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Effort-Based Decision Making in Psychopathology: A Transdiagnostic Multilevel Meta-Analysis and Systematic Review of Behavioral Patterns and Mechanisms Underlying Amotivational Psychopathology

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Effort-based decision making (EBDM) is fundamental to motivation. This cognitive process weighs the effort required to obtain a reward against its magnitude and the probability of obtaining it. Research suggests both transdiagnostic and disorder-specific patterns and mechanisms of EBDM in individuals with schizophrenia spectrum disorders, major depressive disorder, bipolar disorder, and those at risk for these conditions. This preregistered meta-analysis (CRD42022344605) aimed to quantify EBDM across these conditions compared to healthy controls and examine mechanisms underlying altered effort allocation. PubMed, APA PsycInfo, Embase, and Web of Science databases were systematically searched for eligible studies. We synthesized 115 effect sizes from 68 studies (N = 3,697). Three-level random-effects meta-analyses revealed moderate deficits in EBDM across schizophrenia spectrum disorders ($g = -0.40$) and bipolar disorder ($g = -0.39$), a smaller impairment in major depressive disorder ($g = -0.28$), and no significant reductions in at-risk samples ($g = -0.01$). Altered effort allocation patterns were marked by reduced effort exertion across increasing reward and probability conditions, with schizophrenia spectrum disorders showing the most pronounced deficits. The most robust moderators were the number of probability conditions, ethnicity, gender, and the severity of positive and negative symptoms. A narrative synthesis of computational modeling studies indicated that maladaptive EBDM may reflect increased effort sensitivity, reduced reward sensitivity, and cognitive impairment, with variations across disorders. Whereas schizophrenia spectrum disorders are associated with unsystematic effort allocation and cognitive deficits, major depressive disorder and bipolar disorder show heightened effort sensitivity. Overall, findings point to both shared and disorder-specific mechanisms of EBDM, offering directions for targeted interventions.

Public Significance Statement

Many people living with serious mental illnesses, such as schizophrenia, major depression, and bipolar disorder, struggle with motivation, which can make it harder to work, study, and maintain relationships. In this meta-analysis, we combined results from previous studies and found that these conditions are often linked to reduced effort to pursue rewards, especially among men and people from ethnic minority backgrounds. Reduced effort is driven by diagnosis-specific problems in thinking or in how effort demands are experienced. These disorder-specific mechanisms have important implications for the development of tailored treatments that improve motivation, support meaningful participation in daily life, and reduce long-term disability and costs.

Keywords: effort-based decision making, transdiagnostic, schizophrenia, depression, bipolar

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continued

A sense of motivation and sufficient drive or interest in pursuing personal goals are essential for having rewarding and pleasurable experiences in everyday life. These abilities enable individuals to lead fulfilling and self-determined lives and are therefore pivotal for mental health. Problems with motivation are common in psychopathology, where they manifest as amotivational symptoms and significantly impair daily functioning by impeding goal-directed behavior (e.g., Granholm et al., 2020). One key mechanism proposed to underlie amotivational symptoms is impaired effort-based decision making (EBDM; Husain & Roiser, 2018). EBDM refers to the process of deciding whether to exert effort to obtain a desired outcome. Impairments in EBDM are increasingly recognized as a hallmark of serious mental illnesses, particularly schizophrenia spectrum disorders (SSD; Blouzard et al., 2023), major depressive disorder (MDD; Horne et al., 2021), and bipolar disorder (BD; Barch et al., 2023; Culbreth, Moran, et al., 2024). Notably, motivational deficits are not confined to clinical diagnoses. They span a continuum from the healthy general population to individuals at risk for mental illness and those diagnosed with mental disorders, underscoring their transdiagnostic significance (Strauss & Cohen, 2017). The present study synthesizes more than 15 years of research on behavioral patterns in EBDM, offering a novel cross-diagnostic and meta-analytic perspective that highlights both commonalities and differences in EBDM. By integrating findings across diagnostic groups, we aim to provide a comprehensive perspective on motivational impairments and their potential underlying transdiagnostic and disorder-specific mechanisms.

Amotivational Psychopathology and Mental Health

Amotivational symptoms are often described using overlapping terms, including anhedonia (Craske et al., 2024), avolition (Foussias & Remington, 2008), amotivation (Kaliuzhna et al., 2021), or apathy (Thant & Yager, 2019). Anhedonia is defined as markedly diminished interest or pleasure in most activities (Husain & Roiser, 2018) and comprises both an affective (i.e., a diminished capacity to experience pleasure from a reward) and a motivational component (i.e., the reduced interest in seeking pleasurable/rewarding experiences). Similarly, avolition, amotivation, and apathy refer to a sense of diminished motivation for physical, cognitive, or emotional activity or simply a lack of motivation that can be observed in reduced goal-directed behavior and is not attributable to intellectual impairment, emotional distress, or diminished level of consciousness (Marin, 1991).

Many mental disorders are associated with amotivational symptoms. According to the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (American Psychiatric Association, 2013), anhedonia is one of the cardinal symptoms of MDD. Along with anhedonia, MDD symptoms may include negative or depressed mood and various cognitive and physical symptoms that impair daily functioning. MDD is common with a lifetime prevalence of 11% to 15% (Bromet et al., 2011) and is globally ranked as the single largest contributor to nonfatal health loss (World Heatlth Organization, 2017). Among individuals diagnosed with MDD, up to 90% may experience anhedonia (Haarasilta et al., 2001). The experience of anhedonia in people with MDD has been found to predict unfavorable trajectories (Morris et al., 2009), suicidal ideation (Gillissie et al., 2023), and is associated with poor treatment response (Alsayednasser et al., 2022), highlighting its central role in the disorder.

Aberrant motivational states are also strikingly present in individuals diagnosed with BD. Bipolar disorders are characterized by extreme mood swings and have a prevalence of 1%–2% in the general population (Vieta et al., 2018). Manic and hypomanic episodes in BD are linked to a significant increase in motivation. This increased motivation manifests as excessive energy, increased drive, euphoria, or impulsive behavior. By contrast, depressive episodes within BD are frequently accompanied by the previously described amotivational symptoms such as anhedonia with a prevalence of up to 90% (Dimick et al., 2021). In BD, depressive episodes constitute a significant risk factor for suicide-related mortality (Carvalho et al., 2020). Moreover, particularly anhedonia is associated with greater severity of mania and increased prescription of antipsychotic medications in individuals with BD (Dimick et al., 2021). Residual anhedonia may also be present in individuals experiencing euthymic phases of BD or in those with remitted MDD (Di Nicola et al., 2013). The presence of anhedonia in these states suggests that amotivational psychopathology can represent an enduring trait in affective disorders and that neurobiological dysfunctions in motivational processes may persist regardless of mood state.

SSD represent the third mental health condition in which amotivational symptoms are recognized as a prominent diagnostic feature in up to 80% of individuals diagnosed with SSD (Husain & Roiser, 2018). The prevalence of SSD is approximately 1% of the world population. SSD are associated with a substantial burden of disease and premature mortality (Charlson et al., 2018; Correll et al., 2022). The symptoms of SSD encompass a range of conditions marked by profound disruptions in thinking (e.g., delusions), perception (i.e., hallucinations), and behavior that clearly exceed the limits of normative experience (McCutcheon et al., 2020). These are typically categorized as either positive or negative. Positive symptoms are experiences that add to a person's typical perceptions and behavior including psychotic experiences such as hallucinations and delusions. Negative symptoms involve reductions in normal functioning and form an umbrella category of amotivational phenomena. Within

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this category, anhedonia and amotivation/avolition/apathy are often considered the core negative symptoms (Chan et al., 2024; Foussias & Remington, 2008). Negative symptoms also include expressive deficits, such as blunted affective expression and alogia, which manifest as diminished facial expressivity, reduced vocal inflection, and impoverished speech (Kirkpatrick et al., 2006). In SSD, motivational negative symptoms in particular are associated with declines in psychosocial functioning (Fervaha et al., 2015; Pillny & Lincoln, 2016), lower cognitive functioning (Bora et al., 2017; Pillny et al., 2022), and psychotic prodromes (i.e., early warning signs of exacerbating SSDs) and may predict the transition from at-risk mental states to acute psychotic episodes (Bang et al., 2019; Radua et al., 2018).

Research suggests that amotivational symptoms are not confined to specific disorders but can be observed on a continuum from normal functioning to a range of pathological conditions (e.g., Chan et al., 2024; McCarthy et al., 2015; Riehle et al., 2022, 2024; Strauss & Cohen, 2017). Although amotivational psychopathology may present with similar phenomenology across diagnostic categories and their continua, its underlying mechanisms within and across diagnostic categories remain largely unclear. This limited mechanistic understanding of amotivational psychopathology constrains the effectiveness of current interventions and hampers the development of tailored, more effective approaches for addressing amotivation and anhedonia in specific populations (e.g., Cella et al., 2023).

Effort-Based Decision Making: Conceptual and Psychobiological Foundations and Relevance to Mental Health

Recent accounts of amotivational symptoms converge in arguing that several reward-related processes are involved in diminished motivation, which can be integrated into a conceptual framework of EBDM (Husain & Roiser, 2018; Pessiglione et al., 2017; Treadway & Zald, 2013; Whitton et al., 2015). EBDM entails the cognitive process of weighing the costs, such as behavioral or cognitive effort, against the anticipated rewards associated with a particular action, while also considering the probability with which an expected reward will be retrieved. Adaptive EBDM would thus result in increased exertion of effort as reward and probability magnitudes increase. For example, deciding to organize a group outing with friends involves weighing the effort needed to coordinate schedules and plan the event against the anticipated rewards of quality time, shared experiences, and strengthened friendships. If one finds the effort manageable and the rewards promising, one may be more inclined to proceed. Conversely, if one feels overwhelmed or doubtful about the anticipated enjoyment, one will more likely opt out. Thus, EBDM can be seen as the result of an internal cost–benefit computation that is highly relevant to human motivation and engagement in goal-directed behavior.

Central to this perspective is the understanding that EBDM encompasses multiple stages of reward processing (e.g., Treadway & Zald, 2013). Reward processing is essential for motivating behavior, learning from experience, and guiding decision making. It enables individuals to anticipate, pursue, and derive pleasure from positive stimuli, thereby supporting goal-directed actions. Despite these diverse functions, reward processing deficits have traditionally been viewed as a unitary construct: a reduced ability to experience pleasure in response to rewarding stimuli (for a historical review, see Strauss & Cohen, 2018). Although this view may seem plausible at first glance, it has been criticized as being too simplistic (e.g., Treadway & Zald, 2011, 2013). One objection is that reduced motivation for a reward does not necessarily coincide with a reduction in pleasure derived from the reward (e.g., one can find it difficult to get up to play a sport even though they generally enjoy the sporting activity). Conversely, a goal may still be pursued even when the pleasure derived from attaining the reward has long since dissipated (e.g., a marathon is completed even though running is much less enjoyable after the first quarter of the distance). In fact, extensive research has shown that reward processing extends far beyond the mere experience of pleasure in response to the receipt of a reward (for a review, see Glazer et al., 2018). Instead, reward processing is better conceptualized as a multifaceted construct involving several distinct stages and psychological processes that unfold dynamically over time and correspond to disparate neural mechanisms (Berridge & Kringelbach, 2008; Berridge et al., 2009; Knutson et al., 2001). This multidimensional perspective on reward processing provides a framework for examining motivation through EBDM across reward stages. Although different authors lack agreement in terminology and classification of these phases, they can be categorized into reward anticipation, reward consumption, effort computation, cost–benefit analysis, and reward learning (Berridge et al., 2009; Craske et al., 2024; Halahakoon et al., 2020; Nusslock & Alloy, 2017).

A considerable body of research has elucidated the neurobiological foundations of reward processing and EBDM. A recent systematic review of human neuroimaging studies investigating the neural circuits underlying EBDM identified critical fronto-striatal networks, including the ventral striatum, ventromedial prefrontal cortex, and dorsal anterior cingulate cortex, as pivotal for representing reward values and effort costs, as well as integrating this information (Brassard et al., 2024). Specifically, the ventromedial prefrontal cortex has been shown to encode subjective reward value signals that integrate both reward value and effort costs. Another key region implicated in EBDM is the dorsal anterior cingulate cortex (also referred to as the dorsomedial prefrontal cortex). Nevertheless, its precise role remains debated, as it has been associated with overlapping functions, such as monitoring choices, encoding effort demands, and tracking effort investment during decision making (Clairis & Pessiglione, 2024; Hogan et al., 2018). Moreover, Brassard et al. (2024) suggested that the ventral striatum plays a critical role in encoding expected subjective reward values, particularly in relation to reward magnitude. A more nuanced pattern emerges from a study suggesting that distinct striatal subregions encode the motivation to initiate movement, reward valuation, and the effort costs (Suzuki et al., 2021). Thus, beyond the encoding of reward prediction, the ventral striatum integrates cost–benefit information into a unified valuation signal. Additional regions implicated in EBDM include the insula, which is thought to encode effort valuation independently of reward, and motor areas such as the supplementary motor area, primary motor cortex, and cingulate motor areas, which are involved in preparing for effort expenditure and monitoring physical effort exertion (Brassard et al., 2024).

Beyond structural and functional neuroanatomy, there is robust evidence identifying dopamine as a key neurotransmitter modulating EBDM (Salamone & Correa, 2024). Dopamine depletion and dopamine antagonists have consistently been shown to bias choices toward low-effort options, in both animal (e.g., Robles & Johnson, 2017) and human studies (e.g., Berg et al., 2022). Conversely, dopamine agonists appear to have the opposite effect, enhancing

effort exertion, particularly for higher rewards (Bardgett et al., 2009; Soder et al., 2021; Treadway, Bossaller, et al., 2012). Dysfunctions in the dopaminergic system have also been implicated in several mental health conditions, particularly SSD, BD, and MDD (Ashok et al., 2017; Howes & Kapur, 2009; Mann & Kapur, 1995). While amotivational symptoms in these disorders are often linked to dopaminergic hypofunction, positive symptoms and manic or hypomanic states are associated with dopaminergic hyperfunction (Argyropoulos & Nutt, 2013; Arjmand et al., 2018; Berk et al., 2007). Nonetheless, it is important to note that neurotransmitters beyond dopamine also modulate EBDM. Acetylcholine and the catecholamines norepinephrine and serotonin have been shown to play significant roles in effort valuation and decision-making processes (Borderies et al., 2020; Erfanian Abdoust et al., 2024; Meyniel et al., 2016).

To interpret the findings from neuroimaging and neurochemical research, particularly considering mechanisms underlying amotivational psychopathology, EBDM must be investigated at the behavioral level. Investigating EBDM at the behavioral level enables identification of behavioral patterns in EBDM specific to the domains of psychopathology and to disentangle the cognitive operations involved.

Experimental Tasks for Assessing Effort-Based Decision Making

Over the past 2 decades, several experimental tasks have been designed to measure EBDM (for a review, see Bogdanov & Pizzagalli, 2025; Hartmann-Riemer et al., 2018). A common feature of these tasks is that participants decide whether, or to what extent, to exert effort for a reward. Depending on the paradigm, effort demand, reward magnitude, and reward probability can be systematically manipulated to assess their influence on EBDM. Furthermore, the specific design of EBDM tasks varies considerably, potentially influencing individual task behavior and likely the associated neural substrates. Key design features include the type of effort required, which can be physical (e.g., button presses), cognitive (e.g., card sorting), or perceptual (e.g., stimulus detection against different contrast levels); the nature of the reward, which may consist of money, food, tokens, or social incentives such as positive feedback or happy faces; the structure of reward and probability conditions, which can vary across trials; the calibration of required effort to an individual's performance; and the extent to which participants receive their earned rewards after completing the task. Despite these variations, EBDM tasks can be broadly categorized into three types of paradigms.

Dual-Choice Paradigms

In dual-choice paradigms, participants must choose between performing a low- or high-effort task to receive different levels of reward, with some tasks also including different reward probabilities. The most widely used task is the Effort Expenditure for Reward Task (EEfRT; Treadway et al., 2009). The EEfRT is a computerized effort-based decision-making paradigm with monetary rewards. In each trial, participants choose between an easy option (30 button presses in 7 s for \$1) and a hard option (100 presses in 21 s for higher rewards). The magnitude of rewards assigned to the hard task ranges from low (\$1.24–\$2) to moderate (\$2.01–\$3) and high (\$3.01–\$4.12). The probability of obtaining the reward varies across the trials (low probability 12%; medium 50%; high 88%). At the beginning of each trial, participants are presented with the two choice alternatives (easy vs. hard task) together with the reward magnitude assigned to each task and the reward probability of the trial. Once participants have chosen one of the options, visual feedback on their progress in performing the respective task is provided on the screen as a white bar that fills up with each button press. Participants receive feedback on whether they successfully performed the task they chose and how much they have earned.

Another example of a dual-choice paradigm is the Balloon Effort Task (Gold et al., 2013). In the Balloon Effort Task, participants inflate a virtual balloon by pressing a button until it pops. Each trial offers an easy option (e.g., 20 presses for a fixed low reward, such as \$1) and a hard option (e.g., 100 presses for a larger reward, such as \$3–\$7). Reward probability (e.g., 50% vs. 100%) can be manipulated across trials. Balloon inflation is shown in real time, and participants receive feedback about their earnings after each trial (Gold et al., 2013).

A third dual-choice paradigm, the Deck Choice Task, requires participants to exert cognitive instead of physical effort to obtain a reward (Kool et al., 2010). Here, participants choose between playing an easy or hard deck of cards. Each deck contains 10 cards in two colors (orange and blue), numbered 1–9. In each trial, they must make quick judgments based on the card's number and color: For blue cards, they indicate whether the number shown is less than or equal to five; for orange cards, they determine whether the number is even or odd. Easy decks contain only one color and offer a fixed low reward, while hard decks include both colors (requiring switching between tasks, thereby increasing cognitive demands) and provide variable rewards.

Across all dual-choice paradigms, the primary outcome measure is the proportion of high-effort choices. The proportion of high-effort choices can be examined within each reward/probability condition and across different reward magnitudes and probabilities to assess how effort allocation varies with increasing reward and probability.

Effort-Discounting Paradigms

Effort-discounting tasks constitute a specialized form of dual-choice paradigms. Effort-discounting tasks allow researchers to quantify how much subjective value of a reward is lost as effort demands increase and to derive an individual discounting curve that characterizes "effort sensitivity." As in the classical dual-choice tasks, participants are presented with two decision alternatives, including a low-effort and a high-effort option. The crucial distinction between the two paradigms is that effort-discounting tasks primarily manipulate the effort component of the high-effort option while maintaining a constant reward magnitude. In addition, some effort-discounting tasks adjust the reward magnitude of the low-effort option across trials, a procedure which is known as titration. In titration procedures, the reward for the low-effort option is increased each time the high-effort option is chosen. This continues until the "indifference point" is reached, where the participant is equally likely to choose either option. Plotting these indifference points across different effort levels produces the discounting curve, reflecting how steeply reward value declines with increasing effort. Two key indices of EBDM can be derived: the proportion of effortful choices across increasing effort conditions (e.g., Culbreth,

Chib, et al., 2024) and the indifference point (e.g., Docx et al., 2015; Hartmann et al., 2014). The indifference points determined across different effort levels parameterize the resulting discounting curve, which is often modeled using a hyperbolic function. This function yields the *k* parameter, which provides an index for the steepness of the devaluation of subjective reward utility due to increasing effort (e.g., Ang et al., 2023).

Effort discounting paradigms can be applied to physical or cognitive tasks. In physical effort-discounting tasks, effort is typically operationalized as hand-grip force measured with a dynamometer, with individual effort levels calibrated to a participant's maximum voluntary contraction (e.g., Hartmann et al., 2014). On each trial of a physical effort-discounting task, participants choose between a low-effort option associated with a smaller reward and a high-effort option that requires exerting greater grip force for a larger fixed reward. The reward magnitude of the low-effort option is adjusted across trials until the subjective indifference point is reached.

An example of a widely used cognitive effort-discounting paradigm is the Cognitive Effort Discounting Task (Westbrook et al., 2013). The Cognitive Effort Discounting Task is based on the *n*-back task, a working memory paradigm in which participants monitor a sequence of stimuli (e.g., letters) and indicate when the current stimulus matches one presented *n* trials earlier. Across trials, the value of *n* is systematically increased to raise cognitive demand. In the Cognitive Effort Discounting Task, participants first complete practice blocks at different *n*-back levels to familiarize themselves with task difficulty. During the subsequent discounting phase, they make repeated choices between performing a low-demand 1-back task for a smaller reward and a higher demand *n*-back task (e.g., 2-back to 5-back) for a fixed larger reward. The reward associated with the 1-back option is titrated trial-by-trial until the subjective indifference point is reached.

Progressive Ratio Paradigms

Progressive ratio tasks assess EBDM by measuring the amount of effort participants are willing to exert before ceasing to work for a reward. Unlike dual-choice paradigms, progressive ratio tasks do not present alternative options. Instead, participants are required to repeatedly perform a single task with progressively increasing effort demands across trials. Trials are typically grouped into blocks, defined as consecutive trials performed under identical reward conditions. Within a block, the reward magnitude remains constant, whereas in some versions of these tasks, the reward magnitude may increase across blocks but never within a block. The trial at which a participant stops responding is referred to as the individual breakpoint. The individual breakpoint reflects the point where the required effort outweighs the perceived reward value. Effort can be physical (e.g., repeated button presses or handgrip exertions; Klawohn et al., 2022; Strauss et al., 2016) or cognitive (e.g., solving increasingly difficult arithmetic problems; Hershenberg et al., 2016). To illustrate, in a physical progressive ratio task, participants are required to repeatedly press a button to obtain a reward. While the reward (e.g., $2) is held constant, the number of required button presses increases across the trials within a block. Thus, participants are initially required to complete a small number of presses to obtain a reward, but the required number increases exponentially in subsequent trials (e.g., 5, 10, 20, 40, 80 presses per reward).

Differentiation Between Effort-Based Decision-Making Tasks and Related Paradigms

Some researchers adopt a broader definition of EBDM tasks, incorporating paradigms like delay discounting, reward learning, and decision-making under uncertainty (Brassard & Balodis, 2021; Brassard et al., 2024). This broader perspective is justified by shared cost–benefit trade-offs and significant associations between these constructs (Escobar et al., 2023; Malesza & Ostaszewski, 2013). Nonetheless, research suggests that these paradigms are at least partly governed by dissociable neural mechanisms, necessitating clearer differentiation between tasks assessing EBDM and those tapping into related, yet distinct constructs (Prévost et al., 2010).

A key distinction concerns delay discounting tasks, where individuals choose between a smaller immediate reward and a larger delayed reward (Matta et al., 2012). While the decision to wait might involve patience or self-control, it primarily reflects the trade-off between reward magnitude and delay rather than motivation to actively exert effort. Similarly, probabilistic learning tasks like the Iowa Gambling Task assess risk-based decision making under uncertain circumstances (Bechara et al., 1994). Participants must learn to maximize rewards by adjusting their choices based on unknown and varying reinforcement contingencies. Unlike EBDM tasks, these paradigms primarily measure reward and punishment sensitivity, governed more by learning, attentional, and problem-solving processes than by effort-cost decisions (Gansler et al., 2011).

Thus, despite some conceptual overlap, these paradigms tap into distinct psychological constructs underpinned by different neurobiological mechanisms. While EBDM is closely linked to the fronto-striatal network, delay discounting and probabilistic learning are more strongly associated with prefrontal-executive and limbic processes facilitating impulse control and risk evaluation (Aram et al., 2019; Chen et al., 2021).

Psychometric Properties of EBDM Task Outcomes

Effort-based decision-making tasks have been evaluated for their psychometric properties including reliability, validity, and inter-correlations across paradigms. Several studies have examined the internal consistency of these tasks. For example, the EEfRT showed high split-half reliability ($r = .87–.96$) in large community samples (Ohmann et al., 2022). Ohmann et al. (2022) also confirmed the internal validity of the EEfRT, showing that high-effort choices increased as a function of reward magnitude and probability, which was independent of motor ability and task length.

Furthermore, research has demonstrated significant correlations between EBDM task performance, interview-based measures, and ecological momentary assessment of amotivational psychopathology, whereas self-report measures of motivation and amotivation often showed weaker or nonsignificant associations (Horan et al., 2015; Luther et al., 2018; Moran et al., 2017; Ohmann et al., 2022; Renz et al., 2023). A previous meta-analysis examining correlations among different motivation measures in SSD suggests that various EBDM paradigms capture overlapping but distinct components of motivation (Luther et al., 2019). Specifically, effort-discounting tasks have shown stronger associations with amotivational psychopathology in SSD compared to other paradigms.

Intertask comparisons revealed small-to-moderate correlations across different EBDM tasks, both in healthy community samples

(Renz et al., 2023) and in SSD and healthy controls, indicating convergent validity (Culbreth et al., 2023; Horan et al., 2015). Given the at most moderate correlations, this evidence suggests that while EBDM tasks share considerable overlap, they may also tap into distinct aspects and mechanisms of motivated behavior.

Together, these findings highlight the reliability, as well as the internal, criterion, convergent, and external validity of EBDM tasks, while also underscoring their heterogeneity. The unifying feature of these tasks is that they assess real-time effort allocation in response to varying incentives or effort demands. Therefore, EBDM tasks provide an observable and quantifiable measure of motivated behavior, independent of self-report biases. Unlike self-reported motivation, which can be influenced by cognitive distortions, low insight, or social desirability effects, EBDM tasks directly measure decision-making processes under controlled experimental conditions. This objectivity has led some researchers to promote EBDM tasks as valuable tools for assessing amotivational psychopathology in psychiatric populations (Green & Horan, 2015; Green et al., 2015; Hartmann-Riemer et al., 2018). However, the variability across paradigms and the relatively modest intercorrelations between tasks suggest that no single paradigm can comprehensively capture EBDM or its underlying motivational processes. Given this heterogeneity, a meta-analytic approach is needed to systematically quantify the magnitude of EBDM alterations across psychiatric populations and task designs. Examining how specific task characteristics (e.g., reward type, probability structure, effort modality) shape behavioral patterns may clarify mechanisms underlying EBDM impairments and guide the development of more standardized, clinically relevant assessment tools.

Computational Modeling: A New Avenue for Investigating Mechanisms Underlying Behavioral Patterns in Effort-Based Decision-Making Tasks

Computational modeling represents an emerging avenue for investigating behavioral patterns in EBDM. Computational modeling offers a powerful framework for quantifying latent cognitive, motivational, and neurobiological processes that potentially underlie amotivational psychopathology and EBDM (Deserno et al., 2017). Unlike the directly observable task performance (e.g., the proportion of high-effort choices), computational models decompose decision making into mechanistic components such as effort sensitivity, reward valuation, or probability weighting. Computational modeling works by fitting mathematical models to behavioral data and to infer the underlying latent cognitive mechanisms that drive behavioral parameters (Pessiglione et al., 2018). Computational models are typically built on principles of reinforcement learning, utility maximization, or Bayesian inference (Friston et al., 2014; Wilson & Collins, 2019) and provide a framework to understand how individuals integrate effort, reward, and probability information when making decisions (Stephan & Mathys, 2014). For example, drift-diffusion models estimate the speed and accuracy of evidence accumulation before a decision, while reinforcement learning models quantify how past outcomes shape future effort allocation. Moreover, computational phenotyping can be used to classify individuals into clusters of specific parameter combinations to yield distinct mechanistic decision-making profiles.

Three key processes have been proposed to account for alterations in EBDM: reward sensitivity (i.e., estimating the expected value of an action/reward), effort sensitivity (i.e., assessing the effort cost required to achieve the reward), and cognitive operations (e.g., weighing expected reward values against expected costs to decide whether to act; Gold et al., 2015). One might question whether dysfunctions in these mechanisms represent transdiagnostic features or whether there are disorder-specific pathways to aberrant EBDM and amotivational psychopathology (for a review, see Strauss & Cohen, 2017). Moreover, the field lacks a systematic synthesis of the evidence from computational modeling studies investigating mechanisms underlying altered EBDM in psychopathology.

While computational modeling may provide deeper insights into EBDM mechanisms, its parameters cannot be directly aggregated in classical meta-analyses due to variations in model structures, parameter definitions, and task designs. Unlike standardized effect sizes used in traditional meta-analyses, computational parameters are model-dependent and often require study-specific assumptions, making direct comparisons across studies challenging or unfeasible. Nevertheless, a systematic literature review employing narrative synthesis can help to identify overarching patterns and discrepancies in EBDM mechanisms across disorders.

Prior Evidence on EBDM in Mental Disorders

Over the past 15 years, numerous studies have examined EBDM in clinical samples and at-risk populations. Broadly, this research suggests that, compared to healthy controls, these groups exhibit fewer high-effort choices (i.e., effort exertion). More specifically, a common pattern observed in clinical samples is diminished effort allocation, reflected in a reduced increase in high-effort choices as reward and probability magnitudes increase.

A recent meta-analysis of 20 studies compared EBDM task performance between individuals with SSD and healthy controls (HC). Overall, participants with SSD exerted significantly less effort than controls, with a medium effect size (SMD = 0.43, 95% confidence interval, CI [0.30, 0.56]; Blouzard et al., 2023). Indeed, in the subgroup analyses, Blouzard et al. (2023), found that while SSD and HC groups did not significantly differ in trials with low-reward magnitude and probability (k = 9, SMD = −0.03, 95% CI [−0.39, 0.34]), SSD participants showed significantly reduced rates of high-effort choices in trials with medium- (k = 13, SMD = 0.39, 95% CI [0.22, 0.56]) and high-reward magnitude and probability (k = 10, SMD = 0.87, 95% CI [0.60, 1.13]), suggesting that individuals with SSD deviate most from normative effort allocation precisely when exerting effort is most advantageous (i.e., when potential rewards are high and certain). This finding is particularly paradoxical because, if decision making were guided by maximizing reward relative to effort expenditure, high-reward trials should be the primary instances in which participants select the high-effort options. By contrast, individuals with SSD do not adjust their effort allocation in accordance with increasing reward and probability. Moreover, Blouzard et al. found significant correlations between overall reductions in EBDM and negative symptoms in SSD (r = −.33), suggesting that this attenuated response to high-reward trials may reflect a core mechanism underlying amotivational psychopathology.

However, Blouzard et al. pointed out that their meta-analysis was limited by the relatively small number of included studies, particularly in the subgroup analyses, which was mainly because many authors did not report means and variances of outcomes. The small number of included studies might have produced an incomplete picture of the evidence and could account for the significant and unexplained between-study heterogeneity, which hindered a precise disentanglement of effort allocation across task conditions. A reliable investigation of effort allocation across task conditions would necessitate more data and a multilevel meta-regression approach predicting effect sizes across task conditions. Software-assisted data extraction from plotted data could bypass the incomplete reporting and authors not responding to data requests in many cases which could increase the amount of available data. A number of these software solutions are available and have been shown to yield high accuracy and interrater reliability (Aydin & Yassikaya, 2022; Flower et al., 2016; Kadic et al., 2016).

With regard to transdiagnosticity, although systematic reviews have provided narrative syntheses, suggesting that individuals with MDD exhibit EBDM patterns similar to those observed in SSD (e.g., Horne et al., 2021), the field lacks a meta-analytic aggregation of EBDM in MDD, BD, and at-risk samples to pinpoint transdiagnostic and disorder-specific mechanisms underlying amotivational psychopathology. For instance, a systematic review of EBDM studies in SSD and MDD suggests that reduced EBDM in SSD may be largely driven by impaired cognitive functioning, whereas in MDD, this reduction in EBDM may primarily reflect diminished reward responsivity, indicating distinct pathways underlying EBDM deficits across these disorders (Culbreth et al., 2018). Similarly, it has been proposed that while amotivational features in MDD are linked to reward hyposensitivity, BD may be characterized by reward hypersensitivity, which drives increased motivation during (hypo-) manic episodes (Nusslock & Alloy, 2017).

To clarify these differential mechanisms and identify transdiagnostic features, a crucial next step is to meta-analytically compare the overall EBDM reductions across SSD, MDD, and BD while also examining group-specific effort allocation patterns across task conditions (i.e., variations in effort exertion as a function of reward and probability magnitudes by diagnostic group). Furthermore, extending this investigation to at-risk populations could provide valuable insights into whether EBDM reductions reflect a predisposing vulnerability to amotivational psychopathology or emerge as a result of disorder progression. Although recent studies have begun to explore EBDM from a transdiagnostic perspective and have highlighted potential similarities and differences between SSD, MDD, and BD (e.g., Barch et al., 2023), a meta-analytic synthesis of these findings is currently missing.

The Case for a Cross-Diagnostic Approach to EBDM and Amotivational Psychopathology

In recent years, there has been growing interest in transdiagnostic approaches to amotivational symptoms (Husain & Roiser, 2018; Moritz et al., 2018; Nusslock & Alloy, 2017; Strauss & Cohen, 2017). According to the Research Domain Criteria Initiative (Insel et al., 2010) and the Hierarchical Taxonomy of Psychopathology (Conway et al., 2019), the term "transdiagnostic" refers to factors, processes, or symptoms that cut across multiple diagnostic categories rather than being specific to a single disorder. The transdiagnostic perspective has introduced a "tectonic shift" to the field of psychopathology as it challenges the traditional and rather arbitrary categorical approach of the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (American Psychiatric Association, 2013), and the *International Statistical Classification of Diseases and Related Health Problems*, 10th revision (World Health Organization, 2016). In contrast to these categorical diagnostic systems, in which mental health conditions are classified as distinct entities, transdiagnostic models focus on underlying pathophysiological mechanisms that are common across a wide range of disorders (Insel et al., 2010). A transdiagnostic approach is particularly relevant to amotivational symptoms, as these symptoms occur across multiple mental health conditions and present with similar phenomenology. Although this may suggest transdiagnostic pathomechanisms, a common clinical phenotype does not necessarily imply a common underlying mechanism. According to the principle of equifinality, similar motivational impairments may arise from distinct processes across different disorders (Nusslock et al., 2025).

The Present Study

In the current meta-analysis, we aggregated available behavioral data on EBDM and aimed to (a) investigate the magnitude of EBDM reductions across SSD, MDD, BD, and at-risk samples compared to HC, (b) test whether the magnitude of EBDM reductions differs between diagnostic groups, (c) test for differences in effort allocation across task conditions between diagnostic groups, and (d) examine whether the effect size between clinical and nonclinical groups varies by specific task (e.g., monetary vs. social reward) or sample characteristics (e.g., symptom profiles, cognitive functioning, demographics). Additionally, we aimed to provide a comprehensive narrative review of computational modeling studies to enhance understanding of the mechanisms underlying EBDM deficits and to identify commonalities and discrepancies across psychiatric disorders.

By addressing these aims, this study offers a novel and comprehensive synthesis of EBDM in mental disorders associated with disruptions in motivation. Going beyond previous work, it adopts a cross-diagnostic perspective to systematically analyze effort allocation across task conditions while also examining specific task and sample characteristics as potential moderators of EBDM reductions in the context of amotivational psychopathology.

Method

Transparency and Openness

This meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (Page et al., 2021) and adheres to the Meta-Analytic Reporting Standards (Appelbaum et al., 2018). The original protocol was preregistered with PROSPERO (CRD42022344605). Deviations from the original protocol were proposed during peer review, including expanding the study population beyond SSD and MDD and incorporating a review of computational modeling studies. All study materials, including data sets and analysis scripts, are available online (https://osf.io/69exu/; Pillny, 2025).

Search Strategy and Selection Criteria

PubMed, APA PsycInfo, Embase, and Web of Science were searched on July 26, 2022, and updated through November 11, 2024, using the following search terms: (effort-based decision-making) OR ("effort" OR "effort-based decision-making" OR cost-benefit OR discounting) AND (anhedonia OR motivation OR avolition OR apathy OR schizophren* OR psychotic OR psychos* OR depress* OR affective disorder OR mood disorder OR bipolar). Searches included peer-reviewed publications, published dissertations, preprints, and conference proceedings written in either English or German. Reference lists of included studies, relevant reviews, and Google Scholar were used for manual searches.

Titles and abstracts were screened by KER and MP. Full-text screenings and eligibility assessments were performed by two independent researchers and double-checked by MP. Studies were eligible if they examined differences in hard task choices, indifference points, or progressive ratio breakpoints in any EBDM task. Eligible studies included comparisons between individuals with SSD, major depressive disorder, bipolar disorders, or those at elevated risk for these conditions or amotivational psychopathology, and healthy controls. Studies that directly compared EBDM outcomes between two or more (sub-)clinical samples were also included for direct cross-transdiagnostic comparisons. Eligible EBDM tasks were defined as paradigms that included a decision of whether or how much effort (either physical, cognitive, or perceptual) to exert to obtain a certain amount of reward. The paradigm needed to include at least two task conditions (e.g., easy and hard) or increasing effort levels and to assign different levels of reward and/or probability to the task. Tasks tapping into related yet distinct constructs such as delay discounting, probabilistic learning, or risk aversion under uncertainty without requiring active effort exertion were not considered EBDM tasks and were not included in this analysis.

Risk of Bias

Two researchers independently evaluated the risk of bias for each study with the adapted version (Pillny, 2024) of the Quality Assessment Tool for Quantitative Studies (Thomas et al., 2004). Inconsistencies were resolved by discussion and, if needed, by including a third evaluation by DF and MP. Rating domains included potential sources of bias in sample selection, study design, confounding variables, data collection procedures, and statistical analyses. Each dimension was evaluated on a scale from 1 (*low risk*) to 3 (*high risk*). For further analyses, dimension scores were averaged and rounded to whole numbers on the study level (i.e., 1 = *low risk*; 2 = *moderate risk*; 3 = *high risk*).

Data Extraction and Effect Size Calculation

Two reviewers independently extracted data. MP checked the final data set for inconsistencies. Where data were not reported, corresponding authors were contacted to obtain additional information. In cases of nonresponse but available plotted data, data were extracted from graphs ($k = 12$) using the open-source PlotDigitizer software (Version 2.6.9). Multiple studies have demonstrated near-perfect reliability and validity for graph-digitizing software, with exceptionally high correlation coefficients, demonstrating strong alignment between digitized data and original values or effect sizes derived from published studies, as well as high accuracy (e.g., Kadic et al., 2016). Specifically, comparisons of software-extracted data with hypothetical data sets and effect size calculations achieved Pearson correlation coefficients of up to $r = .99$ (Aydin & Yassikaya, 2022). Similarly, another study comparing two software programs with manual data extraction demonstrated high interrater reliability ($r > .99$) and exceptionally high correlations with known data ($r > .99$), ensuring consistent results across different raters and software tools (Flower et al., 2016). In the present meta-analysis, four studies provided both graphical and numerical data as means and variance measures. We compared the software-extracted data with the original values and found an almost identical overlap, with negligible deviations beyond the third decimal place. Together with prior research, this overlap supports the conclusion that software-based data extraction is a highly reliable and valid method for converting graphical data into usable numerical formats, making it an indispensable tool to increase statistical power in meta-analyses by including a substantially larger proportion of the evidence.

For the main outcomes, group-level means and standard deviations of hard task choices or progressive ratio breakpoints across all task conditions were transformed into standardized mean differences (SMD) and corrected for overestimation using Hedges's g. If a study reported more than one relevant effect size, these were extracted separately by assigning a unique effect size identifier. Secondary outcomes included hard task choices, indifference points, and progressive ratio breakpoints for each task condition (e.g., low-reward trials).

Coding of Demographic, Clinical, and Task Characteristics

Demographic sample characteristics included sample size, age, gender (measured as percentage of female participants), education (years), setting (out- vs. inpatient), percentage of participants of minority ethnicity, and medication status (yes/no/partial).

Clinical sample characteristics included symptom severity (depressive, manic, positive, and negative), levels of cognitive functioning (i.e., either specific domains such as working memory and processing speed or composite scores of comprehensive neuropsychological test batteries), and anhedonia. There were three different types of measures assessing anhedonia-related constructs: (a) designated negative symptom scales, (b) designated anhedonia scales, and (c) scales assessing hedonic capacity. Because it is not possible to aggregate the anhedonia scales with the inversely scored hedonic capacity scales into one meta-analytic model and to prevent confounding between negative symptoms and anhedonia scales, all three measures were coded separately.

Task characteristics included paradigm type (dual-choice vs. effort-discounting vs. progressive ratio), effort modality (physical vs. cognitive), effort measure (fixed effort vs. progressive ratio), number of reward conditions, number of probability conditions, reward type (e.g., monetary vs. social), reward payoff (fixed, random, none), and individual task calibration (yes vs. no).

Data Analysis

Statistical analyses were conducted using the "metafor" package implemented in RStudio (Viechtbauer, 2010). Three-level random-effects meta-analytic models with restricted maximum likelihood

estimation were computed to account for the hierarchical data structure, in which multiple effect sizes were nested within studies (e.g., when different EBDM tasks were used or subgroups from the same sample were reported). Group differences in EBDM between (sub-)clinical and control groups were tested, with negative effect sizes interpreted as reduced EBDM in (sub-)clinical populations. The summary effect sizes were calculated based on 95% confidence intervals. Between-study heterogeneity was assessed using the Q-statistic and the I^2 index. Due to the lack of available methods for assessing publication bias and conducting sensitivity analyses in three-level meta-analytic models, visual inspection of funnel plots and rank correlation tests were used to examine publication bias. In addition, Egger's regression test was performed on the main model with a two-level random-effect structure (for guidelines, see Harrer et al., 2021). When there was evidence of funnel plot asymmetry, the trim-and-fill method was applied to estimate the potential impact of publication bias and adjust for missing studies (Shi et al., 2019). Sensitivity analyses were done according to the outlier and influence diagnostics procedure for meta-analyses (Viechtbauer & Cheung, 2010). Significant outliers were tested for their influence on the main findings using the leave-one-out method (Viechtbauer, 2021). Moderator analyses were conducted using three-level meta-regression with random effects and restricted maximum likelihood estimation. All numerical moderator variables were z-standardized to facilitate comparability of effect sizes across moderators. Meta-regressions were calculated (a) across all samples, (b) across all "clinical" samples excluding AR samples, and (c) separately for each diagnostic group.

Results

Study Selection and Sample Characteristics for Meta-Analysis

The systematic literature search initially identified 97,651 records. After removing duplicates, 83,062 records remained and were screened based on title and abstract. Of these, 218 underwent full-text screening, with 73 studies meeting the inclusion criteria for the meta-analysis. Five eligible studies could not be included due to unavailable statistics and lack of responses from corresponding authors (M. Y. Chu et al., 2020; Docx et al., 2015; Suchting et al., 2023; Westbrook et al., 2023; Yang et al., 2016), resulting in 68 studies included in the analysis (see Figure 1).

The studies were geographically distributed as follows: 33 in North America, 19 in Europe, 16 in Asia, and one in New Zealand. The settings comprised outpatient facilities (k = 33), inpatient facilities (k = 3), or a combination of both (k = 7). A total of 115 effect sizes were retrieved from the included studies, comprising 3,697 participants diagnosed with SSD, MDD, or BD or identified as at risk for these conditions and 4,096 healthy controls. Across diagnostic categories, effect sizes were distributed as follows: 51 effect sizes from 1,645 participants with SSD, 27 effect sizes from 967 participants with MDD, 12 effect sizes from 404 participants with BD, and 25 effect sizes from 681 participants from "at-risk groups." Among the at-risk samples, nine studies focused on participants with elevated psychotic experiences or ultra-high risk for SSD or BD, while 11 studies examined those with elevated anhedonia, amotivation, or subthreshold depression. Twelve direct within-study comparison effect sizes were available for SSD (n = 461) versus MDD (n = 522), 11 effect sizes for SSD (n = 380) versus BD (n = 416), and 11 effect sizes for BD (n = 394) versus MDD (n = 414). Only two studies compared an at-risk sample to diagnosed participants (Valton et al., 2025; Yang et al., 2014).

Social demographics by group were as follows: 41% of participants in SSD samples were female, 60% in MDD samples, 51% in BD samples, 65% in at-risk samples, and 57% in HC samples. The mean age was 36.25 years (SD = 6.83) for SSD, 34.18 years (SD = 6.14) for MDD, 34.70 years (SD = 4.89) for BD, and 21.64 years (SD = 3.30) for at-risk participants. Mean years of education were 12.80 (SD = 1.17) for SSD, 14.48 (SD = 2.33) for MDD, 14.16 (SD = 1.55) for BD, and 13.78 (SD = 1.55) for at-risk participants. The proportion of participants identifying as ethnic minorities was 44% in SSD, 30% in MDD, 27% in BD, and 38% in at-risk samples. In 50 of 54 (91%) studies investigating clinical samples, *DSM* diagnoses were confirmed using structured clinical interviews. Two studies did not report how diagnoses were assessed, and two relied on psychiatric records. Eleven of the 40 studies (28%) assessing SSD excluded participants with comorbid depression. No study excluded participants taking psychotropic medication.

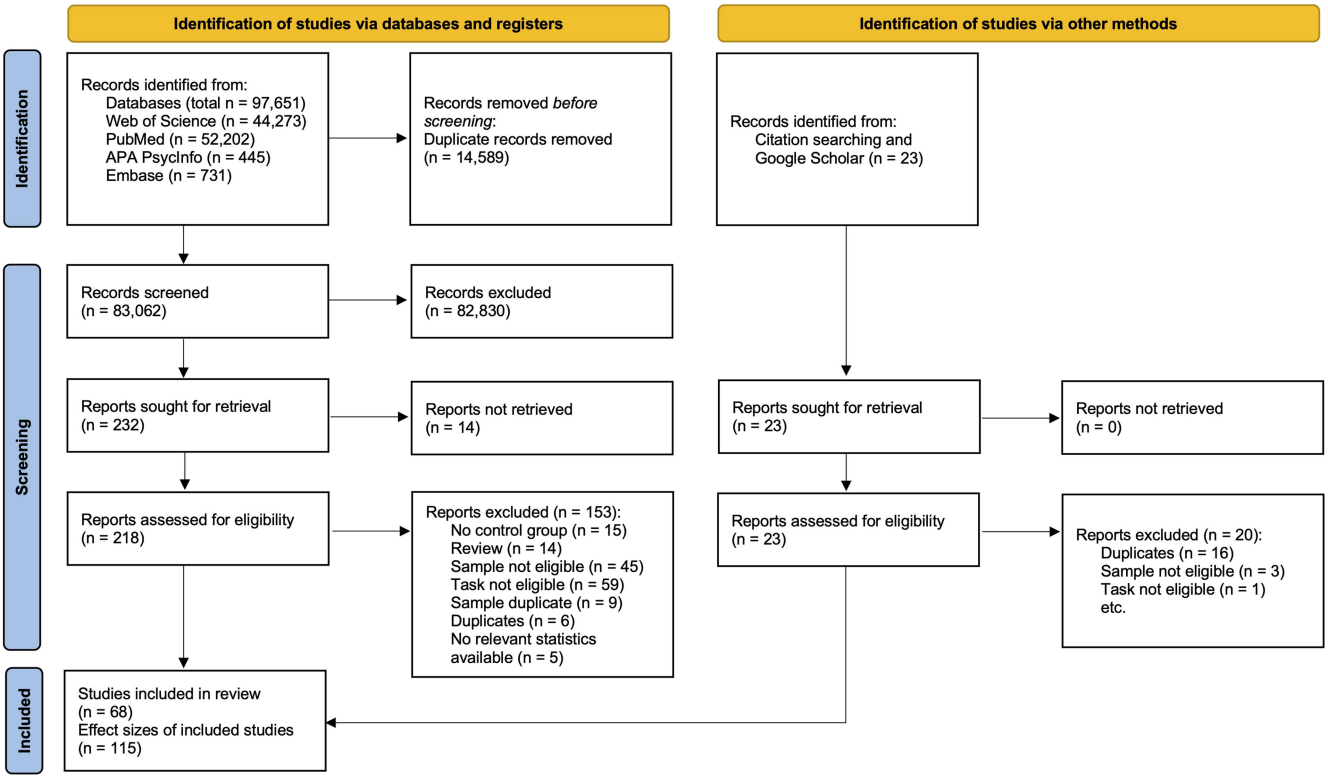
In terms of methodology, most effect sizes (k = 84) were based on classical dual-choice tasks, with additional paradigms including effort-discounting tasks (k = 20), and progressive ratio tasks (k = 11). Measured effort outcomes included hard task choices (k = 88), breakpoints (k = 11), and various cognitive task performances (k = 16). Most tasks required physical effort (k = 94) or cognitive effort (k = 19), with rewards primarily being monetary (k = 100), social (k = 2), or other types (k = 13, including humorous videos or tokens). Detailed study descriptions are provided in Supplemental Table S1.

Meta-Analyses

Overall Differences in EBDM Between (Sub-)Clinical Groups and Controls

Compared to control groups, the clinical and at-risk samples showed a small significant difference in EBDM, suggesting that overall, participants in clinical and at-risk samples exert less effort in EBDM tasks, k = 115, g = −0.32, $t(114)$ = −6.65, p < .001, 95% CI [−0.42, −0.23]. The total between-study heterogeneity was moderate, $Q(114)$ = 298.94, p < .001 with I^2 = 67% (see Supplemental Figure S2 in the online supplement for the decomposition of between-study variance). Visual inspection of the funnel plot, along with the rank correlation test (Kendall's τ = −.16, p = .01), suggested funnel plot asymmetry, indicating that smaller studies tended to report more extreme (negative) effect sizes compared to larger studies (see Supplemental S3). Sensitivity analyses identified the study by J. Wang et al. (2018) as a potential outlier (see Supplemental S4). Leave-one-out analyses showed that excluding Wang et al. had little effect on the summary effect size (g = −0.29, 95% CI [−0.35, −0.22]; see Supplemental S5), suggesting their removal did not substantially alter the results. Yet, influence diagnostics flagged Wang et al. as an extreme outlier. This study showed a very large studentized residual (r = −7.58), strong influence on the model (DFFITS = −0.54, Cook's D = 0.27), and a significant contribution to between-study heterogeneity (i.e., between-study variance: ${\tau_{del}}^2$ = 0.05, *residual heterogeneity test statistic:* QE_{del} = 229.15). Given Wang et al.'s extreme deviation and high impact on model stability, it was excluded from all subsequent analyses.

Figure 1
PRISMA Flowchart



Note. Overview of the study selection process following systematic literature searches. See the online article for the color version of this figure.

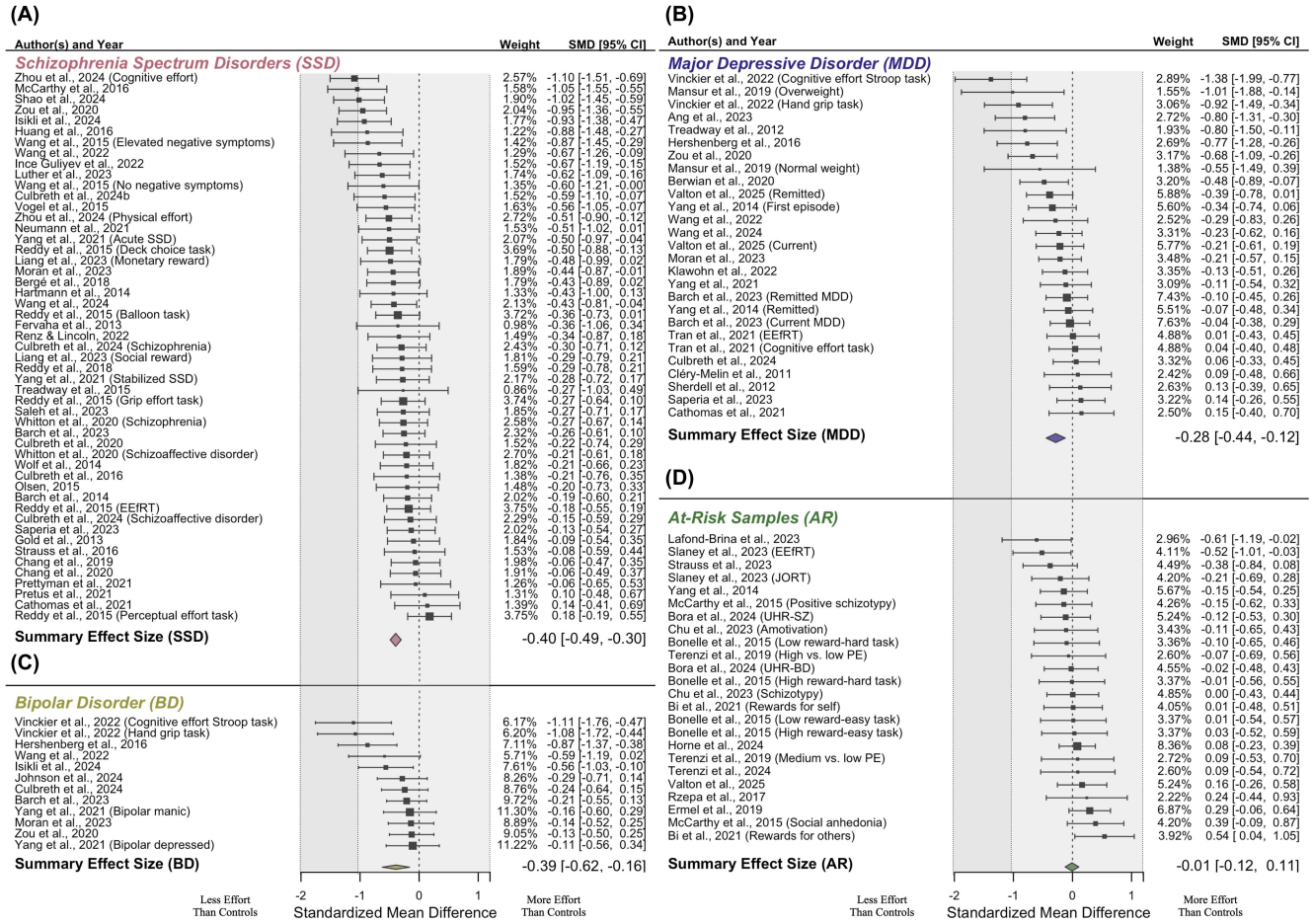
The model excluding J. Wang et al. (2018) showed a significant negative overall summary effect in EBDM, $k = 114$, $g = -0.305$, $t(113) = -7.22$, $p < .001$, 95% CI [−0.38, −0.21], indicating that clinical and at-risk samples exert less effort than controls. The total between-study heterogeneity was moderate, $Q(113) = 229.15$, $p < .001$ with $I^2 = 55\%$ (see Supplemental Figure S6 in the online supplement for the decomposition of between-study variance). Visual inspection of the funnel plot did not indicate publication bias (see Supplemental S7), although the rank correlation test was still significant (Kendall's $\tau = -.14$, $p = .024$), suggesting funnel plot asymmetry. The trim-and-fill method estimated that no studies were missing on the right side, indicating no evidence of publication bias. The adjusted random-effects model showed moderate heterogeneity ($I^2 = 50.59\%$) with the effect size remaining significant and similar in magnitude ($g = -0.29$, $p < .001$) after adjusting for potential bias (see Supplemental S8). Figure 2 depicts the forest plot of the summary effects by diagnostic group. Meta-analyses testing the effect sizes in EBDM separately by diagnostic group revealed a significant moderate summary effect size for SSD, $k = 51$, $g = -0.40$, $t(50) = -8.50$, $p < .001$, 95% CI [−0.49, −0.30]; significant small summary effect sizes for MDD, $k = 27$, $g = -0.28$, $t(26) = -3.62$, $p < .001$, 95% CI [−0.45, −0.12] and BD, $k = 12$, $g = -0.39$, $t(11) = -3.75$, $p < .01$, 95% CI [−0.62, −0.16]; but a nonsignificant summary effect size for at-risk samples, $k = 24$, $g = -0.01$, $t(23) = -0.16$, $p = .880$, 95% CI [−0.13, 0.11]. Between-study heterogeneity was significant and moderate in SSD, $Q(50) = 84.58$, $p < .01$, $I^2 = 40\%$, MDD, $Q(26) = 57.20$, $p < .001$, $I^2 = 60\%$, and in BD studies, $Q(11) = 21.41$, $p = .03$, $I^2 = 55\%$, but nonsignificant in at-risk studies, $Q(23) = 24.37$, $p = .384$, $I^2 = 16\%$. The test of subgroup differences was significant, $Q_M = 7.51$, $df = 3$, $p < .001$. Specifically, when compared to AR, SSD exhibited the largest negative effect, $b = -0.41$, $t(110) = -4.61$, $p < .001$, 95% CI [−0.59, −0.23], followed by BD, $b = -0.28$, $t(110) = -2.54$, $p = .013$, 95% CI [−0.50, −0.06], and MDD, $b = -0.25$, $t(110) = -2.71$, $p = .008$, 95% CI [−0.43, −0.07]. The intercept was not significant, $b = -0.02$, $t(110) = -0.20$, $p = .840$, 95% CI [−0.17, 0.14], suggesting no significant effect for the AR group. An additional exploratory model was conducted categorizing the AR samples into two groups: "elevated risk for psychosis" and "elevated anhedonia/amotivation." The summary effect sizes for both groups were nonsignificant, of similar magnitude, and had almost identical 95% CIs. Specifically, for the elevated risk for psychosis group ($k = 9$), the effect size was $g = -0.01$ (95% CI [−0.21, 0.18]) and $g = 0.00$ (95% CI [−0.18, 0.18]) and for the elevated anhedonia/amotivation group ($k = 15$), suggesting no significant differences between the at-risk categories (see Supplemental S9 for the forest plot).

Within-Study Comparisons Between Clinical Groups

Meta-analyses of studies that directly compared at least two clinical samples showed that SSD exerted significantly less effort in EBDM tasks than MDD with a small effect size, $k = 12$, $g = -0.23$,

Figure 2

Forest Plots of Effect Sizes for Effort-Based Decision Making by Diagnostic Group



Note. Panel A depicts the summary effect for effort-based decision making across all task conditions, comparing individuals with schizophrenia spectrum disorders to controls. Panel B depicts the summary effect for effort-based decision making across all task conditions, comparing individuals with major depressive disorder to controls. Panel C depicts the summary effect for effort-based decision making across all task conditions, comparing individuals with bipolar disorder to controls. Panel D depicts the summary effect for effort-based decision making across all task conditions in at-risk samples (i.e., individuals at increased risk for psychosis or reporting elevated levels of anhedonia, amotivation, or subthreshold depression) compared to controls. Negative effect sizes indicate reduced effort-based decision making in the respective clinical or at-risk group relative to controls. CI = confidence interval; SMD = standardized mean differences; PE = psychotic experiences. See the online article for the color version of this figure.

$t(11) = -3.55$, $p = .005$, 95% CI [−0.37, −0.09], and BD, $k = 11$, $g = -0.27$, $t(10) = -2.38$, $p = .039$, 95% CI [−0.51, −0.02]. The difference between MDD and BD was nonsignificant, $k = 11$, $g = -0.06$, $t(10) = 0.41$, $p = .689$, 95% CI [−0.41, 0.28] (see Figure 3).

Effort Allocation Across Task Conditions

Moderating Effect of Reward Magnitude

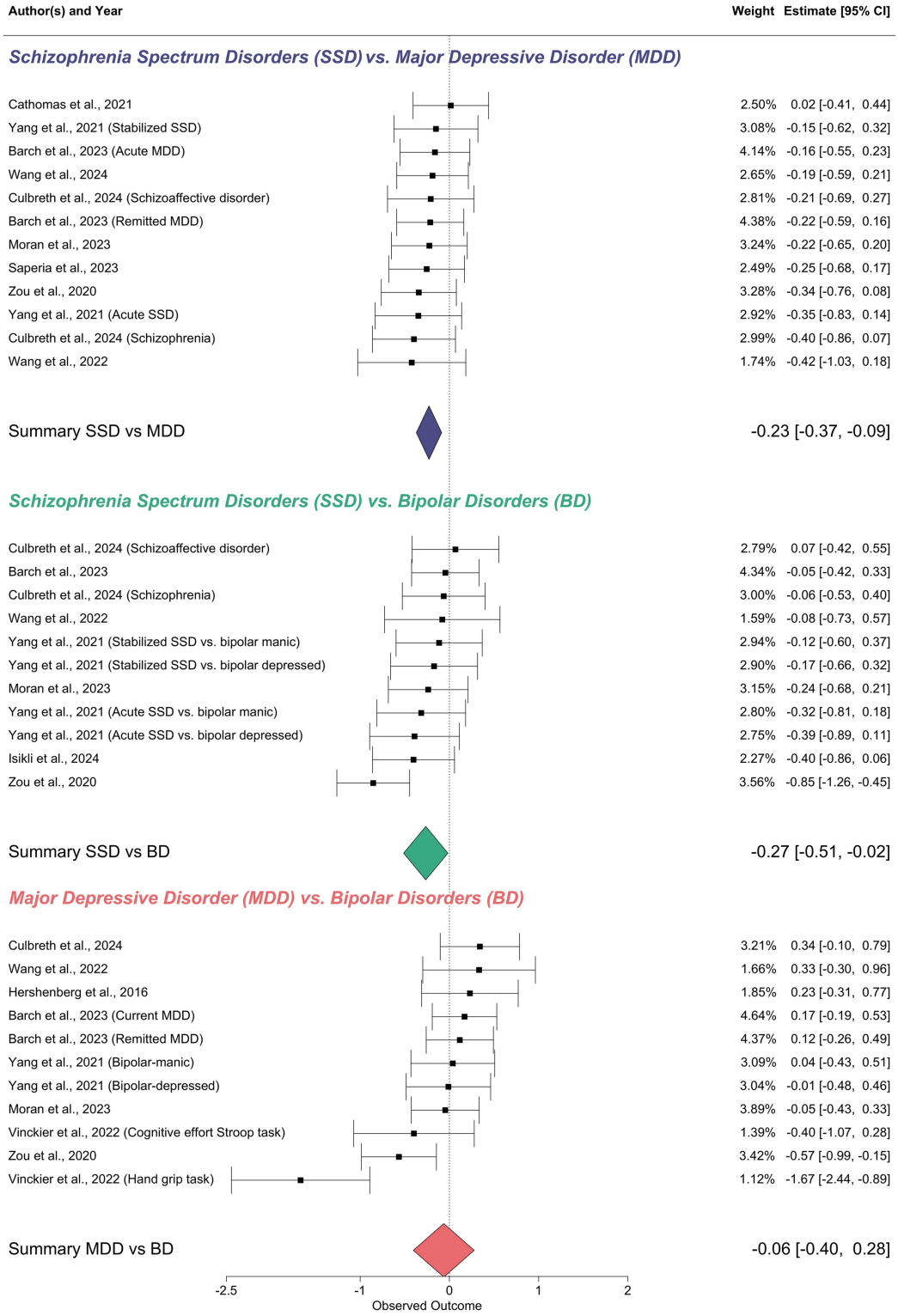
Figure 4A illustrates the effect sizes across diagnostic groups as a function of reward magnitude. The main effect of reward magnitude was significant, $F(2, 220) = 29.59$, $p < .001$, indicating that absolute effect sizes (i.e., differences relative to healthy controls) increased with reward magnitude. Moreover, the interaction between reward magnitude (low, medium, high) and diagnostic group (SSD, MDD, BD, AR) was significant, $F(11, 211) = 9.04$, $p < .001$, suggesting that the pattern of effort allocation across reward magnitudes varied by diagnostic group.

Specifically, the absolute effect size significantly increased from low to medium reward trials in SSD, $k = 66$, $b = -0.17$, $t(4) = -2.84$, $p = .006$, 95% CI [−0.28, −0.05], and in BD, $k = 18$, $b = -0.26$, $t(16) = -2.19$, $p = .044$, 95% CI [−0.50, −0.01], but not in MDD, $k = 34$, $b = -0.004$, $t(32) = -0.05$, $p = .962$, 95% CI [−0.17, 0.17], or AR, $k = 22$, $b = -0.13$, $t(20) = -1.35$, $p = .191$, 95% CI [−0.32, 0.07]. From medium to high reward trials, the absolute effect size further increased significantly in SSD, $k = 66$, $b = -0.165$, $t(64) = -2.84$, $p = .006$, 95% CI [−0.28, −0.05], but not in MDD, $k = 34$, $b = -0.06$, $t(32) = -0.67$, $p = .507$, 95% CI [−0.23, 0.11]; BD, $k = 18$, $b = 0.04$, $t(16) = 0.33$, $p = .743$, 95% CI [−0.21, 0.28]; or AR, $k = 24$, $b = 0.11$, $t(22) = 1.12$, $p = .276$, 95% CI [−0.09, 0.30].

In low-reward trials, effect size magnitudes did not differ significantly across diagnostic groups, $F(3, 77) = 2.37$, $p = .077$. When

Figure 3

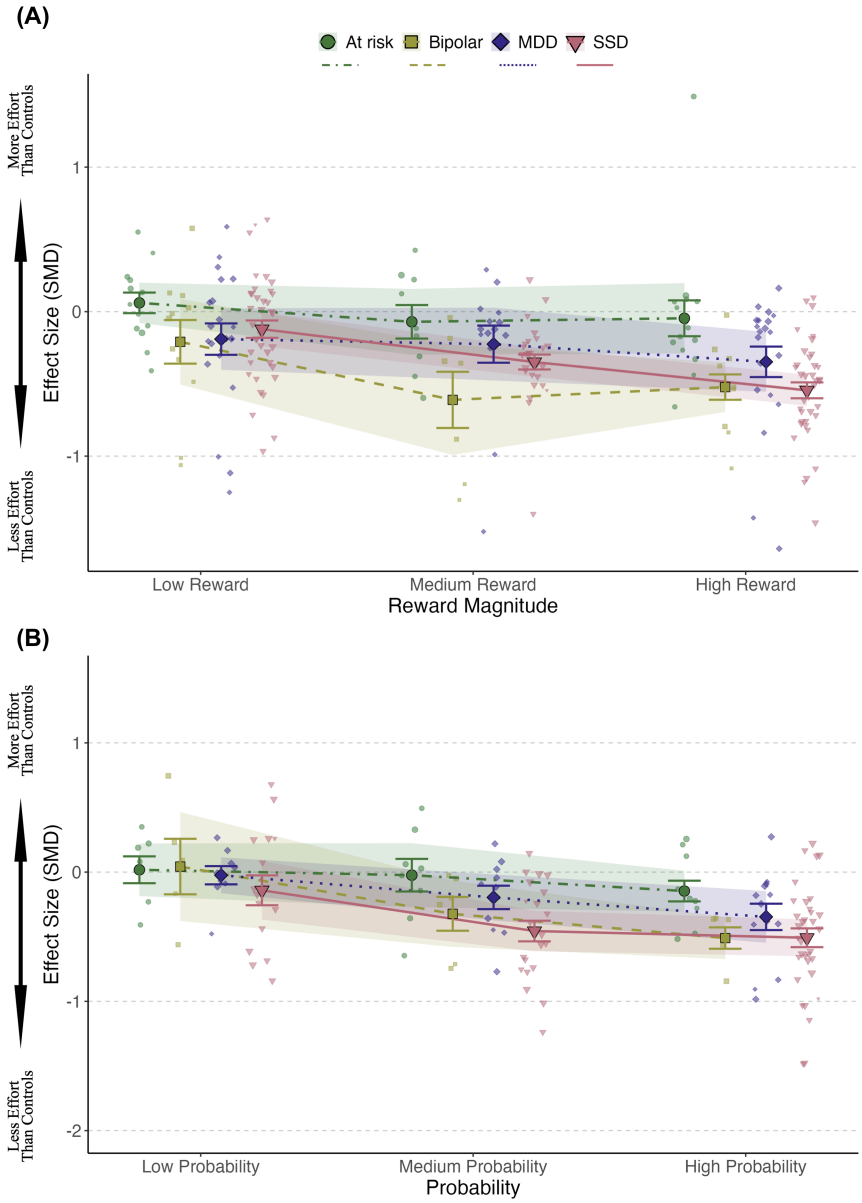
Direct Study-Level Comparisons Between Diagnostic Groups



Note. The figure depicts direct study-level comparisons in overall effort-based decision making between diagnostic groups. Negative effect sizes indicate larger effort-based decision-making impairment in schizophrenia spectrum disorders than in bipolar disorder or major depressive disorder and in major depressive disorder relative to bipolar disorder. CI = confidence interval. See the online article for the color version of this figure.

Figure 4

Effort Allocation Across Reward and Probability Conditions



Note. Panel A depicts effect sizes across trials with low-, medium-, and high-reward magnitude by diagnostic group. Panel B depicts effect sizes across trials with low-, medium-, and high-reward probability by diagnostic group. Error bars represent standard errors. Ribbons represent 95% confidence intervals. Point sizes represent study weights. Negative effect sizes indicate reduced effort allocation relative to controls. Positive effect sizes indicate increased effort allocation relative to controls. MDD = major depressive disorder; SSD = schizophrenia spectrum disorders; SMD = standardized mean differences. See the online article for the color version of this figure.

comparing each group's effect size to zero, only the SSD group exhibited a significantly lower estimated marginal mean effect size, $M_{SMD} = -0.18$, 95% CI [−0.32, −0.05], indicating a significant reduction in effort allocation compared to healthy controls. Effect sizes in BD, $M_{SMD} = -0.01$, 95% CI [−0.20, 0.18]; MDD, $M_{SMD} = -0.11$, 95% CI [−0.27, 0.05]; and AR, $M_{SMD} = 0.09$, 95% CI [−0.16, 0.33], did not differ significantly from zero, indicating that, in low reward trials, these groups did not show substantial deviations from healthy controls.

In medium-reward trials, effect size magnitudes differed significantly by diagnostic group, $F(3, 54) = 2.88$, $p = .044$. Compared to SSD, absolute effect sizes were significantly smaller in AR, $b = 0.35$, $SE = 0.14$, $p = .017$, and in MDD, $b = 0.19$, $SE = 0.10$, $p = .052$. No significant differences were found between BD and the

other groups ($ps > .07$). When comparing each group's effect size to zero, SSD, $M_{SMD} = -0.40$, 95% CI [−0.54, −0.26]; BD, $M_{SMD} = -0.35$, 95% CI [−0.57, −0.13]; and MDD, $M_{SMD} = -0.21$, 95% CI [−0.39, −0.03], all showed significantly lower estimated marginal mean effect sizes, indicating significant reductions in effort allocation compared to healthy controls. In contrast, effect sizes in AR, $M_{SMD} = -0.05$, 95% CI [−0.30, 0.20], did not differ significantly from zero, suggesting that this group did not exhibit substantial deviations from healthy controls.

In high-reward trials, effect size magnitudes again differed significantly by diagnostic group, $F(3, 80) = 7.47$, $p < .001$. Compared to SSD, absolute effect sizes were significantly smaller in AR, $b = 0.53$, $SE = 0.13$, $p < .001$, and MDD, $b = 0.26$, $SE = 0.08$, $p = .003$. AR also showed a significantly smaller effect size than BD, $b = 0.33$, $SE = 0.15$, $p = .030$, and MDD, $b = 0.274$, $SE = 0.13$, $p = .04$. BD and MDD did not differ significantly from each other ($p = .59$). When comparing each group's effect size to zero, SSD, $M_{SMD} = -0.58$, 95% CI [−0.71, −0.44]; MDD, $M_{SMD} = -0.31$, 95% CI [−0.48, −0.15]; and BD, $M_{SMD} = -0.37$, 95% CI [−0.57, −0.17], showed significantly lower estimated marginal mean effect sizes, indicating significant reductions in effort allocation compared to healthy controls. In contrast, the AR group, $M_{SMD} = -0.042$, 95% CI [−0.27, 0.19], did not significantly differ from zero, suggesting no substantial deviation from healthy controls.

Moderating Effect of Probability

Figure 4B illustrates the effect sizes across diagnostic groups as a function of probability condition. The main effect of probability condition was significant, $F(2, 142) = 38.85$, $p < .001$, indicating that absolute effect sizes (i.e., differences relative to healthy controls) increased with probability. Moreover, the interaction between probability condition (low, medium, high) and diagnostic group (SSD, MDD, BD, AR) was significant, $F(11, 133) = 8.83$, $p < .001$, suggesting that the pattern of effort allocation across probability conditions varied by diagnostic group.

Specifically, the absolute effect size significantly increased from low to medium probability trials in SSD, $k = 37$, $b = -0.37$, $t(35) = -4.56$, $p < .001$, 95% CI [−0.53, −0.20], and in BD, $k = 11$, $b = -0.44$, $t(9) = -3.02$, $p = .014$, 95% CI [−0.77, −0.11], but not in MDD, $k = 19$, $b = -0.20$, $t(17) = -1.86$, $p = .081$, 95% CI [−0.43, 0.03], or AR, $k = 15$, $b = -0.09$, $t(13) = -0.74$, $p = .473$, 95% CI [−0.34, 0.17]. From medium to high probability trials, the absolute effect size further increased significantly in SSD, $k = 55$, $b = -0.20$, $t(53) = -2.88$, $p = .006$, 95% CI [−0.33, −0.06], but not in MDD, $k = 23$, $b = -0.13$, $t(21) = -1.28$, $p = .213$, 95% CI [−0.34, 0.08]; BD, $k = 12$, $b = -0.25$, $t(10) = -1.92$, $p = .084$, 95% CI [−0.55, 0.04]; or AR, $k = 18$, $b = -0.12$, $t(16) = -1.15$, $p = .266$, 95% CI [−0.34, 0.17].

In low-probability trials, effect size magnitudes did not differ significantly across diagnostic groups, $F(3, 16.11) = 1.37$, $p = .287$. When comparing each group's effect size to zero, none of the groups showed significant deviations. Specifically, the SSD group, $M = -0.15$, 95% CI [−0.36, 0.06]; the BD group, $M = 0.16$, 95% CI [−0.18, 0.50]; the MDD group, $M = -0.04$, 95% CI [−0.32, 0.23]; and the AR group, $M = -0.004$, 95% CI [−0.37, 0.36], all exhibited effect sizes that did not significantly differ from zero, indicating no substantial deviations from healthy controls.

In medium-probability trials, a significant difference in effect size magnitudes was observed across diagnostic groups, $F(3, 20.06) = 3.62$, $p = .031$. Yet, none of the pairwise comparisons between groups were significant. When comparing each group's effect size to zero, the SSD group, $M_{SMD} = -0.45$, 95% CI [−0.61, −0.29], and the MDD group, $M_{SMD} = -0.22$, 95% CI [−0.44, −0.01], exhibited a significantly lower estimated marginal mean effect sizes, indicating significant reductions in effort allocation compared to healthy controls. Effect sizes in BD, $M_{SMD} = -0.24$, 95% CI [−0.48, 0.01], and AR, $M_{SMD} = -0.06$, 95% CI [−0.35, 0.23], did not differ significantly from zero, indicating that these groups did not show substantial deviations from healthy controls.

In high-probability trials, a significant difference in effect size magnitudes was observed across diagnostic groups, $F(3, 28.60) = 6.28$, $p = .002$. Compared to SSD, absolute effect sizes were smaller in AR, $b = 0.43$, $SE = .16$, $p = .045$, and MDD, $b = 0.33$, $SE = .09$, $p = .005$. When comparing each group's effect size to zero, the SSD group, $M_{SMD} = -0.58$, 95% CI [−0.71, −0.44]; MDD group, $M_{SMD} = -0.24$, 95% CI [−0.43, −0.06]; and BD group, $M_{SMD} = -0.39$, 95% CI [−0.63, −0.16], showed significantly lower estimated marginal mean effect sizes compared to zero, indicating significant reductions in effort allocation compared to healthy controls. The AR group did not show a significant deviation from zero, $M_{SMD} = -0.14$, 95% CI [−0.43, 0.14], indicating no substantial difference from healthy controls.

Moderator Analyses

Task Characteristics

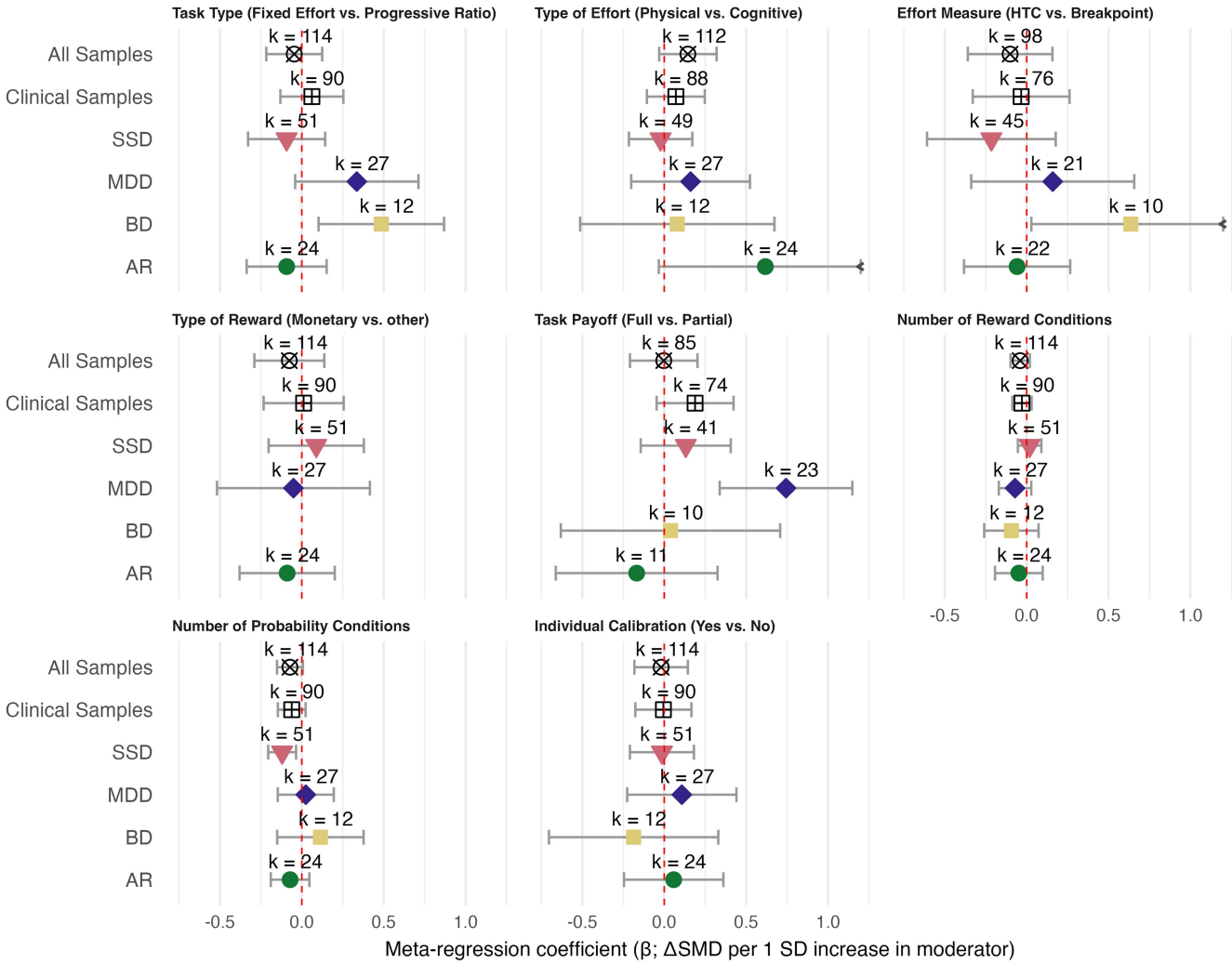
Figure 5 depicts the results from moderation analyses testing task characteristics across all task conditions. Overall effect sizes in EBDM between (sub-)clinical and control groups did not differ by type of task (dual-choice vs. effort-discounting vs. progressive ratio), $F(2,111) = 1.47$, $p = .234$. In the overall sample, none of the task characteristics significantly moderated the effect of effort-based decision making across all task conditions. In MDD, the task payoff was the only significant moderator, $b = 0.74$, $p = .002$, 95% CI [0.31, 1.17], indicating that differences to healthy controls were smaller, when participants received only partial payout ($k = 3$) compared to studies in which participants received all task rewards ($k = 20$). In BD, the moderator effects of task type, $b = 0.49$, $p = .018$, 95% CI [0.10, 0.87], and effort measure, $b = 0.64$, $p = .042$, 95% CI [0.03, 1.24], were significant, suggesting that in BD, differences to healthy controls were smaller in studies using progressive ratio tasks ($k = 9$) and assessing individual breakpoints ($k = 9$) than in studies assessing the proportion of hard task choices ($k = 1$). In SSD, the number of probability conditions was the only significant moderator, $b = -0.12$, $p = .007$, 95% CI [−0.21, −0.04], suggesting that differences in EBDM relative to healthy controls increased as a function of the number of probability conditions across studies. See Supplemental S10 for full model parameters.

Sample Characteristics

Figure 6 depicts the results from moderation analyses testing sample characteristics across all task conditions. Across all samples, a significant moderation effect was found for the percentage of

Figure 5

Task Characteristics Moderation Analyses



Note. The figure depicts meta-regression models testing task characteristics as moderators of overall differences in effort-based decision making. Negative β coefficients indicate larger effect size differences between groups per 1 standard deviation increase in the moderator. SSD = schizophrenia spectrum disorders; MDD = major depressive disorder; BD = bipolar disorder; AR = at-risk samples; SMD = standardized mean differences; HTC = hard task choices. See the online article for the color version of this figure.

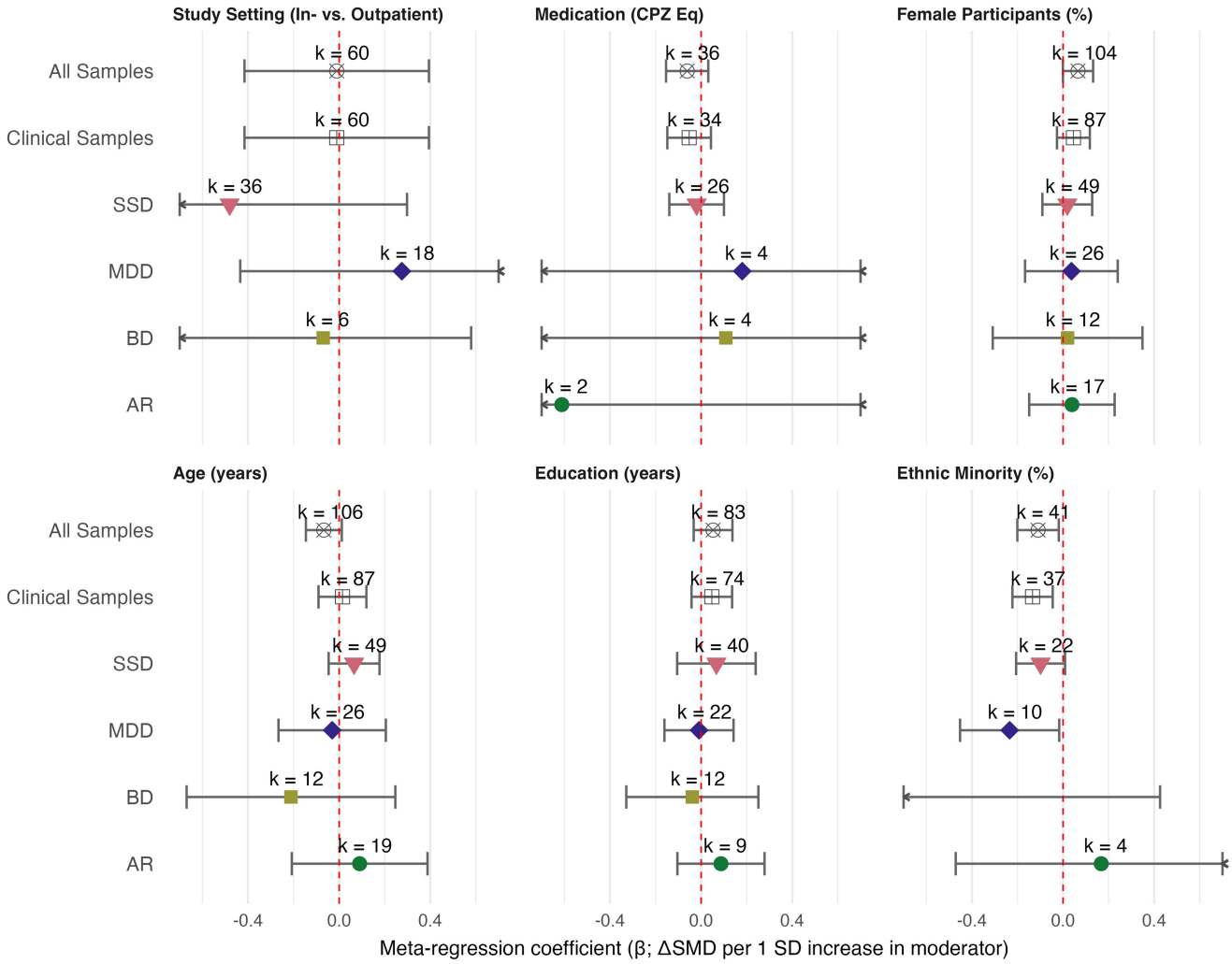
ethnic minority participants, indicating that differences between (sub-)clinical and control groups in EBDM were larger in samples with a higher percentage of ethnic minority participants, $b = -0.11$, $p = .020$, 95% CI [−0.20, −0.02]. The percentage of female participants showed a trend-level effect, suggesting a tendency for smaller clinical-control differences in EBDM in samples with higher proportions of female participants, $b = 0.07$, $p = .055$, 95% CI [0.00, 0.13]. In the clinical samples, a significant moderation effect was found for the percentage of ethnic minority participants, indicating that differences in EBDM within clinical groups were larger in samples with higher percentage of ethnic minority participants, $b = -0.13$, $p < .01$, 95% CI [−0.22, −0.04]. A trend-level moderation effect for the percentage of ethnic minority participants was observed in both SSD and MDD samples, suggesting that differences in EBDM tended to be larger in samples with a higher percentage of ethnic minority participants, SSD: $b = -0.10$, $p = .070$, 95% CI [−0.21, 0.02], and MDD: $b = -0.23$, $p = .052$ 95% CI [−0.46, 0.00]. See Supplemental S11 for full model parameters.

Clinical Characteristics

Figure 7 depicts the results from moderation analyses testing clinical characteristics across all task conditions. Across all samples, significant moderation effects were found for severity of positive symptoms, $b = -0.14$, $p = .007$, 95% CI [−0.24, −0.04], and of negative symptoms, $b = -0.10$, $p = .029$, 95% CI [−0.18, −0.01], indicating that with increasing severity of both positive and negative symptoms differences between clinical and control groups in EBDM increased significantly across studies. In the clinical samples (excluding AR), significant moderation effects were found for severity of positive symptoms, $b = -0.15$, $p = .007$, 95% CI [−0.25, −0.04], and at trend level, negative symptoms, $b = -0.09$, $p = .058$,

Figure 6

Sample Characteristics Moderation Analyses



Note. The figure depicts meta-regression models testing sample characteristics as moderators of overall differences in effort-based decision making. All numerical moderators were *z*-standardized. Negative β coefficients indicate larger effect sizes between groups per 1 standard deviation increase in the moderator. SSD = schizophrenia spectrum disorders; MDD = major depressive disorder; BD = bipolar disorder; AR = at-risk samples; CPZ Eq = chlorpromazine equivalent in milligrams; SMD = standardized mean differences. See the online article for the color version of this figure.

95% CI [−0.18, 0.00], indicating that with increasing severity of positive and negative symptoms, differences between clinical and control groups in EBDM increased significantly across studies. In SSD, a significant moderation effect was found for severity of positive symptoms, $b = -0.12$, $p = .019$, 95% CI [−0.22, −0.02], and at trend level, for negative symptoms, $b = -0.08$, $p = .086$, 95% CI [−0.18, 0.01]. In BD, a significant moderation effect was found for severity of anhedonia, $b = 0.53$, $p = .05$, 95% CI [0.02, 1.03], and severity of depressive symptoms, $b = -0.21$, $p = .053$, 95% CI [−0.42, 0.00], indicating that with increasing severity of depressive symptoms, differences between clinical and control groups in EBDM performance increased significantly but decreased as a function of increasing anhedonia scores. In the AR samples, no significant moderation effects were found for any clinical characteristic. See Supplemental S12 for full model parameters.

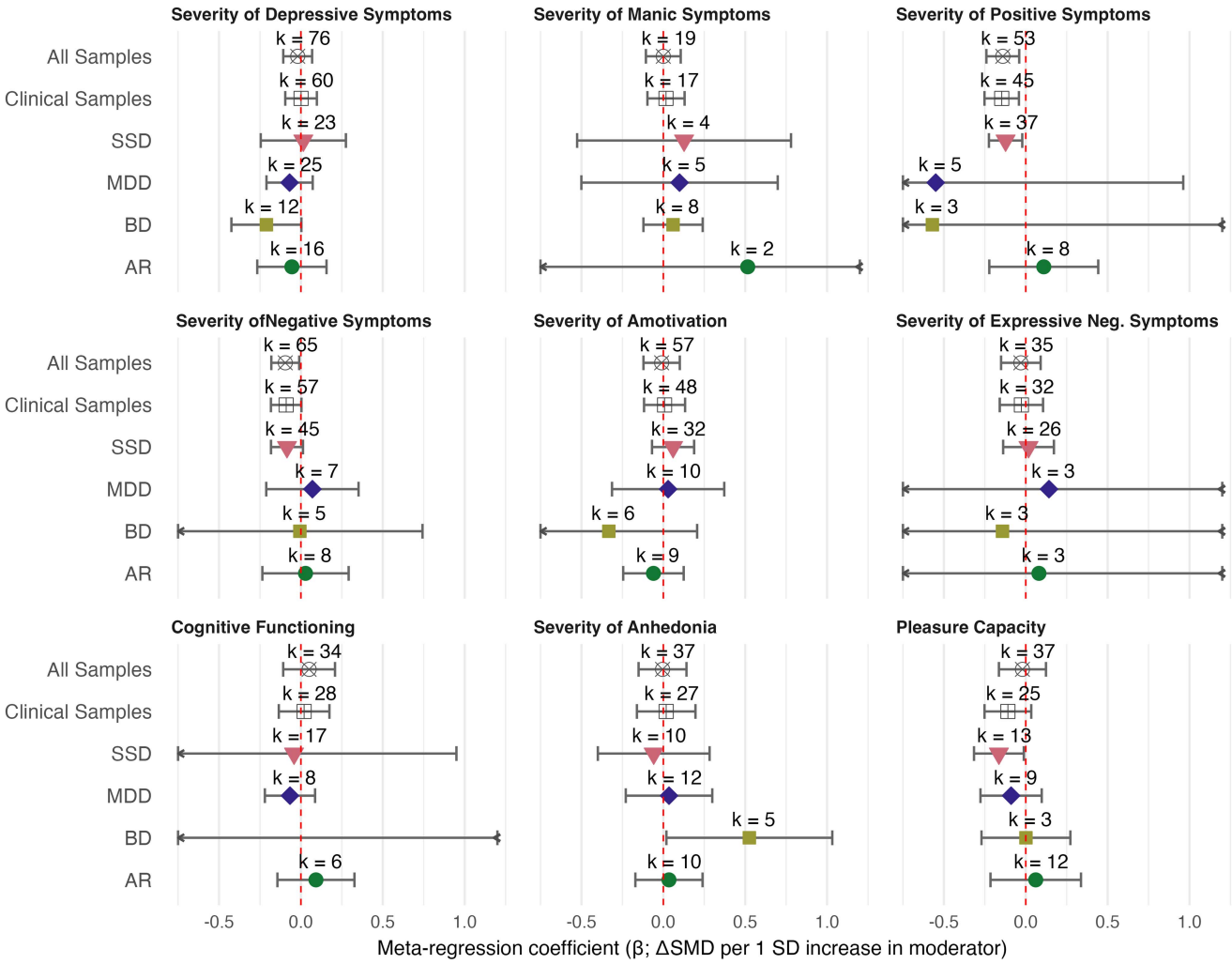
Additional Exploratory Analyses

Additional exploratory meta-regression models were calculated to test whether clinical characteristics moderated the effect on EBDM in high-reward or high-probability trials, where the largest differences between clinical groups and controls were observed. These analyses revealed a similar pattern of findings in the high-reward trials as in the moderation analyses across all task conditions, but no significant moderation effects in the high-probability trials, likely due to the substantially smaller number of available studies reporting data from high-probability trials (see Supplemental S13–S14).

Following previous findings (Horan et al., 2015; Reddy et al., 2015), we examined whether increases in effort allocation from low to high reward/probability conditions varied by levels of cognitive functioning, by modeling the Condition (low vs. high) × Cognitive

Figure 7

Clinical Characteristics Moderation Analyses



Note. The figure depicts meta-regression models testing clinical characteristics as moderators of overall differences in effort-based decision making. All numerical moderators were *z*-standardized. Negative β coefficients indicate larger effect size differences between groups per 1 standard deviation increase in the moderator. SSD = schizophrenia spectrum disorders; MDD = major depressive disorder; BD = bipolar disorder; AR = at-risk samples; SMD = standardized mean differences. See the online article for the color version of this figure.

Functioning interaction predicting group differences in EBDM. These analyses revealed a significant Reward × Cognitive Functioning interaction, $k = 16$, $b = -0.02$, $p = .041$, 95% CI [−0.04, −0.00], and a significant Probability × Cognitive Functioning interaction predicting group differences in EBDM in SSD studies, $k = 12$, $b = -0.03$, $p = .041$, 95% CI [−0.05, −0.00], indicating that group differences in effort allocation from low to high reward and probability conditions were amplified as a function of cognitive impairment. All other models revealed nonsignificant interactions or could not be computed due to the low number of studies available for these analyses (see Supplemental S15).

A third set of exploratory meta-regression models was calculated to test the effect of cognitive functioning domains separately. Domain-specific data were available for working memory, processing speed, verbal memory, attention, and executive functioning. In these models, processing speed significantly moderated the effect in EBDM across all samples, $b = -0.31$, $p = .006$, CI [−0.52, −0.12], and in clinical samples (excluding AR), $b = -0.32$, $p = .014$, 95% CI [−0.56, −0.08]. A trend-level effect was observed in SSD samples, $b = -0.27$, $p = .071$, 95% CI [−0.57, 0.03], indicating that slower processing speed was associated with larger differences in EBDM between clinical and control groups. No significant effects were found for working memory, verbal memory, attention, or executive functioning across any sample (see Supplemental S16).

Risk of Bias Assessment

Overall, risk of bias was rated as moderate ($M = 1.97$, $SD = 0.30$). Sixty-two studies received a moderate risk-of-bias rating (including J. Wang et al., 2018), while four and two studies received low and

high risk-of-bias ratings, respectively. The most common concerns were based on potential bias due to samples being not representative of the target population (e.g., SSD studies excluding participants with current, or lifetime substance use disorders, or a narrow age range of included participants) and studies that did not report psychometric properties of EBDM tasks. Nine studies were classified as high risk in the "study design" domain due to the absence of clearly formulated a priori hypotheses. Statistical concerns included missing adjustment of significance thresholds for multiple testing and low statistical power due to small sample sizes or missing a priori sample size calculations. Risk of bias did not moderate the overall effect in EBDM between (sub-)clinical and control groups, indicating robustness of the evidence, $F(2,111) = 1.02$, $p = .365$.

Narrative Synthesis of Computational Modeling Studies

Study Selection and Sample Characteristics

As Table 1 shows, nine studies were identified that investigated EBDM using computational modeling across (sub-)clinical populations compared to controls, including participants diagnosed with MDD, SSD, BD, and individuals with elevated subclinical psychotic symptoms. Specifically, five studies examined samples with SSD, with a total of 273 individuals with SSD (Cooper et al., 2019; Culbreth, Chib, et al., 2024; Saleh et al., 2023; L. L. Wang et al., 2024; Whitton et al., 2024). Five studies focused on MDD, including remitted and current episodes as well as MDD with psychotic features, with a combined sample size of 276 individuals (Berwian et al., 2020; Valton et al., 2025; Vinckier et al., 2022; L. L. Wang et al., 2024; Whitton et al., 2024). Two studies investigated a total of 105 individuals with BD and a current depressive episode (Vinckier et al., 2022) or with BD with psychotic features (Whitton et al., 2024). Another study explored EBDM in subclinical psychotic symptoms, involving 19 participants with high psychotic experiences and 20 with low psychotic experiences (Terenzi et al., 2024). Direct within-study transdiagnostic comparisons across MDD, BD, and SSD were available from three of these studies (Vinckier et al., 2022; L. L. Wang et al., 2024; Whitton et al., 2024).

Common Parameters of Putative Mechanisms Underlying Effort-Based Decision Making

Across these studies, effort sensitivity, the degree to which an individual reacts to the cost of effort, and reward sensitivity, the degree to which an individual values incentives or rewards, emerged as the most frequently assessed parameters. Further parameters included decision thresholds (i.e., the amount of evidence required before committing to a decision), drift rate (i.e., the speed of evidence accumulation during decision making), acceptance bias (i.e., a general tendency to accept or reject tasks, independent of reward or effort), and nondecision time (i.e., time unrelated to the decision process, such as motor response time).

Narrative Synthesis of Putatively Underlying Mechanisms

Computational Phenotyping of Effort-Based Decision Making Across Disorders. Studies using a computational phenotyping approach have identified distinct clusters of effort allocation patterns across and within diagnostic categories. These clusters are thought to represent distinct EBDM phenotypes and to reflect etiological pathways to motivational impairment.

Whitton et al. (2024) aimed to categorize effort allocation strategies into computational phenotypes across psychotic disorders, including schizophrenia, schizoaffective disorder, bipolar disorder with psychotic features, major depressive disorder with psychotic features, psychotic disorder not otherwise specified, and schizophreniform disorder. The authors prespecified three subtypes: (a) the full subjective value phenotype, which integrates reward value and probability and is associated with better cognitive functioning and lower symptom severity; (b) the reward-only phenotype, which considers only reward value, disregarding probability; and (c) the bias phenotype, which exhibits unsystematic effort allocation and is linked to more severe positive and negative symptoms, lower community functioning, and cognitive impairments. Their findings revealed that the full subjective value phenotype was more common in individuals with psychotic bipolar disorder (58.1%) and healthy controls but less frequent in schizophrenia and schizoaffective disorder (21.6%). In contrast, the bias phenotype was more prevalent in schizophrenia and schizoaffective disorder (67.0%) compared to psychotic bipolar disorder (34.9%). Within the bias phenotype, Whitton et al. identified two subgroups: (a) effort-averse responders, who avoided high-effort tasks, and (b) unsystematic responders, who displayed inconsistent effort allocation patterns. The study found that while unsystematic effort allocation in the bias phenotype was associated with working memory impairment, systematic effort allocation in the full subjective value phenotype was associated to younger age, a bipolar disorder diagnosis, less severe negative symptoms, and better cognitive functioning.

Consistent with these findings, Cooper et al. (2019) reanalyzed a large, combined data set and replicated two clusters of EBDM patterns in SSD: (a) biased or unsystematic effort allocation which was most prevalent in SSD (71%) and (b) full subjective value effort allocation which was found in only 29% of participants with SSD. In this study, nonsystematic effort allocation across trials was also associated with cognitive impairment. In those who systematically allocated effort on a trial-by-trial basis (i.e., those categorized into the full subjective value phenotype), increased effort sensitivity in the EBDM task was associated with higher amotivational psychopathology.

In sum, these studies converge on the existence of at least two phenotypes of EBDM that have been interpreted as representing distinct pathways to EBDM deficits in people with SSD. The more prevalent pattern is unsystematic or biased effort allocation, which is most strongly associated with cognitive impairment and may lead to difficulties integrating reward, probability, and effort information across trials. At the same time, a substantial subgroup of individuals with SSD still shows more systematic effort allocation, associated with better cognitive functioning and lower symptom severity relative to people with SSD in the unsystematic or bias phenotype. Still, in this phenotype, interindividual differences in effort sensitivity are associated with levels of amotivation, potentially indicating a second pathway to EBDM alterations in SSD that is marked by elevated effort sensitivity and subjective experiences of amotivation.

Effort and Reward Sensitivity in SSD. Culbreth, Chib, et al. (2024) found that, compared to healthy controls, individuals with SSD exhibit heightened sensitivity to effort, particularly during high-effort trials, while showing no significant differences in reward sensitivity. Furthermore, compared to controls, participants with SSD overestimated effort costs in subjective ratings of effort, even in

Table 1
Overview of Computational Modeling Studies Included for the Narrative Synthesis

Study	Participant	Study aim	EBDM task	Computational modeling approach and parameters assessed	Key finding
Berwian et al. (2020)	123 remitted MDD patients, 66 healthy controls	Investigate effort-reward decision making in remitted MDD and its link to relapse risk after antidepressant discontinuation	BET	Drift-diffusion model with hierarchical Bayesian fitting. Parameters: reward sensitivity, effort sensitivity, decision thresholds, and "pswitch"	Remitted MDD patients showed slower decision times, lower willingness to exert effort, and deviations in decision making (e.g., "pswitch" parameter). Long decision times predicted relapse risk
Cooper et al. (2019)	150 individuals with schizophrenia or schizoaffective disorder, 105 healthy controls	Examine EBDM impairments in schizophrenia, focusing on motivational and cognitive functioning	EEfRT	Three computational models: full subjective value model, reward-only subjective value model, and bias model. Parameters: effort discounting, sensitivity to reward probability, sensitivity of choices to subjective values, bias toward low effort option. Model fit evaluated using Bayesian information criterion (BIC)	SSD were found to exhibit nonsystematic effort allocation based on reward/probability, with ~50% best fit by the "bias model." Participants fit by the bias model showed greater cognitive impairment. Within participants fit by the full subjective value model, greater effort aversion was associated with higher levels of amotivation
Culbreth, Chib, et al. (2024)	30 schizophrenia patients, 30 healthy controls	Explore whether reduced EBDM in schizophrenia is driven by reward or effort sensitivity	AGT and RAT	Two-parameter model for effort sensitivity during Risk Aversion Task. Parameters: effort sensitivity, variability of choice behavior	Schizophrenia patients exhibited increased effort sensitivity, particularly during high-effort trials, with no significant differences in the variability of choice behavior
Saleh et al. (2023)	40 treatment-resistant schizophrenia patients, 40 healthy controls	Investigate EBDM deficits and their links to cognitive impairment and negative symptoms	AGT	Bayesian hierarchical drift-diffusion model. Parameters: decision threshold, drift rate (evidence accumulation speed), bias (starting point for evidence accumulation), and nondecision time (time unrelated to decision making)	Patients were less sensitive to increasing rewards and less averse to increasing effort. Drift-diffusion modelling showed higher decision thresholds and non-decision time, lower baseline drift rates, and reduced modulation of drift rate by both reward and effort. Reward sensitivity was associated with cognitive impairment, blunted effort aversion was associated to expressive negative symptoms
Terenzi et al. (2024)	39 participants with high (n = 19) and low (n = 20) psychotic symptoms	Examine effort discounting and the role of dopamine/norepinephrine in subclinical psychotic symptoms	EDT	Reinforcement meta learner model. Parameters: dopamine and norepinephrine modulation, effort sensitivity, and reward valuation	High psychotic symptom group showed reduced willingness to exert effort under high-cost conditions, linked to dopamine dysregulation
Valton et al. (2025)	57 healthy controls, 36 relatives of MDD patients, 46 remitted MDD, 41 current MDD	Examine whether EBDM impairments in MDD are driven by altered reward sensitivity, effort sensitivity, or acceptance bias	AGT	Bayesian hierarchical model with 70 competing models. Parameters: acceptance bias, linear reward sensitivity, linear effort sensitivity, quadratic effort sensitivity (nonlinear perception of effort costs)	MDD groups (current/remitted) accepted fewer offers due to reduced acceptance bias rather than altered reward/effort sensitivity. Reduced acceptance bias in remitted MDD suggests a stable trait or "scar" of depression
Vinckier et al. (2022)	15 BD patients, 20 MDD patients, 35 healthy controls	Investigate effort sensitivity, reward sensitivity, and punishment sensitivity across seven tasks	Seven tasks	Single computational model across tasks with task-specific parameters. Parameters: effort sensitivity, reward sensitivity, punishment sensitivity, and subjective values (modeled using a softmax function)	Elevated effort sensitivity was the primary driver of motivational deficits in both MDD and BD. No significant differences in reward or punishment sensitivity were observed across clinical groups. Elevated effort sensitivity correlated with higher apathy, but not with depressive symptoms or anhedonia

(*table continues*)

Table 1 (*continued*)

Study	Participant	Study aim	EBDM task	Computational modeling approach and parameters assessed	Key finding
L. L. Wang et al. (2024)	50 schizophrenia patients, 46 MDD patients, 56 healthy controls	Study range adaptation for reward processing and its relationship to amotivation and anhedonia	E-PET	Range adaptation model. Parameters: observed value adaptation (response slope to value ranges) and expected value adaptation (interaction between previous and current EVs)	Schizophrenia patients showed reduced expected value (EV) adaptation and excessive outcome value (OV) adaptation. MDD patients exhibited reduced OV adaptation but intact EV adaptation, highlighting distinct reward processing deficits in each disorder
Whitton et al. (2024)	242 participants: 190 patients with psychotic disorders (90 with psychotic bipolar disorder, 49 with schizoaffective disorder, 42 with schizophrenia, five with major depressive disorder with psychotic features, three with psychotic disorder not otherwise specified, one with schizophreniform disorder) and 52 healthy controls	To examine whether shared or divergent mechanisms underpin impairments in effort-cost decision making across psychotic disorders and identify clinical and cognitive characteristics of effort allocation strategies	EEfRT	Three computational models applied to EEfRT data: (1) full subjective value model: integrates reward value, reward probability, and effort cost; (2) reward-only model: considers reward value and effort cost but disregards probability; (3) bias model: assumes no use of reward value or probability, focusing on bias toward low or unsystematic effort allocation. Parameters assessed: effort discounting, probability sensitivity, sensitivity of choices to subjective values, and bias. Models fit using maximum likelihood estimation, with comparisons made using BIC	1. Computational phenotypes differed across psychotic disorders: *Bias phenotype* (no systematic use of reward value or probability) was more common in schizophrenia/schizoaffective disorder (67%) than psychotic bipolar disorder (34.9%). *Full subjective value phenotype* was more common in psychotic bipolar disorder (58.1%) than schizophrenia/schizoaffective disorder (21.6%) 2. *Bias phenotype* associated with more severe positive and negative symptoms, greater cognitive impairment (working memory, verbal memory, verbal fluency, processing speed), and poorer community functioning 3. Working memory impairments were linked to unsystematic effort allocation in the *bias phenotype*, suggesting cognitive deficits hinder integration of reward value and probability 4. Effort allocation strategies differed based on diagnosis: psychotic bipolar disorder showed dampened sensitivity to reward value/probability, while schizophrenia/schizoaffective disorder showed issues integrating these factors

Note. AGT = Apple Gathering Task; BET = Balloon Effort Task; EDT = Effort Discounting Task; EEfRT = Effort Expenditure for Reward Task; E-PET = Effort-Based Pleasure Experience Task; RAT = Risk Aversion Task; MDD = major depressive disorder; EBDM = Effort-based decision making; SSD = schizophrenia spectrum disorders; BD = bipolar disorder.

low-effort tasks. Culbreth et al. concluded that this increased effort sensitivity represents a key driver of reduced willingness to exert effort.

Saleh et al. (2023) examined the role of cognitive parameters in relation to both reward and effort sensitivity in participants with "treatment-resistant" SSD compared to healthy controls. Using Bayesian hierarchical drift-diffusion modeling, they found that treatment-resistant SSD participants required higher decision thresholds, exhibited slower evidence accumulation, and showed reduced responsiveness to reward and effort information, indicating diminished reward sensitivity and blunted effort aversion. In their subgroup analyses, they found distinct associations: Cognitive impairment was linked to diminished reward sensitivity, while higher expressive negative symptoms were associated with reduced effort sensitivity. Although the negative association between effort sensitivity and expressive negative symptoms diverges from the findings in other studies (e.g., Cooper et al., 2019; Culbreth, Chib, et al., 2024), the findings by Saleh et al. further substantiate the evidence indicating that cognitive impairment and negative symptoms might represent two distinct pathways to maladaptive effort allocation.

Taken together, these studies provide evidence that altered effort sensitivity is a consistent feature of effort allocation patterns in SSD. However, whether these alterations present as heightened or attenuated sensitivity to effort in SSD may vary across specific samples and subgroups. Findings also replicate the association between cognitive impairment and reduced reward sensitivity, while negative symptoms are more closely related to altered effort sensitivity. Across studies, the dissociation between effort- and reward-related processes emerges as a robust pattern, with altered effort sensitivity and cognitive impairment putatively representing the most common mechanisms behind altered EBDM in SSD.

Effort and Reward Sensitivity in Major Depressive Disorder. Findings on effort and reward sensitivity in MDD remain inconclusive, with some studies suggesting that MDD is characterized more by a generalized avoidance to engage in effortful tasks rather than specific alterations in reward or effort sensitivity.

Valton et al. (2025) reported that individuals with both current and remitted MDD exhibited lower acceptance rates for effortful tasks, driven by a reduced acceptance bias rather than differences in reward or effort sensitivity. The authors interpreted these lower acceptance rates as generalized unwillingness to exert effort and as a traitlike characteristic, persisting even in remitted MDD. However, the fact that healthy relatives of MDD patients did not display this pattern raises questions about whether this bias serves as a specific risk factor for MDD.

In contrast, Vinckier et al. (2022) found increased effort sensitivity in MDD in relation to amotivational psychopathology, similar to the association patterns found in SSD (Cooper et al., 2019). Across seven tasks, patients exhibited heightened effort sensitivity, associated with the avoidance of effortful actions despite the availability of significant rewards. Consistent with Valton et al. (2025), this study found no differences in reward sensitivity.

Berwian et al. (2020) observed similar effort-related alterations using drift-diffusion modeling. Typically, in standard drift-diffusion models, more deterministic decisions, that is, those strongly favoring one option, are associated with faster reaction times. The authors found that when MDD patients evaluated high-effort, low-reward options, they made highly deterministic choices but took longer to decide. To account for this deviation, Berwian et al. introduced the "pswitch" parameter, which the authors suggest, reflects a predisposition in MDD patients to select low-effort options under low-reward conditions, possibly indicating an altered "change-of-mind" mechanism in decision making. Notably, longer decision times were predictive of relapse following antidepressant discontinuation with up to 71% accuracy. Compared to patients who remained in remission, those who relapsed within 6 months displayed significantly higher decision boundaries which could be interpreted as requiring more evidence before committing to a choice.

Overall, these studies converge in attributing altered EBDM patterns in individuals with MDD to increased effort sensitivity rather than altered reward sensitivity. Both Vinckier et al. (2022) and Berwian et al. (2020) reported heightened effort sensitivity, while Valton et al. (2025) identified a reduced acceptance bias that similarly manifested as reduced effort expenditure across trials, even in remitted MDD. By contrast, there was no consistent alteration in parameters of reward sensitivity across studies. Together, these results suggest that increased effort sensitivity currently represents the most robust and replicated finding of a putative mechanism of altered effort allocation in MDD.

Effort and Reward Sensitivity in Bipolar Disorder. Evidence on the computational mechanisms underlying EBDM in BD remains limited and provided mixed findings.

Whitton et al. (2024) found that "clinically stable" individuals with psychotic BD were more likely to exhibit the full subjective value phenotype that people with SSD. The full subjective value phenotype is characterized by a relatively intact integration of reward value and reward probability and was associated with better cognitive functioning. The study by Whitton et al. also found tentative evidence that, in BD, the full subjective value phenotype may be associated with less severe amotivation compared to the bias phenotype. However, this association was not robust against correcting for multiple testing.

Vinckier et al. (2022) examined individuals with BD in a current depressive episode. In this sample, they found elevated effort sensitivity and similar reward sensitivity when compared to healthy controls. Increased effort sensitivity was associated with higher amotivation, but not with depression severity or anhedonia. This pattern resembles the association between increased effort sensitivity and higher amotivation found in MDD in this.

In sum, findings across studies investigating computational mechanisms of EBDM in BD are mixed but might indicate that effort allocation mechanisms depend on the current clinical state of BD. While effort allocation patterns in psychotic, but clinically stable BD states may be characterized by relatively intact integration of reward value and probability information, depressive states in BD seem to be reflected by elevated effort sensitivity that is particularly associated with amotivation. Nevertheless, given the small sample size in Vinckier et al. and the episodic course of BD, the mechanisms underlying altered effort-allocation patterns in BD remain insufficiently understood and require more systematic investigation in larger, well-characterized samples.

Other Mechanisms in Effort-Based Decision Making. While the primary focus of computational modeling studies was on effort and reward sensitivity, some recent studies have proposed additional processes that may be linked to EBDM. For instance, L. L. Wang et al. (2024) adopted the concept of range adaptation. Range adaptation describes the brain's ability to dynamically adjust neural responses to the context of the available range of reward (Kaliuzhna et al., 2024).

Specifically, it is assumed that dopaminergic neurons adapt their sensitivity to reward to ensure precise reward value representation within a limited coding range. In their study, Wang et al. sought to infer range adaptation by applying computational modeling to behavioral outcomes of EBDM in SSD and MDD compared to HC. Indeed, their findings were indicative of altered range adaptation in the clinical groups with distinct patterns across SSD and MDD, which was correlated with amotivational psychopathology in both groups. Nonetheless, their interpretation of range adaptation may be constrained by the challenge of translating neural adaptation principles to behavioral outcomes.

In a similar vein, Terenzi et al. (2024) sought to infer the role of dopamine in effort-discounting behaviors in individuals with elevated psychotic symptoms by fitting behavioral EBDM outcomes to a parameter they interpret as a computational proxy of dopamine functioning. Their findings suggest altered effort-discounting behaviors in individuals with elevated psychotic symptoms, particularly under high-cost conditions. Although Terenzi et al. attributed these results to dopamine dysregulation, highlighting the proposed neurochemical mechanism, it is important to acknowledge that their conclusions are based on indirect measures (i.e., behavior) and computational modeling, which inherently limit the ability to draw direct conclusions about dopamine function.

In sum, these studies showcase promising directions for understanding neurocomputational mechanisms underlying maladaptive EBDM, namely, altered range adaptation and dopaminergic function. However, the evidence remains preliminary, as findings are based on indirect behavioral proxies of neural processes and computational modeling assumptions, which limit the strength of mechanistic conclusions.

Discussion

This meta-analysis investigated effort-based decision making (EBDM) across populations with SSD, MDD, BD, and AR. Findings revealed small-to-moderate impairments in EBDM across clinical groups, with SSD and BD showing the most pronounced deficits, followed by MDD, while AR individuals exhibited no significant reductions. Mechanistic insights from computational modeling identified distinct but overlapping pathways, including heightened effort sensitivity, reduced reward sensitivity, and cognitive impairment.

Cross-Diagnostic Alterations in Effort-Based Decision Making

Reductions in EBDM were most notable in SSD ($g = -0.40$) and BD ($g = -0.39$), followed by smaller reductions in MDD ($g = -0.28$) and nonsignificant differences from healthy controls in AR ($g = -0.01$). Direct comparisons revealed that SSD exerted significantly less effort than MDD ($g = -0.23$) and BD ($g = -0.27$), though BD and MDD did not differ significantly ($g = -0.06$). Our findings are consistent with the only previous meta-analysis on EBDM in SSD populations and substantiate the existing evidence by incorporating a significantly larger number of study-level effect sizes for SSD ($k = 51$ vs. $k = 20$; Blouzard et al., 2023). Additionally, the present meta-analysis is the first to complement previous systematic reviews that did not synthesize effect sizes for EBDM in MDD or BD (e.g., Horne et al., 2021). Overall, the findings of the current meta-analysis indicate that, although the severity may vary, all examined clinical populations show reduced EBDM with altered effort allocation patterns, reflecting a shared phenomenology of motivational deficits across SSD, MDD, and BD. The nonsignificant effect in AR might suggest that EBDM deficits emerge as psychopathology progresses to clinically diagnosable levels and is probably not a general characteristic of individuals at risk. At the same time, not all individuals in AR samples will progress to develop SSD, BD, or MDD. Consequently, the absence of a significant effect does not necessarily rule out a predictive role for EBDM in those who do convert. Future research with longitudinal designs is needed to determine whether EBDM deficits can identify individuals at heightened risk of transitioning to a clinical disorder that is associated with amotivational psychopathology.

The nonsignificant difference between BD and MDD ($g = -0.063$) suggests potential overlap in EBDM patterns, possibly reflecting shared underlying mechanisms. However, given that most BD studies did not specify the phase of bipolar disorder, it remains unclear whether EBDM patterns differ between depressive, euthymic, and (hypo-)manic states. As many BD participants may have been in a depressive or euthymic episode at the time of assessment, potential differences between MDD and BD could have been obscured. One study explicitly recruited participants experiencing acute (hypo-)mania alongside groups with schizophrenia, unipolar MDD, and currently depressed BD (Yang et al., 2021). While the overall effect size of the (hypo-)mania group from Yang et al.'s study did not differ notably from the other BD effect sizes in our meta-analysis, Yang et al. identified distinct EBDM patterns among individuals with unipolar depression, bipolar depression, and bipolar mania. Specifically, while participants with unipolar and bipolar depression exhibited similar EBDM patterns, those with bipolar mania showed a dysregulated effort allocation pattern which was marked by excessive effort expenditure in low reward/probability trials but diminished effort in high reward/probability trials. This effort allocation pattern may seem paradoxical because increased reward sensitivity does not translate to effort expenditure under high reward/probability conditions. Thus, further factors such as deficits in executive functioning may contribute to altered effort allocation in individuals with acute (hypo-)mania. More specifically, reduced working memory capacity and difficulties maintaining goal representations may impair the integration of anticipated rewards with associated effort costs. Therefore, the affected individuals may show impulsive effort expenditure in uncertain, low-reward contexts while failing to adjust their effort in response to increasing reward magnitude and probability. Yet, this interpretation remains speculative and requires further empirical validation. Future studies should account for mood states to clarify differentiating phenomenology and mechanisms across diagnostic categories and to elucidate the role of dopaminergic processes in maladaptive effort allocation.

Effort Allocation Across Increasing Levels of Reward and Probability

Clinical groups showed significantly and similarly reduced effort allocation, particularly in high-reward and high-probability conditions. Our meta-regression models, testing effort allocation across varying reward and probability conditions, revealed that while AR samples did not show significant deviations from healthy controls, individuals with SSD, MDD, and BD exerted significantly less effort than healthy controls, especially when the potential rewards

were high and certain. These findings align with and further substantiate the previously identified pattern of inefficient effort allocation in SSD (Blouzard et al., 2023; Gold et al., 2015) and extend the existing literature by offering a meta-analytic comparison across SSD, MDD, BD, and AR. Again, these findings indicate a largely shared phenomenology of EBDM deficits across diagnostic categories (with the exception of AR) that are marked by altered effort allocation with increasing reward magnitude and probability, which could suggest that individuals with SSD, MDD, and BD face difficulties to accurately compute and represent expected value, particularly as reward magnitude and probability increase. While our effort allocation analyses have identified the behavioral patterns underlying reduced effort exertion in EBDM tasks, the precise mechanisms driving this inefficient effort allocation pattern remain unclear from these analyses.

Potential Mechanisms Underlying Altered Effort Allocation

Our review and narrative synthesis of computational modeling studies investigating effort allocation suggests that EBDM deficits across SSD, MDD, and BD may stem from both shared and distinct yet interrelated mechanisms. The mechanisms identified in these studies indeed largely align with those proposed in the literature as potential drivers of inefficient effort allocation and amotivational psychopathology (Gold et al., 2015; Strauss & Cohen, 2017).

Sensitivity to Effort and Reward

Increased sensitivity to effort (i.e., how individuals perceive or react to effort costs) emerged as a unifying mechanism linked to diminished effort allocation across disorders, except for the participants with "treatment resistant schizophrenia" in the study be Saleh et al. (2023). Diminished reward sensitivity (i.e., how individuals perceive or react to incentives), on the other hand, presented a more nuanced picture. While individuals with SSD demonstrated mixed patterns with some showing intact reward processing and others exhibited deficits associated with cognitive impairments and severe negative symptoms, reward sensitivity deficits were less evident in MDD and BD. In MDD and BD, EBDM alterations were more strongly linked to increased effort sensitivity than to reward processing deficits. Together, these differences underscore disorder-specific mechanisms contributing to inefficient effort allocation and exemplify equifinality. The concept of equifinality assumes that amotivational psychopathology has a similar phenomenology across diagnostic categories but stems from distinct etiological mechanisms (for a discussion of transdiagnosticity vs. equifinality in reward processing deficits, see Nusslock et al., 2025).

The equifinality perspective is in line with the proposition of two distinct pathways, a hedonic and a cognitive mechanism, underlying amotivational psychopathology (Strauss & Cohen, 2017). The hedonic pathway is thought to stem from neural dysfunctions in reward processing, which directly affect an individual's ability to anticipate and to experience pleasure. The cognitive pathway involves disruptions in higher order cognitive processes, including working memory, cognitive control, and decision making, which may impair the ability to integrate effort and reward information, accumulate evidence over time, and may result in unsystematic effort allocation.

Recent cluster analyses provide further insight into reward processing deficits in SSD (Luther et al., 2023; Spilka et al., 2025). Both studies suggest that a subset of individuals with SSD exhibits global reward processing deficits linked to amotivational psychopathology. Within this global deficit cluster, impairments in effort-cost computation, reinforcement learning, and value representation were most strongly associated with reduced goal-directed behavior. In contrast, deficits restricted to hedonic reactivity showed no link to amotivational psychopathology, challenging earlier models that attribute such psychopathology solely to impaired hedonic capacity. Thus, while some individuals with SSD may maintain intact reward responsiveness, others, particularly those with more severe cognitive and amotivational symptoms, display significant deficits in reward processing that contribute to altered EBDM and reduced goal-directed behavior in daily life.

Diminished reward sensitivity is likely represented by inaccurate reward value representations and inefficient adaptations of these mental representations to the increasing reward and probability information (Gold et al., 2008; Waltz & Gold, 2016; L. L. Wang et al., 2024). This notion is bolstered by evidence suggesting that reward salience can modulate effort exertion in EBDM tasks. A study using the Balloon Effort Task found that when reward salience was experimentally enhanced, effort exertion significantly increased in a healthy community sample (Renz et al., 2022). At the subjective level, inaccurate reward value representations may manifest as less vivid mental representations of rewarding experiences, which have been associated with amotivational psychopathology, reduced reward anticipation, and diminished activity engagement in SSD (Pillny et al., 2024), contributing to well-documented reductions in anticipatory pleasure (Hallford & Sharma, 2019) which in turn impede goal-directed activity in daily life (Edwards et al., 2018; Pillny et al., 2020).

Increased effort sensitivity may result in a subjective overestimation of effort costs as shown in SSD (Culbreth, Chib, et al., 2024). It may also be linked to dysfunctional attitudes toward effort, such as defeatist performance beliefs (e.g., "It's not worth the effort") and low success expectancies (e.g., "Even when I try my hardest, I won't succeed"), which can amplify perceived effort costs and undermine reward anticipation (Pillny et al., 2018; Saperia et al., 2025). A recent study investigating pupil dilation as a biomarker of effort exertion in response to a cognitive effort task in SSD found a significant correlation between reduced pupil dilation (indicating diminished effort exertion) and the severity of defeatist beliefs (Bismark et al., 2024). Moreover, Bismark et al. found an interaction between defeatist beliefs and increasing effort costs, such that individuals with more severe defeatist beliefs showed reduced pupil dilation at higher difficulty levels, underscoring the link between pronounced defeatist beliefs and increased effort sensitivity.

Cognitive Mechanisms and Unsystematic Effort Allocation

Alterations in cognitive computational parameters appeared more pronounced in SSD than in BD or MDD, likely reflecting disorder-specific challenges in processing and integrating decision-relevant information. In SSD, higher decision thresholds, slower evidence accumulation, and maladaptive range adaptation point to difficulties in gathering and processing information before making decisions. These impairments may prolong decision making and contribute to the unsystematic effort allocation commonly observed in SSD.

Consistent with this, our exploratory analyses suggest that greater cognitive impairment in SSD is associated with diminished increase in effort allocation from low- to high-reward/probability conditions. Specifically, cognitive impairment in SSD appears to selectively diminish the ability to integrate information of increasing reward magnitude and probability into effort-reward computations, rather than exerting a generalized demotivating effect on high-effort choices. Among cognitive domains, this effect may be most pronounced for working memory (Whitton et al., 2024) and, indicated by our moderator analyses, by processing speed. Although current evidence is insufficient to draw definite conclusions in this regard.

By contrast, we did not find an overall moderating effect of cognitive functioning on differences in EBDM. This could be explained by the dissociation between the constructs measured by neuropsychological tests and those captured by cognitive parameters in computational modeling studies. While neuropsychological tests often assess broader and general cognitive abilities, such as processing speed or working memory, the cognitive parameters estimated in computational modeling studies such as drift rate, decision thresholds, and nondecision times are more directly tied to cognitive operations and are directly inferred from behavioral outcomes in the EBDM tasks. These computational parameters may provide a more direct measure of how cognitive deficits manifest in specific decision-making contexts and contribute to unsystematic effort allocation.

Neurobiological Basis of the Putative Mechanisms

Dopamine dysregulation has been widely implicated in the pathophysiology of SSD, MDD, and BD, with potential links to altered EBDM and amotivational psychopathology (Argyropoulos & Nutt, 2013; Arjmand et al., 2018; Berk et al., 2007). Although dopamine-related mechanisms are thought to contribute to the neural dysfunctions driving altered EBDM and amotivational psychopathology, our review provides only vague evidence supporting this notion. Terenzi et al. (2024) cautiously interpreted their findings as suggesting dopamine dysregulation might underlie altered effort-discounting behaviors, particularly in high-effort conditions. Still, it should be noted that their conclusions are speculative, relying solely on behavioral patterns rather than direct assessments of dopaminergic function, such as through positron-emission tomography or pharmacological studies. Nevertheless, a large body of prior research highlights dopamine's role in effort-related processes, particularly reward anticipation and effort-cost valuation (Kirschner et al., 2016; Le Heron, 2018; Salamone & Correa, 2024; Webber et al., 2021). However, direct evidence linking dopamine dynamics to EBDM in SSD, MDD, and BD remains limited, highlighting the need for studies using more direct methodologies.

Moderator Analyses

Task Characteristics

The findings from the task moderator analyses revealed no clear evidence that any specific task characteristics modulated the effects of EBDM deficits between clinical and control groups. Despite the number of probability conditions showing modulating effect sizes in SSD, the overall lack of significant moderation suggests that the overlap in constructs captured by different EBDM tasks is substantial. This observation contrasts with previous studies that found rather low intercorrelations between different EBDM tasks. The rather weak association across EBDM tasks has been interpreted as evidence that EBDM tasks may capture partly distinct facets of motivation (Horan et al., 2015; Renz et al., 2023). In contrast, the consistency of EBDM deficits across various task designs, revealed by our meta-analysis, speaks to the robustness of these deficits in SSD, MDD, and BD, reinforcing their potential as transdiagnostic markers of amotivational psychopathology.

At the same time, we were unable to explain the substantial between-study heterogeneity of 55% through the moderators examined. This moderate heterogeneity suggests that additional, unmeasured factors contribute to variability in EBDM outcomes and should be investigated in future work.

Although significant moderation effects were observed for task payoff in MDD and for task type and effort measure in BD, these findings must be interpreted with extreme caution given the very small number of studies in some categories and the uneven distribution of moderators in these subgroup analyses. If valid, the MDD finding could suggest that reduced differences to healthy controls emerge when participants are only partially rewarded, possibly because limited payoffs dampen motivation across both groups and thereby obscure disorder-specific deficits. In BD, the smaller group differences in studies using progressive ratio tasks and breakpoints might indicate that these paradigms are less sensitive to group differences than proportion-based measures of hard task choices. Still, given the low representation of these task characteristics in these subgroup analyses, interpretations are rendered speculative and should be considered as hypotheses for future research rather than definite conclusions.

Sample Characteristics

Studies with a higher proportion of ethnic minority participants showed larger differences in EBDM between clinical and control groups. This may be explained by the impact of social exclusion and adversity on motivation (Jaya et al., 2017, 2021; Lincoln et al., 2021; Pillny & Lincoln, 2020; Weittenhiller & Kring, 2025). Experiences of social exclusion, along with related dynamics such as cultural mistrust, stereotype threat, and encounters with unfair treatment, disproportionately affect ethnic minority individuals and may amplify mechanisms underlying amotivational psychopathology. These interrelated sociocultural and psychosocial factors may therefore account for the heightened motivational deficits observed in clinical samples with more ethnic minority participants as seen in the current meta-analysis. This finding highlights the need for interventions tailored to address the specific challenges faced by ethnic minority patients experiencing amotivation.

The moderation analyses also revealed a trend-level effect for the proportion of female participants, with smaller differences in EBDM between clinical and control groups observed in samples with a higher proportion of females. This could be explained by research suggesting higher prevalences of amotivational psychopathology among males than females (e.g., Carter et al., 2022), which may account for the larger clinical-control differences observed in male-dominated samples. Thus, gender may modulate the severity of EBDM impairments, reflecting broader differences in symptomatology and motivational processes across genders. Further research

is needed to clarify these dynamics and explore the implications for clinical assessment and intervention.

Clinical Characteristics

The moderator analyses of clinical characteristics revealed critical insights into the relationship between symptomatology and EBDM deficits. Both positive and negative symptoms significantly moderated EBDM deficits across the clinical samples, underscoring their broad association with psychotic symptomatology. Specifically, the severity of positive symptoms was a significant moderator of EBDM deficits in the overall clinical sample and in individuals with SSD. Negative symptoms also emerged as significant moderators across clinical samples, with a trend-level effect observed in SSD. These findings suggest that EBDM deficits are relatively nonspecific, encompassing both positive and negative symptoms, and may reflect a general link to psychotic symptomatology rather than being confined to specific symptom domains. Notably, the findings expand on the previous meta-analysis in SSD by Blouzard et al. (2023), which failed to identify a significant moderation effect of negative symptoms, despite observed within-study correlations between EBDM and negative symptoms.

This discrepancy may arise from the limitations of the "first-generation" negative symptom scales predominantly used in the studies included in the current meta-analysis. First-generation scales have been criticized for failing to differentiate internal experiences from motivated behaviors and conflating separable symptoms or processes. By contrast, second-generation scales were designed to address these limitations and have demonstrated stronger correlations with specific negative symptom domains. A previous meta-analysis investigating the convergent validity of self-reported, clinician-rated, and performance-based measures of motivation has demonstrated significantly stronger correlations of EBDM deficits with second-generation negative symptom scales than with first-generation scales (Luther et al., 2019). Further, EBDM tasks have been shown to correlate more strongly with ecological momentary assessment of motivational negative symptoms than with clinician-rated scales and to predict engagement in daily goal-directed behavior (Barch et al., 2023; Moran et al., 2017). Taken together, this suggests that EBDM tasks and clinician-rated negative symptom scales may assess distinct but complementary aspects of motivational impairment. While EBDM tasks may reflect real-time behavioral manifestations of negative symptoms, clinician-rated scales might provide a broader assessment of subjective motivational experiences.

Depressive symptoms did not significantly moderate EBDM deficits in any diagnostic group, including SSD. Thus, despite the significant but small correlation between depressive and negative symptoms in SSD ($r = .19$; for a meta-analysis, see Edwards et al., 2019), the absence of a significant moderation by depressive symptoms indicates that reduced EBDM in SSD is more likely attributable to amotivational symptoms rather than depressive mood or other MDD symptoms. Although speculative, this could reflect the heterogeneity of depressive syndromes, with some individuals experiencing little to no amotivational psychopathology. This, in turn, suggests that the link between EBDM deficits and amotivational psychopathology is not primarily driven by depressive symptoms such as negative mood or low positive affect.

Limitations

The current meta-analysis has some limitations. The moderate between-study heterogeneity remained unexplained by moderator analyses, which was most likely due to extremely unequal cell sizes in some of the individual analyses. This may have reduced the reliability and generalizability of the current findings. Moreover, the unexplained heterogeneity suggests that there are important factors influencing the EBDM deficits in SSD, MDD, and BD that have not been identified by the current meta-analysis and require further research. Furthermore, the current meta-analysis included relatively few studies on BD compared to SSD and MDD. This imbalance may limit the robustness of conclusions about BD. On a related note, most BD studies did not specify whether participants were in depressive, manic, or euthymic phases, potentially obscuring phase-specific EBDM patterns. Another potential confound in the present meta-analysis is that most individuals in the SSD and BD samples were treated with dopamine-antagonistic medication, which could, in principle, impair EBDM. However, this concern is mitigated by our moderation analyses, which provided no evidence that chlorpromazine-equivalent daily dose significantly moderated EBDM differences between clinical and control groups. Last, cross-study comparisons of computational modeling studies remain an unresolved challenge. A meta-analytic aggregation was not feasible due to substantial variability in computational approaches and the parameters reported across studies. Furthermore, the limited number of transdiagnostic comparisons constrains the interpretability and generalizability of results across diagnostic groups. Nevertheless, this study is the first to systematically synthesize the available evidence from computational modeling studies and to apply a transdiagnostic perspective, providing a foundation for future research in this area.

Directions for Further Research

The findings of this meta-analysis highlight several critical gaps in our understanding of EBDM and amotivational psychopathology that warrant further research. First, the unexplained moderate heterogeneity in EBDM outcomes across studies underscores the need to identify additional factors influencing variability, such as unexamined task characteristics or participant-specific variables. Second, the differential mechanisms underlying EBDM deficits across SSD, MDD, and BD, particularly the role of cognitive impairments in SSD versus motivational biases in MDD, suggest the importance of further exploring disorder-specific pathways to amotivational psychopathology. Third, the lack of significant EBDM impairments in at-risk populations raises questions about whether these deficits emerge as a consequence of disorder progression or reflect a diagnostic threshold, necessitating longitudinal studies. Finally, the influence of demographic factors, such as ethnicity and gender, on EBDM outcomes remains underexplored and warrants deeper investigation to develop culturally and contextually sensitive interventions. Addressing these gaps could deepen our understanding of EBDM in clinical psychology and psychiatry, refine diagnostic models, and inform the development of targeted treatments for amotivational psychopathology.

Conclusions

In conclusion, this transdiagnostic meta-analysis demonstrates that EBDM is consistently impaired in SSD, MDD, and BD,

whereas AR groups do not differ from controls. Across disorders, the largest deficits emerge under high-reward and high-probability conditions, indicating inefficient effort allocation precisely when exerting effort should be most advantageous. Meta-analytic and computational evidence converge on three partially overlapping mechanisms: altered effort and reward sensitivity, and cognitive dysfunction. These mechanisms appear to be weighted differently across diagnoses and subgroups, with cognitive dysfunction and unsystematic effort allocation more prominent in SSD, and motivational biases and increased effort sensitivity more central in MDD and BD. Together, these findings position EBDM as a promising transdiagnostic marker of amotivational psychopathology and highlight the need for targeted, diagnosis-specific interventions.

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