying degrees of severity on a particular symptom dimension like loss of control eating or extreme dietary restraint [15, 18]. Given that "other specified feeding or eating disorder" continues to be the modal diagnosis in eating disorders [27, 28], adopting a broader sampling frame could increase the validity of eating disorders research. Furthermore, high rates of comorbid psychopathology [3] and a lack of diagnostic stability in eating disorders [29] support the utility of employing inclusion and exclusion criteria that go beyond traditional diagnostic categories.

Second, in traditional research studies, presenting symptoms or constellations of symptoms that satisfy the criteria for a particular categorical diagnosis (e.g., anorexia nervosa) typically are the unit of analysis on which participants are grouped and compared on a set of outcomes. However, in an RDoC study, an element from any unit of analysis could be used as the grouping (i.e., independent) variable, depending on the hypothesis under consideration [16•, 23]. For example, an investigator interested in the role of reward learning in eating disorder severity/chronicity might group patients

Table 1 Schematic overview of the RDoC matrix

Domains	Units of analysis							
Constructs	Genes	Molecules	Cells	Circuits	Physiology	Behavior	Self-reports	Paradigms
Negative valence systems								
Acute threat ("fear")								
Potential threat ("anxiety")								
Sustained threat								
Loss								
Frustrative nonreward								
Positive valence systems								
Approach motivation								
Initial responsiveness to reward								
Sustained responsiveness to reward								
Reward learning								
Habit								
Cognitive systems								
Attention								
Perception								
Declarative memory								
Language behavior								
Cognitive (effortful) control								
Working memory								
Systems for social processes								
Affiliation and attachment								
Social communication								
Perception and understanding of self								
Perception and understanding of others								
Arousal and regulatory systems								
Arousal								
Circadian rhythms								
Sleep and wakefulness								

Adapted from http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml#toc_matrix). Developmental processes and environmental factors are included as dimensions orthogonal to the constructs in the RDoC matrix [20]. "Paradigms" are tasks that are especially useful for the study of a particular construct; they are not "units of analysis" per se [23]

presenting to an eating disorders clinic according to response in fronto-striatal circuitry during a probabilistic reversal learning task (measured using functional imaging) and then examine whether different patterns of circuit response predict self-report measures of symptom severity and illness duration, performance on behavioral tasks (e.g., a laboratory meal), or response to a particular treatment (e.g., olanzapine).

Finally, RDoC emphasizes the importance of studying the full range of variation in dimensional constructs, recognizing that in many cases, extremes at both ends of a spectrum can contribute to psychopathology [16•]. This approach is particularly relevant to the study of eating disorders where observable symptoms often span the full range of behavior (e.g., from extreme overcontrol of eating to loss of control and binge eating), and initial evidence suggests that both underactivity and overactivity in circuits mediating the same RDoC dimension (e.g., cognitive control) are salient to the expression of different forms of eating-related psychopathology [30•].

RDoC Domains and Eating Disorders

Eating disorders have been linked with constructs represented in all five of the RDoC domains (for reviews, see [31–34]). Although most studies have utilized traditional research designs in which eating disorder symptoms or diagnoses serve as the independent variable, interest in alternative and dimensional approaches to the classification of eating disorders is growing [35••, 36, 37]. Below, we review recent theory and research documenting the relevance of RDoC constructs to eating disorders, focusing on studies that have utilized RDoC-informed research designs.

Negative Valence Systems

The relevance of negative valence systems to eating disorders is supported by multiple lines of research. Hyper-responsivity in fear circuits (i.e., amygdala, anterior cingulate cortex, hippocampus, insular cortex, striatum, and prefrontal cortex regions) to symptom-provoking stimuli (e.g., food and body image cues) [32, 33] and increased serotonin function has been documented in underweight eating disorder phenotypes [32], consistent with the salience of the acute threat dimension. Moreover, high levels of punishment sensitivity [38–40] and attentional bias to threatening stimuli (e.g., eating disorder-related words, angry faces) [41, 42] distinguish individuals with threshold and subthreshold eating disturbances from healthy controls, supporting the role of sustained threat processes. Finally, observations of reduced serotonin function in phenotypes characterized by loss of control eating and purging [32] are consistent with the notion that alterations in frustrative nonreward are salient to eating disorders.

Studies employing RDoC-compatible designs to examine negative valence systems in eating disorders are sparse. One report in 53 female college students found that startle-blink magnitude in response to body image cues (an index of acute threat) was a better predictor of the severity of eating disorder psychopathology (measured via self-report) than self-reported response to body image cues [43•]. This work is consistent with RDoC's emphases on studying the full range of functioning and validating measures for future research [16•].

Another study in college women examined relations between startle response to disorder-specific and nonspecific aversive stimuli and the severity of self-reported bulimia nervosa (BN) and obsessive compulsive disorder (OCD) symptoms [44•]. Results showed that increased startle potentiation to food pictures was associated with the severity of BN symptoms, but not OCD symptoms, whereas increased startle potentiation to contamination and general threat pictures was associated with the severity of OCD symptoms, but not BN symptoms. Startle magnitude in response to contamination and general threat pictures was especially high among women reporting both OCD and BN symptoms. These findings illustrate the utility of RDoC constructs for identifying mechanisms that may be shared across current diagnostic categories, as well as processes that differentiate phenotypic presentations.

Tanofsky-Kraff et al. [45••] have proposed an RDoC-compatible approach for studying the relation between loss of control eating and stress, focusing on processes related to acute threat. Specifically, these authors describe a research design in which assessments of genes (e.g., serotonin transporter gene [SCL6A4]), molecules (cortisol levels), and circuits (amygdala-orbitofrontal cortex activity during a social stress task) might be used to predict eating behavior in the laboratory and self-reported levels of negative affect. Such a design could be used to test hypotheses about the role of stress in the development of aberrant eating and the identification of risk factors for binge eating.

Positive Valence Systems

All of the constructs included under positive valence systems (i.e., approach motivation, initial responsiveness to reward, sustained responsiveness to reward, reward learning, and habit) have been implicated in eating disorders. Few studies have utilized RDoC designs explicitly, but results from several lines of research support the notion that alterations at both ends of the positive valence spectrum are salient to the classification eating disorders.

For example, broad alterations in the subconstructs of approach motivation have been documented in individuals with eating disorders. First, studies have shown that *reward valuation* is abnormally elevated in individuals with binge eating syndromes but excessively diminished in patients with

AN [46–48, 49•]. Second, studies have documented increased willingness to work for disorder-specific rewards (e.g., exercise, binge foods) in patients across the anorexia nervosa (AN)-BN spectrum [50, 51], suggesting that alterations in *effort valuation* are predictive of a broad range of eating disorder phenotypes. Third, data indicate that ill and recovered individuals with AN exhibit increased activity in the orbitofrontal cortex, insula, and ventral striatum in response to cues predicting food receipt, whereas individuals with full-threshold and subthreshold BN show hypoactivity in these regions to food cues [31], suggesting that abnormalities in *expectancy/reward prediction error* are salient to eating disorders. Finally, Chan et al. [52] examined cognitive processes underlying *preference-based decision-making* in individuals with AN and BN; results indicated that impairments in memory function were responsible for poor performance in patients with AN, whereas greater relative sensitivity to gains versus losses characterized poor performance in patients with BN.

In addition to heterogeneity across eating disorder phenotypes in approach motivation, preliminary data suggest that variability may exist within current eating disorder diagnoses, which may have implications for treatment. Balodis et al. [53••] administered a monetary incentive delay task during functional magnetic resonance imaging (fMRI) to assess activity in neural circuits mediating reward expectancy and outcome in 19 obese individuals seeking treatment for binge eating disorder (BED). Results showed that diminished activity in the ventral striatum and inferior frontal gyrus during the anticipatory phase of reward processing, and diminished activity in the medial prefrontal cortex during the outcome phase of reward processing, predicted continued binge eating after 4 months of treatment. These findings could suggest that individual differences in expectancy/reward prediction error represent a potential target for the development of subgroup-specific interventions, which is consistent with the RDoC goal of facilitating precision medicine in psychiatry [19].

Studies using taste reactivity paradigms have examined the role of reward attainment constructs (i.e., initial and sustained responsiveness to reward) in eating disorders. For instance, Oberndofer et al. [54•] found that response in the right anterior insula to the taste of a sucrose solution was diminished in women recovered from AN and exaggerated in women recovered from BN, relative to healthy controls. Similarly, Filbey et al. [55•] showed that hyper-responsivity in neural reward regions (e.g., amygdala, putamen, insula, posterior cingulate gyrus) to "personally relevant" high-calorie taste cues (e.g., delivery of a preferred sugared soda) was positively correlated with the severity of binge eating symptoms in a sample of 26 overweight and obese individuals. Conversely, research focusing on the neural correlates of palatable food consumption in individuals with full- and sub-threshold BN suggests that hypo-functioning reward circuitry may characterize longer-term responsiveness to reward attainment [56].

Studies of reward learning in eating disorders mirror findings for other positive valence constructs in that phenotypes characterized by binge eating and purging appear to differ from phenotypes characterized by low body weight. For example, Frank and colleagues conducted a series of studies in which they examined the neural responses of individuals with AN and BN to the unexpected violation of learned associations between conditioned visual stimuli and unconditioned taste stimuli (sucrose or tasteless solution) [57, 58]. AN was associated with increased activity in the orbitofrontal cortex to the unexpected receipt and omission of taste reward, whereas BN was associated with diminished activity, relative to controls. Furthermore, reward circuit hypoactivity predicted the severity of binge eating and purging in individuals with BN [58].

Finally, there is increasing interest in the potential role of mechanisms underlying habit formation in the persistence of eating disorders, especially AN [59••]. Notably, one study in a sample of 12 women with AN showed that severity of "compulsive acts" was negatively associated with fronto-striatal activity to food cues [60]; this is consistent with cognitive neuroscience models of habit formation, which emphasize a transition from impulsive to compulsive processes in the establishment of habitual behaviors [61]. An important direction for future RDoC-informed research will be to examine whether assessment of the habit dimension along several units of analysis (e.g., molecules, circuits, behavior) is predictive of clinically meaningful outcomes (e.g., duration of illness, response to particular treatments) in patients with eating disorders.

Cognitive Systems

The cognitive system most frequently implicated in eating disorders is cognitive control, particularly the subconstruct *response selection*, *inhibition*, or *suppression*. In general, data suggest that individuals with AN, BN, and BED exhibit hypoactivity in fronto-striatal circuits during performance on response inhibition tasks relative to healthy controls [62–65]. However, Lock et al. [66] reported that adolescents with AN-binge/purge type and BN (n=13) exhibited increased activity in the right dorsolateral prefrontal cortex compared to adolescents with restricting AN and healthy controls during performance on a response inhibition task. Discrepant findings may be due to differences among studies with respect to behavioral tasks or sample characteristics (e.g., age, eating disorder phenotypes).

Findings are mixed regarding the potential role of circuit activity during response inhibition tasks for classifying eating disorder patients. In a sample of 20 women with BN, Marsh et al. [64] found an inverse relation between number of binge eating episodes and activity in the right medial prefrontal, temporal, and inferior parietal cortices, as well as the caudate

nucleus, during performance on a response inhibition task, suggesting that individual differences in fronto-striatal activity might serve as a marker of illness severity. Similarly, Balodis et al. [62] reported that dietary restraint scores were negatively associated with activity in the ventromedial prefrontal cortex and interior frontal gyrus during a Stroop task in 11 patients with BED. In contrast, Lock et al. [66] found no relation between activity in cognitive control circuits and indicators of illness severity in adolescents with eating disorders beyond the effects of categorical diagnosis.

Finally, "set shifting"—a concept that maps on most closely to the cognitive control subconstruct of *goal selection*, *updating*, *representation*, and *maintenance*—has received considerable attention in the eating disorders field as a putative mechanism that might increase risk for disease onset and influence illness course and treatment response [67•]. Set shifting has been conceptualized as an index of cognitive inflexibility and perseveration in individuals with eating disorders, and recent data suggest that neural activation during set-shifting tasks in the scanner predicts subsequent neuropsychological test scores outside of the scanner [68••]. Specifically, in a sample of 21 patients with AN, Garrett et al. [68••] showed that lower activation in the ventrolateral prefrontal cortex and insula and higher activation in anterior middle frontal regions during a set shifting task predicted greater change on neuropsychological measures of set shifting after 16 weeks of treatment. Circuit activity during set shifting did not predict change in body mass index or eating disorder psychopathology, however, which raises some question about the clinical utility of classifying individuals with AN according to neural correlates of set shifting. Moreover, with few exceptions [69, 70], set shifting research in eating disorders has been limited by a reliance on multidimensional neuropsychological tasks that tend to conflate cognitive and reward processes [67•]. Thus, future research using precise neurocognitive tasks is needed to evaluate the relative salience of cognitive control and positive valence systems for the classification of eating disorders.

Systems for Social Processes

Research linking eating disorders to the constructs included in the social processes domain was summarized recently in a systematic review and meta-analysis [71••]. Briefly, Caglar-Nazali et al. [71••] found evidence that, relative to healthy controls, individuals with eating disorders exhibit impairments in all of the constructs listed under systems for social processes, that is, affiliation and attachment, social communication, perception and understanding of self, and perception and understanding of others. Effect sizes were largest for measures of insecure attachment, lower facial communication, negative self-evaluation, difficulties understanding the mental states of others, and social inferiority. Nevertheless, most work focusing on systems for social processes in eating disorders has utilized behavioral tasks and self-report measures, and little is known about the relevance of social constructs across other units of analysis (e.g., cells, circuits). In addition, studies primarily have focused on comparisons between individuals with eating disorders, especially AN, and healthy controls rather than adopting a dimensional approach to participant classification.

Arousal and Regulatory Systems

There has been a longstanding interest in the role of circadian rhythms in the classification of eating disorders, and night eating syndrome (NES)—a disorder characterized by a pattern of circadian delayed eating behavior—is included as an "other specified feeding or eating disorder" in DSM-5 [13]. A few studies have examined eating disorder symptoms in samples classified on the basis of self-reported circadian typology (i.e., morning versus evening chronotype), with mixed results. Harb et al. [72] found significant relations between evening chronotype and severity of self-reported binge eating and night eating behaviors in 100 individuals admitted to an outpatient nutrition clinic. However, the association between night eating and evening chronotype no longer was significant after controlling for binge eating symptoms. In contrast, Lemoine et al. [73] found no relation between eating disorder diagnosis and evening chronotype in a sample of 1468 individuals receiving inpatient treatment for a psychiatric disorder.

A burgeoning literature has sought to characterize the neural correlates of night eating behavior, which could have implications for the classification of eating disorders. For example, Birketvedt et al. [74] reviewed studies focusing on neuroendocrine disturbances in NES; in general, they found evidence for alterations in the circadian profiles of cortisol and melatonin in individuals with NES relative to healthy controls, although results have been inconsistent. Similarly, Pollack and Lundgren [75] reviewed studies examining associations between sleep deprivation and behavioral and neural facets of eating behavior, which could suggest a role for sleep-wakefulness in the classification of eating disorders. Although most studies have focused on healthy adults, results indicate that sleep deprivation alters brain reward circuit activation to food cues and increases the desirability of palatable foods [75]. Moreover, one study found that self-reported daytime sleepiness was associated with decreased activation in the ventrolateral prefrontal cortex to high- versus low-calorie food images; this pattern of neural activation also was linked to self-reported overeating in women, but not men [76].

The arousal and regulatory systems domain focuses on constructs related to sleep, but investigators in the eating disorders field likely will be interested in regulatory systems related to energy intake and expenditure, as well. For example, although feeding behavior is influenced by other RDoC

domains, especially positive valence systems and cognitive systems, these systems interact with homeostatic mechanisms in the hypothalamus and brainstem to regulate energy intake, as well as peripheral signals from the gut, pancreas, liver, adipose tissue, and muscle that are not currently considered in the RDoC framework [34, 77]. Aberrant eating behaviors are associated with numerous forms of psychopathology, besides eating disorders (e.g., mood disorders, schizophrenia, impulse control disorders, and substance use disorders). Furthermore, it now is apparent that homeostatic eating (i.e., eating designed to preserve energy balance) and hedonic eating (i.e., food intake in the absence of any energy depletion signals) do not occur in isolation, and endocrine hormones implicated in the regulation of homeostatic eating (e.g., insulin, leptin, ghrelin) also modulate the effects of drugs of abuse [78]. Thus, there may be a place for mechanisms involved in the regulation of homeostatic eating in future RDoC schemes.

Conclusions

The RDoC framework offers a novel approach to the study of eating disorders. Rather than conceptualizing participants in terms of categorical diagnoses, RDoC provides an opportunity to investigate how individual differences in neural mechanisms associated with aberrant eating may be linked to etiologically and clinically relevant outcomes. Although emerging data suggest that many of the constructs included in the RDoC matrix are relevant to eating disorders, few studies have utilized RDoC-compatible designs, and most work to date has focused on comparisons between individuals meeting DSM or ICD criteria for an eating disorder and controls. Studies utilizing broader inclusion criteria (e.g., anyone presenting to an eating disorders clinic), novel approaches to classification (e.g., grouping on the basis of response to a behavioral task), and outcomes representing multiple units of analysis (e.g., circuits, physiology, self-reports) are needed to advance the field. Furthermore, given the complexity of eating behavior, future studies would benefit from considering potential interactions among various RDoC domains and constructs (e.g., positive valence and cognitive control systems [30•, 67•]).

Ultimately, RDoC seeks to improve treatment for individuals with psychiatric disorders by matching patients to interventions designed to target the pathophysiological mechanisms that underlie symptom expression [19]. Several interventions have been developed to address neural mechanisms thought to drive disordered eating symptoms [78–82]. However, to the extent that neural mechanisms underlying the expression of particular symptoms (e.g., extreme dietary restraint) differ among patients, traditional clinical trials in which participants are recruited on the basis of phenotypic presentation are unlikely to result in significant advances in treatment delivery. The RDoC framework provides an exciting alternative for treatment development research in eating disorders and offers hope that one day, patients will be matched to treatments that are most likely to result in the amelioration of symptoms.

Compliance with Ethics Guidelines

Conflict of Interest Jennifer E. Wildes and Marsha D. Marcus declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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