

抑郁症动机缺陷与努力决策的计算神经机制

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摘 要

抑郁症(MDD)的核心症状不仅体现在情绪低落, 更伴随严重的动机缺陷, 主要表现为情感淡漠与快感缺失。以往研究发现这些缺陷集中体现于基于努力的决策(EBDM)受损, 并在临床上显著影响患者的预后及加重其社会功能受损。然而目前对于抑郁症动机缺陷的具体症状维度界定尚存混淆, 并且关于这些决策行为背后的计算过程及其神经生物学机制仍有待深入整合。本文旨在厘清情感淡漠与快感缺失的异同, 并以EBDM为核心范式, 系统梳理抑郁症患者在权衡努力与奖赏时的行为特征。同时, 结合主观价值计算模型与神经影像学证据, 深入剖析动机障碍的计算机制与神经基础, 为理解抑郁症中动机缺陷的病理机制及开发症状针对性的诊疗策略提供综合性视角。

关键词

抑郁症, 情感淡漠, 快感缺失, 基于努力的决策, 计算神经机制

Motivational Deficits and Computational Neural Mechanisms of Effort Decision-Making in Depression

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Abstract

Major Depressive Disorder (MDD) is characterized not only by low mood but also by severe motivational deficits, primarily manifested as apathy and anhedonia. Previous studies have found that these deficits are concentrated in impaired effort-based decision-making (EBDM), significantly impacting patient prognosis and exacerbating social dysfunction. However, the specific symptom dimensions of motivational deficits in depression remain unclear, and the computational processes and neurobiological mechanisms underlying these decision-making behaviors require further integration. This review aims to clarify the similarities and differences between apathy and anhedonia. Using EBDM as a core paradigm, we systematically reviewed how patients with MDD behave when balancing effort and rewards. At the same time, by combining subjective value computational models with neuroimaging evidence, we analyzed the computational and neural basis of these motivational disorders. This work provides a comprehensive perspective for understanding the pathology of motivational deficits in MDD and for developing targeted treatment strategies.

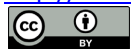
Keywords

Major Depressive Disorder, Apathy, Anhedonia, Effort-Based Decision-Making, Computational Neural Mechanisms

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1. 引言

抑郁症即抑郁障碍(Major Depressive Disorder, MDD), 是一种常见的精神障碍, 其核心临床特征包括持续的情绪低落、兴趣减退或快感缺失, 并常伴随睡眠、食欲和认知功能等多方面的损害(World Health Organization, 2017; McIntyre et al., 2023)。传统的临床关注点主要集中在情绪症状上, 但越来越多的证据表明, 动机缺陷是抑郁症谱系中一个同样重要的维度(Northoff, 2024)。抑郁症里的动机缺陷主要表现为情感淡漠(apathy)和快感缺失(anhedonia), 它们普遍存在, 并与更差的治疗反应、更高的复发风险和更严重的社会功能损害密切相关(Steffens et al., 2022; Connors et al., 2023)。情感淡漠(Apathy)被定义为目标导向行为、认知和情感的减少, 而快感缺失(Anhedonia)则指对愉悦体验能力的丧失(Lanctôt et al., 2023; Post and Warden, 2018), 两者往往共同出现, 但在一定程度上又相互独立, 各自拥有独特的神经生物学基础和临床意义(Lanctôt et al., 2023; Levy et al., 1998; Batail et al., 2018)。厘清二者的异同, 对于精确刻画患者的症状学特征和进行靶向治疗具有重要价值。

在行为层面, 动机缺陷集中体现为基于努力的决策(EBDM)障碍(Salamone & Correa, 2023; Brassard et al., 2024)。研究一致发现, MDD 患者倾向于规避高努力任务, 反映了其对努力成本的敏感性增加及对奖励主观价值的贬低(Cléry-Melin et al., 2011; Zou et al., 2020; Treadway et al., 2009)。为深入探究其认知机制, 计算建模(如主观价值 SV 模型)被用于分解奖励与努力敏感性等参数(Yee, 2024), 结合 fMRI 等技术, 可进一步定位价值评估与行动选择相关的异常脑网络(Kuhn et al., 2025; 陈富琴等, 2016)。

本文拟在现有研究基础上, 深入探讨抑郁症中动机障碍的不同症状维度, 即情感淡漠和快感缺失的概念及其异同。并且重点介绍 EBDM 作为研究动机缺陷的核心行为范式, 总结与梳理抑郁症患者的行为

表现以及计算神经等研究结果, 并在此基础上讨论当前研究的局限与未来的研究方向, 为理解和干预抑郁症的动机障碍提供综合性视角。

2. 抑郁症中的动机缺陷

2.1. 情感淡漠与流行病学

2.1.1. 情感淡漠的定义与维度

近年来, 情感淡漠(Apathy)作为抑郁症中一个独立且普遍存在的维度, 正日益受到研究者的重视。情感淡漠是一种动机减退的跨诊断综合征, 其特征是目标导向行为减少、情感反应减弱和自我激活的缺陷(Marin et al., 1991; Marin, 1996; Levy & Dubois, 2006), 常见于一些神经退行性疾病如阿尔兹海默症、帕金森病, 或精神疾病如抑郁症、精神分裂症中(Pedersen et al., 2009; Nobis & Husain, 2018; Van Reekum et al., 2005; Silva et al., 2021)。情感淡漠不仅显著影响患者的生活质量, 还加重了其生活负担(Pagonabarraga et al., 2015)。除了作为临床症状出现在许多不同的疾病中, 淡漠如今也被认为一定程度存在于健康人群里(Pardini et al., 2016; Brodaty et al., 2010; Kos et al., 2017)。Husain 和 Roiser 在其综述中指出, 淡漠作为一种跨诊断的动机缺陷, 其潜在机制在临床群体和普通人群中可能具有共同的神经认知基础(Husain & Roiser, 2018)。

关于淡漠的内部结构, 研究者提出了许多维度的模型。据以往研究, 淡漠包含以下三个子类型: 情绪情感淡漠(emotional apathy), 即无法参与动机所需的情感过程, 表现为情绪反应性降低和对奖赏刺激的钝化; 认知淡漠(cognitive apathy), 即制定计划的认知功能受损, 难以自主生成目标和组织行动序列; 以及自我激活缺陷(auto-activation deficit), 即自我启动思想和行为的困难, 需要外部提示才能发起活动(Marin & Wilkosz, 2005; Le Heron, 2018)。这种多维度的概念化有助于理解淡漠的异质性表现及其潜在机制。标准化淡漠测量工具的开发如 AES (Apathy Evaluation Scale), AMI (Apathy Motivation Index)进一步揭示了其稳定的维度结构, 更证实了其在临床与健康群体中的跨诊断特征, 有力支持了淡漠作为独立动机构念的合理性(Marin et al., 1991; Ang et al., 2017)。

2.1.2. 淡漠的神经基础

在神经层面, 有关情感淡漠的大量研究指向了前额叶-皮层下回路(frontal-subcortical circuits)的功能障碍, 特别是那些涉及目标选择、计划制定和行为启动的脑区(Bonelli & Cummings, 2007)。神经影像研究发现, 情感淡漠的严重程度与前扣带回(the anterior cingulate cortex, ACC)、眶额皮层(the orbitofrontal cortex, OFC)、脑岛(Insula)、纹状体与边缘系统的结构和功能异常显著相关(Pimontel et al., 2020; Steffens et al., 2022)。尤其是 ACC 在整合奖赏信息、评估努力成本以及行为启动中扮演重要角色, 其功能减退或缺陷可能是患者主动性减退的关键因素(Silva et al., 2021; Lafond-Brina et al., 2023)。此外, 基底节(特别是腹侧纹状体)的损伤会影响奖励处理和行为激活, 而背外侧前额叶皮层(DLPFC)的异常则与认知计划能力的下降有关(Levy & Dubois, 2006)。一些研究还发现, 连接这些关键节点的白质纤维束完整性受损, 也与情感淡漠密切相关(Torso et al., 2015; Hédouin et al., 2024), 这些发现共同塑造了一个复杂的神经网络, 其中任何一个环节受损都可能影响我们目标导向行为的达成, 从而产生或加重淡漠症状。

2.2. 快感缺失

2.2.1. 快感缺失的概念与维度

快感缺失(Anhedonia)是一个精神病理学概念, 它并非指单纯的“不高兴”, 而是指个体在体验快乐的能力上出现下降或丧失, 同时伴随着为获得愉悦而采取目标导向行为的动力缺陷。快感缺失在重度抑

郁症、精神分裂症等多种精神障碍中普遍存在(Pu et al., 2024), 被临床诊断上认为是重度抑郁症的核心标准, 研究表明, 35%~70%的抑郁症患者报告存在快感缺乏(Herr et al., 2025)。快感缺失的概念被分为特质(trait)取向与状态(state)取向(Yang et al., 2013), 特质取向将快感缺失视为一种相对稳定、与生俱来的人格特质, 状态取向将快感缺失看作一种在疾病(尤其是抑郁症)急性期出现的核心心理状态或症状, 包括对预期愉悦(anticipatory pleasure)和消费性愉悦(consummatory pleasure)的减退(Post & Warden, 2018; Ducasse et al., 2018)。研究发现, 抑郁症患者可能同时存在两种类型的快感缺失, 但期待性快感缺失似乎与抑郁症的动机症状和功能损害关系更为密切。例如, 期待性快感缺失的程度能够预测年轻抑郁症患者在日常生活中参与社交和休闲活动的频率及从中获得的乐趣。(Schulz et al., 2024; Sahni & McCabe, 2025)。还有研究发现, 处于亚临床抑郁状态的个体, 会表现出针对社会性奖赏的趋近动机特异性不足。而内源性抑郁症的核心特征也常被描述为这种广泛的、急性的愉悦感体验能力缺陷(Pu et al., 2024)。

2.2.2. 快感缺失的神经基础

在神经生物学上, 快感缺失与大脑奖赏回路功能障碍有关, 特别是多巴胺能中脑边缘通路, 涉及腹侧被盖区、伏隔核和前额叶皮层(Keedwell et al., 2005; Cao et al., 2019; Wardani & Lesmana, 2024)。期待性快感(“想要”)与中脑-边缘多巴胺通路有关, 该通路涉及从腹侧被盖区(VTA)到伏隔核(NAcc)的多巴胺投射, 并且负责编码奖励的预测和动机显著性(Gan et al., 2010; Salamone & Correa, 2023), 也就是说当面对可能获得奖励的线索时, 这条通路的激活会驱动个体去追求奖励。而消费性快感(“喜欢”)则被认为更多地依赖于 OFC 和脑内阿片肽系统等, 负责编码奖励带来的即时感官愉悦(Kaya & McCabe, 2019)。

在抑郁症中, 一项 fMRI 研究的元分析发现, 患者在奖励预期阶段, 腹侧纹状体等区域的活动显著低于健康人; 在奖励反馈阶段, 这些区域的活动同样减弱, 这为快感缺失的神经机制提供了有力证据(Zhao et al., 2024)。计算模型研究进一步指出, 快感缺失可能与奖励学习过程中的奖励预测误差(RPE)信号编码异常有关, 即大脑无法根据实际结果有效地更新对未来奖励的期望(Sahni & McCabe, 2025)。这些发现共同表明, 抑郁症中的快感缺失源于大脑奖励环路在价值编码、动机驱动和学习更新等多个环节上的功能失调。

2.3. 情感淡漠与快感缺失的区别与联系

大量研究在一系列神经退行性疾病与神经认知障碍背景下提示了情感淡漠与抑郁症的可分离性, 支持了情感淡漠作为独立综合征的观点, 他们指出区分情感淡漠与抑郁症对于这些背景下的患者制定合适的治疗和干预措施至关重要(Levy et al., 1998; Lanctôt et al., 2023)。而抑郁症患者中的淡漠研究则更多只集中在老年人群(晚年抑郁症患者中淡漠患病率高达 74.5%)(Groeneweg-Koolhoven et al., 2017)。但目前在临床上淡漠常被忽视或与抑郁症状混淆, 例如目前的一线抗抑郁治疗(如 SSRIs)不仅可能对淡漠无效, 甚至可能加剧其症状(Pimontel et al., 2020)。故而深化对抑郁患者中淡漠特异性的神经环路机制的理解, 有助于推动精准化治疗的发展。

在抑郁症患者中, 情感淡漠与快感缺失在临床表现上有显著重叠, 都涉及动机和兴趣的减退, 导致患者常常被描述为“缺乏动力”或“对任何事都提不起兴趣”。这种现象上的相似性使得在临床实践中区分两者颇具挑战(Post & Warden, 2018; Husain & Roiser, 2018)。从概念上讲, 其核心区别在于动机过程的不同阶段。快感缺失主要是一种与奖励价值评估和体验相关的缺陷, 尤其是情感体验的缺失(Kaya & McCabe, 2019)。而情感淡漠则是一种更广泛的障碍, 不仅涉及对奖励的反应减弱, 更突出地表现为将价值评估转化为目标导向行为的能力受损, 即行动的启动和维持障碍(Levy & Dubois, 2006)。Batail 等人(2018)对抑郁症患者的临床和神经心理学特征进行比较, 发现情感淡漠与快感缺失之间存在负相关, 提示情感淡漠可能代表抑郁症中不同的临床亚型(Lanctôt et al., 2023; Levy et al., 1998; Batail et al., 2018)。

在神经生物学上, 尽管两者都涉及前额叶-纹状体环路, 但其具体的神经基础存在差异。Mehrhof 等人(2025)通过一项在线游戏化任务和计算模型指出情感淡漠、快感缺失和晚期昼夜节律之间存在共同的努力决策改变(Mehrhof et al., 2025), 这表明在动机缺陷的核心, 可能存在一些共享的计算和神经机制。但另一些研究表明, 快感缺失更紧密地与腹侧纹状体、vmPFC 等核心奖励环路的功能障碍相关, 这些脑区主要负责编码奖励的价值和愉悦感(Zhao et al., 2024)。而情感淡漠则与一个更广泛的网络相关, 除了奖励环路, 还特别强调了背侧前扣带回(dACC)、辅助运动区(SMA)和背外侧前额叶皮层(DLPFC)等在认知控制、努力计算和行动选择中起关键作用的区域(Bonnelle et al., 2015; Steffens et al., 2022)。

综上, 情感淡漠和快感缺失是抑郁症动机缺陷的两个关键组成部分。快感缺失侧重于奖励体验的“情感”核心, 而情感淡漠则更侧重于目标导向行为的“行动”执行。虽然它们常常并存且相互影响, 但将它们区分为独立的、具有不同神经基础的症状, 对于我们理解抑郁症的异质性、进行更精准的诊断评估和开发靶向性更强的治疗策略至关重要。而针对努力决策的研究尝试提示我们, 理解抑郁症动机缺陷需要引入成本与收益权衡的量化框架。下一节将围绕基于努力的决策范式, 对这一过程进行系统梳理。

3. 基于努力的决策与计算神经机制

3.1. 基于努力的决策与相关研究

近年研究认为, 动机缺乏的核心机制在于成本-收益权衡决策的异常(Chong et al., 2016; Le Heron, 2018)。基于努力的决策(Effort-Based Decision-Making, EBDM)通过描述个体整合行动成本与潜在回报的过程, 成为量化动机的核心范式(Vandenbos et al., 2006; Kurniawan et al., 2011)。其中, Treadway 开发的 EEfRT 任务应用最为广泛(Treadway et al., 2009), 该任务要求被试在不同奖励大小及概率下, 通过权衡体力消耗(如按键)选择不同难度的任务, 从而评估努力分配模式。此外, 为区分动机缺陷的特异性, 研究者还开发了对比体力与脑力努力(如 N-back)的范式(Tran et al., 2021), 以及涉及认知难度选择(Apps et al., 2015)和虚拟觅食等任务(Bustamante et al., 2024)。这些范式为多维度探究动机构成提供了重要工具。

大量运用 EBDM 范式的研究共同指向一个核心发现: 与健康对照组相比, 抑郁症患者在 EBDM 任务中普遍表现出对高努力任务的规避和对努力成本的敏感性增加(Berwian et al., 2020; Cléry-Melin et al., 2011; Valton et al., 2025)。而其中, 情感淡漠与 EBDM 功能障碍显著相关, 更高的情感淡漠评分者, 也更因高努力而拒绝机会, 并且这种现象跨疾病出现(Prévost et al., 2010; Bonnelle et al., 2015; Bonnelle et al., 2016; Le Heron, 2018)。Saperia 等人(2023)通过计算表型分析, 揭示了精神分裂症、抑郁症和健康对照组在利用成本-效益信息进行努力决策方面存在有意义的个体差异(Saperia et al., 2023)。这些发现共同强调了 EBDM 范式作为一种客观的行为测量工具, 有力地证实了抑郁症患者存在显著的动机缺陷, 并且在症状维度可能与情感淡漠高度相关。区分不同类型的努力(体力/脑力)以及在不同疾病和症状维度间进行比较, 将是未来行为研究深化我们对动机障碍理解的关键方向(Wen et al., 2025)。

3.2. 主观价值计算

计算建模(computational modeling)近年来常被使用在精神疾病研究中, 用数学模型来描述、解释和预测精神疾病的发生、发展和表现(Huys et al., 2021; Liu et al., 2020)。成本-收益权衡的本质是主观价值(Subjective Value, SV)的动态计算过程, 主观价值理论为理解疾病与基于努力的决策之间的关系提供了理论框架。行为的执行取决于是否有足够的 SV 值, 奖励的主观价值计算可能由于努力、概率等成本因素而被贬值(O'Brien & Ahmed, 2019; Salamone & Correa, 2023; Chong et al., 2017)。但通过将试在 EBDM 任务中的一系列选择或反应时数据拟合到这些不同的模型中, 研究者可以比较哪个模型能最好地解释行为数据。

既往疾病研究发现, 在伴有淡漠症状的精神分裂症患者中, 其努力敏感性显著高于普通人群, 这些患者表现出更不愿意为获得奖赏而付出努力, 而奖赏敏感性则无显著差异。这表明, 在此类患者的 EBDM 中, 努力敏感性起主导作用(Culbreth et al., 2025)。然而, Valton 等人(2025)发现, 抑郁症患者为获得奖赏而付出努力的意愿降低, 主要是由于整体努力接受倾向降低, 而非努力敏感性或奖赏敏感性的改变(Valton et al., 2025)。有趣的是, 在健康个体中, 努力成本和奖赏量也被整合到 SV 中, 以指导个体的决策。模型显示, 健康个体的 SV 受奖赏敏感性和努力敏感性的共同影响, 且两者在神经水平上具有独立表征(Westbrook et al., 2019; Yao et al., 2023)。

这表明动机缺陷人群存在 SV 计算异常, 并不被疾病所限制, 并且抑郁症的“动机缺乏”不是一个单一的概念, 而是可能由多个潜在计算过程的异常所共同导致。识别出主导特定患者动机缺陷的计算机制以及对应相关的症状维度, 对于实现个性化治疗至关重要。

3.3. 抑郁症中的计算神经环路异常

3.3.1. 主观价值计算的神经机制

过去十多年的研究已经呈现相对清晰的价值决策神经环路。大量研究得出, 关于成本 - 价值计算在特定过程涉及不同的核心脑区, 例如腹侧纹状体(VS)和腹内侧前额叶皮层(vmPFC)是编码主观价值的核心脑区(Peters & Büchel, 2009; Croxson et al., 2009), 当某个选项的综合主观价值更高时, 这两个脑区的活动就越强(Smith et al., 2010)。与价值编码不同, 对于努力成本, 前扣带回(ACC, 特别是其背侧部分 dACC)和前脑岛(anterior insula)被认为是关键的编码区域(Skvortsova et al., 2014), ACC 还被认为在整合奖励信息和努力信息, 从而在“是否值得努力”的权衡中发挥核心作用(Vassena et al. 2014; Verguts et al., 2015)。另外, dACC 和辅助运动区(SMA)充当了价值比较与选择的角色, 一旦做出选择, 运动皮层和基底节的其他部分(如壳核)则负责将决策转化为具体的行动(Kurniawan et al., 2011; Klein-Flügge et al., 2016)。多巴胺(DA)被广泛认为在价值决策中扮演核心角色, 尤其是在编码奖励预期和驱动动机行为方面(Salamone & Correa, 2023)。然而研究也表明, DA 信号更多地编码预期的收益, 而不是行动的净值(Gan et al., 2010)。基于上述健康大脑的决策模型, 研究者们开始探究抑郁症患者的动机缺陷是否与这个价值决策网络的特定环节功能异常有关。

3.3.2. 抑郁患者的计算神经环路异常

研究者们发现, 与健康对照组相比, 抑郁症患者在奖励预期阶段, 外侧前额叶 - 丘脑环路的活动减弱, 尤其是在腹侧纹状体区域; 在奖励结果呈现阶段, 右侧纹状体和前额叶皮层的活动也显著降低。这些发现与快感缺失的“奖励钝化”(reward blunting)假说高度一致, 即抑郁症患者的大脑奖励系统对积极刺激的反应性下降, 导致他们无法正常地编码和体验奖励的价值(Zhao et al., 2024)。进一步的研究将这种神经活动异常与计算模型中的参数联系起来。Arulpragasam 等人的研究发现, vmPFC 编码了预期的主观价值, 而 dACC 和脑岛的活动则反映了努力折扣的程度以及价值预测误差信号(Arulpragasam et al., 2018)。在抑郁症中, 这些脑区之间信号传递的失衡可能最终导致了回避努力的决策偏好。

除了脑区活动的改变, 脑网络连接的异常也是抑郁动机缺陷的重要机制。情感淡漠的严重程度被发现与 ACC 和 SMA 之间的功能连接减弱有关, 这可能反映了将行动意图有效转化为行动指令的能力受损(Bonnelle et al., 2015)。在老年抑郁症中, 情感淡漠也与默认网络、执行控制网络等多个大尺度脑网络内部及之间的连接异常有关(Oberlin et al., 2022; Roy et al., 2023)。这些网络层面的功能障碍, 使得抑郁症患者难以有效地整合内在状态、外部信息和长期目标, 从而导致了目标导向行为的瓦解。

综上所述, 计算神经科学的研究, 通过结合行为范式、数学模型和神经影像技术, 极大地深化了我

们对抑郁症动机缺陷的理解。研究结果表明, 抑郁症的“动力缺乏”并非一个模糊的表象症状, 而是根植于大脑价值决策系统中特定计算和神经环路的功能障碍。这些发现为理解抑郁症的动机缺陷的病理生理机制开辟了新途径, 也为未来开发基于神经环路和计算过程的精准干预措施提供了理论基础。

4. 基于计算神经机制的临床干预与转化

理解抑郁症动机缺陷的计算与神经机制, 不仅有助于病理机制的阐释, 更为优化临床治疗策略提供了关键的理论依据。目前的转化医学研究正致力于理解不同干预手段如何重塑 EBDM 相关的计算参数及神经环路。

在药物治疗方面, 不同机制的抗抑郁药对动机相关计算过程的影响存在明显差异。传统的选择性 5-羟色胺再摄取抑制剂(SSRIs, 如西酞普兰), 虽能改善部分抑郁症状, 但其对动机缺陷(尤其是情感淡漠)的疗效有限(McCabe et al., 2010), 甚至 SSRIs 在部分患者中可能诱发情感淡漠(Barnhart et al., 2004)。相比之下, 作用于多巴胺和去甲肾上腺素系统的药物(NDRIs)通过增强前额叶-纹状体多巴胺信号, 可能更有效地改善快感缺失与努力决策障碍(Cao et al., 2019)。一项 fMRI 研究进一步表明, 安非他酮通过增强涉及奖赏预期、动机驱动(努力)和愉悦体验的关键脑区(如 vmPFC、纹状体、眶额皮层)的神经反应, 可能促进抑郁症患者的奖赏寻求和厌恶回避行为。此增强效应与之前研究发现的西酞普兰会减弱奖赏和厌恶神经反应的结果形成鲜明对比, 解释了为何安非他酮在理论上更不易引起情绪钝化副作用, 并可能更好地改善奖赏相关缺陷(Dean et al., 2016)。从计算模型的角度看, 这种增强可能通过提高参数中的“奖赏敏感性”或“预期主观价值”, 从而在成本-收益权衡中抵消努力成本的负面影响, 促进目标导向行为的产生。这提示, 未来药物治疗可结合 EBDM 任务与计算模型分析, 实现对患者动机缺陷亚型的精准匹配。

在心理治疗方面, 行为激活疗法(BA)和认知行为疗法(CBT)通过系统且有层次地引导患者自我发现与参与目标导向活动, 可能改善其不良的努力成本评估与主观价值计算过程。Dichter 等人(2009)的研究表明, 简短行为激活疗法(BATD)能够有效缓解抑郁症状, 具体表现为在奖赏预期阶段增强了背侧纹状体的活动, 在奖赏选择和反馈阶段调节了旁扣带回和眶额皮层的活动, 这在计算层面上可能反映了该疗法通过鼓励目标导向行为, 加强了“行为-结果”之间的联结, 这正是抑郁症患者所缺乏的(Dichter et al., 2009)。此外, König 等人(2025)的综述系统总结 CBT 对 MDD 患者大脑活动的纵向影响。研究发现, CBT 治疗后抑郁患者在与情绪和奖赏处理相关的皮质-边缘脑环路和奖赏系统中出现了显著的脑活动变化, 与症状改善相关。研究结果支持了 CBT 通过调节特定神经环路来缓解抑郁症状的机制, 关键脑区的活动变化有望成为治疗反应的潜在生物标志物。这些发现提示, 心理治疗不仅可从行为层面改善回避倾向, 还可能通过神经可塑性改善价值决策环路的功能失调。

综上, 未来研究方向应进一步整合计算精神病学与临床神经科学, 开发基于 EBDM 参数与神经环路特征的预测模型, 用于区分适合多巴胺能药物还是心理治疗的患者亚群, 进一步指导药物选择、优化心理治疗靶点, 从而更科学地推动临床转化向基于计算神经机制的治疗发展。

5. 总结与展望

本综述系统梳理了关于抑郁症动机缺陷的研究进展, 重点关注了情感淡漠与快感缺失的概念与维度、基于努力决策(EBDM)的行为研究, 以及计算神经科学在该领域的临床转化与应用。通过整合多方面的证据, 我们能够从临床症状到行为模式、从计算机制到神经基础多层次系统地解释与理解抑郁症的动机障碍。

概念层面: 情感淡漠和快感缺失是两个既有重叠又可区分的核心动机缺陷。快感缺失更侧重于奖励价值体验的受损, 而情感淡漠则是一种更广泛的、涉及目标导向行为启动和维持的障碍。两者在临床意

义和神经基础上存在差异, 区分它们对理解抑郁症的异质性至关重要。

行为层面: EBDM 范式提供了一种客观量化动机水平的有效工具。大量研究一致表明, 抑郁症患者在 EBDM 任务中倾向于回避高努力选项, 反映了他们对奖励价值的低估或对努力成本的过度敏感。这些行为表现与快感缺失、情感淡漠等临床症状密切相关。

计算层面: 计算模型, 特别是主观价值(SV)模型, 使我们能够超越行为表象, 探究决策背后的认知机制。研究提示, 抑郁症的动机障碍可能并非简单的奖励或努力敏感度改变, 而可能在不同症状维度上有不同节点的价值评估缺陷。

神经层面: 大量神经影像研究已经识别前额叶-纹状体环路的结构与功能连接受损是导致动机障碍的关键病理生理机制, 包括价值编码的 vmPFC、VS; 成本编码的 ACC、脑岛, 以及进行价值比较的 dACC、SMA 等。抑郁症的动机缺陷与这些环路的功能障碍密切相关, 表现为奖励相关脑区的活动减弱和不同节点间功能连接的异常。神经层面也区分了情感淡漠和快感缺失。快感缺失主要涉及腹侧纹状体和 vmPFC; 而情感淡漠与 ACC、SMA 及背外侧前额叶皮层的功能异常更为密切。

临床转化层面: 基础研究的最终目的是指导临床实践。目前的证据表明, 针对动机缺陷的异质性, 药物和心理治疗的作用靶点存在差异。例如, NDRI 类药物可能更特异性地通过调节多巴胺通路来降低努力成本敏感性, 而心理治疗则可能更多地通过重塑认知评价和奖赏学习机制来发挥作用。将 EBDM 的计算参数作为临床评估的一部分, 有望为个体化治疗方案的选择提供客观依据, 从而指导个体化和针对性的治疗选择。

尽管大量研究推动对抑郁症动机缺陷以及具体维度上的区分取得了一定的进步, 但仍存在一些局限, 同时也为未来的研究指明了方向。当前研究已经区分体力努力和脑力努力, 但对于不同类型的脑力努力(如工作记忆、注意力控制)如何差异性地影响决策, 以及它们与不同症状维度的关系, 仍需更深入的探索。此外, 将 EBDM 范式与社会情境相结合(Catalano & Green, 2023; Shao et al., 2024), 探究社会动机在抑郁症中的缺陷, 也是研究者们逐步探索的领域。另外, 情感淡漠和快感缺失广泛存在于精神分裂症、帕金森病等多种疾病中。未来应继续开展跨诊断研究, 整合多模态数据, 为动机缺陷在不同精神疾病中是否存在共享的计算表型和神经标记物提供更多实证证据。

综上所述, 以 EBDM 为核心行为范式, 以计算建模、神经影像为手段的研究路径, 正在不断加深我们对抑郁症动机缺陷这一复杂临床问题的认识。未来的研究应当继续沿着这一路径, 致力于将基础的实验发现转化为能够切实改善抑郁症患者生活质量的临床实践。

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