

• 综述 •

青少年抑郁障碍非自杀性自伤机制与干预进展

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[摘要] 抑郁障碍已成为青少年最常见且危害显著的心理疾病之一, 非自杀性自伤(non-suicidal self-injury, NSSI)在青少年抑郁障碍中的发生率持续上升, 对青少年的身心健康构成严峻挑战。既往多项研究提示, 青少年抑郁障碍及NSSI的发生机制复杂, 涉及神经递质水平、激素变化、免疫炎症反应、遗传易感及心理社会等多重因素的交互作用。NSSI不仅是抑郁障碍患者的重要危险行为表现, 也与自杀风险密切相关。当前针对青少年抑郁障碍与NSSI的治疗以心理治疗为核心, 药物及物理治疗等多模式综合干预为辅, 不同干预方式在NSSI行为改善及预后方面显示出一定差异。文章在梳理相关流行病学、病理机制及风险因素基础上, 系统总结了近年来青少年抑郁障碍合并NSSI的主要研究进展及临床干预策略, 旨在为进一步优化诊疗模式、提升患者预后及生活质量提供理论参考。

[关键词] 青少年; 抑郁障碍; 非自杀性自伤; 发病机制; 治疗

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The mechanisms of non-suicidal self-injury in adolescents with depressive disorders and progresses of intervention

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[Abstract] Depressive disorders have become one of the most common and significantly harmful psychological conditions among adolescents. The incidence of nonsuicidal self-injury (NSSI) in adolescents with depressive disorders is on the rise, posing a severe challenge to their physical and mental health. Previous studies have indicated that the mechanisms underlying adolescent depressive disorders and NSSI are complex, involving the interplay of multiple factors, including neurotransmitter levels, hormonal changes, immune-inflammatory responses, genetic susceptibility, and psychosocial influences. NSSI is not only an important manifestation of risky behavior in patients with depressive disorders but also closely related to suicide risk. Current treatments for adolescent depressive disorders and NSSI are primarily centered on psychotherapy, with multimodal interventions, including pharmacotherapy and physical treatments, as adjuncts. Different intervention approaches have shown certain differences in improving NSSI behaviors and prognosis. This article reviews the relevant epidemiology, pathophysiological mechanisms, and risk factors, and systematically summarizes the major research advances and clinical intervention strategies for adolescent depressive disorders with NSSI in recent years, aiming to provide theoretical references for further optimizing treatment models and improving patients' prognosis and quality of life.

[Key words] adolescent; depressive disorder; nonsuicidal self-injury; pathogenesis; treatment

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抑郁障碍是一类病因复杂的精神疾病,主要表现为持续性情绪低落,常伴有不同程度的认知功能受损及行为改变,具有高发病率、高复发率和高致残率等显著特点。青少年时期是抑郁障碍首次发作的高发期,约1/4的青少年在该阶段罹患抑郁障碍。与成人相比,青少年抑郁障碍的复发率更高,其反复发作显著损害青少年的关键社会心理功能^[1],增加了评估和诊疗的难度。此外,青少年抑郁障碍还可导致多种严重不良后果,其中最为严重的是自杀行为的发生。

非自杀性自伤(non-suicidal self-injury, NSSI)已被纳入美国《精神障碍诊断与统计手册》第5版作为独立的临床诊断单元^[2],其定义为在无明确自杀意图下,个体有意损伤自身身体组织的行为,常见形式包括划伤、烫伤、自我殴打、用头部撞击物体、咬伤、掐伤、抓伤等^[3]。青春期是NSSI的高发阶段,全球范围内青少年NSSI的发生率为17.2%,中国青少年NSSI的总体发生率为14.6%,而在住院患者中这一比例更高,达51.5%,且常与抑郁障碍、焦虑障碍等共病^[4]。此外,NSSI与自杀密切相关,且可引发多种不良行为结局。近年来NSSI呈上升趋势,已对青少年身心健康构成严重威胁,成为该群体不可忽视的重要精神卫生问题。因此,深入探讨青少年抑郁障碍NSSI的独特发病机制,结合最新治疗手段个体化精准治疗,有助于更好地改善患者预后。文章系统梳理了青少年抑郁障碍及NSSI在发病机制、神经生物学基础及干预路径等方面的最新研究进展,以期临床早期识别、综合干预及个体化管理提供科学建议和参考。

1 青少年抑郁障碍的独特发病机制

1.1 脑影像学 and 脑功能连接

多项研究证实,抑郁障碍的发生与神经发育密切相关。一项研究发现,抑郁障碍发病年龄与灰质变化有关,患者在磁共振成像中可见额叶、小脑、顶叶及颞叶等脑区不同程度的萎缩^[5]。Guo等^[6]的研究进一步发现,青少年抑郁障碍患者在左背外侧前额叶皮层、右眶额叶皮层、左岛叶及前扣带回皮层等多个区域的功能连接显著下降,提示结构与功能之间的异常相互作用可能是其重要发病机制之一。大脑对情绪和认知的整合能力可通过神经时间尺度反映,青少年抑郁障碍患者皮层及部分皮层下区的神经时间尺度较健康对照组显著缩短,这可能与情绪及认知功能受损相关^[7]。此外,反刍思维

是青少年抑郁障碍患者的主要思维特征之一,岛叶和背侧前扣带回的活动与反刍思维密切相关^[8]。大量研究证实青少年抑郁障碍患者存在脑结构及功能的多方面异常,为疾病的发生与发展提供了神经生物学基础。青少年正处神经发育关键期,亟需更多研究进一步阐明相关脑区的具体改变及其机制。

1.2 神经递质及激素

神经递质在抑郁障碍的发生与发展中具有关键作用。单胺假说是抑郁障碍的重要发病机制之一,血清素作为核心成分,在抑郁障碍患者的神经回路中含量降低,血清素转运蛋白是突触间隙中血清素再摄取的主要载体,也是当前临床常用抗抑郁药物的重要作用靶点^[9]。青少年抑郁障碍的性别患病率存在显著差异,Oyola等^[10]研究表明,慢性应激状态下,下丘脑-垂体-肾上腺(hypothalamic-pituitary-adrenal, HPA)轴功能紊乱,性腺类固醇对HPA轴的调控存在性别差异,导致HPA轴的响应性也存在性别特异性变化,这为阐释青春期抑郁障碍的性别差异提供了依据。此外,尽管针对青少年的研究发现,HPA轴和下丘脑-垂体-甲状腺轴与抑郁障碍仅部分相关,但下丘脑-垂体-甲状腺轴失调也是青少年抑郁障碍发生、发展的重要影响因素之一^[11]。

1.3 炎症因子

炎症因子与青少年抑郁障碍的发展密切相关,其中白介素(interleukin, IL)-2水平主要与男性抑郁的严重程度相关,而IL-6则更显著地与女性抑郁相关^[12]。同时,干扰素 α/β 介导的促炎通路激活和青少年抑郁障碍的发生相关^[13]。C反应蛋白(C-reactive protein, CRP)水平与成年人抑郁障碍的严重程度呈正相关,然而在青少年抑郁障碍患者中尚未观察到CRP与抑郁严重程度之间的相关性^[14],这提示CRP在青少年抑郁障碍中可能具有独特的识别意义。一项针对18岁以下人群的研究发现,抑郁障碍患者的肿瘤坏死因子(tumor necrosis factor, TNF)- α 水平高于对照组^[15]。此外,血清中吲哚胺2,3-双加氧酶活性指数与IL-1 β 、IL-6、IL-10及TNF- α 呈正相关,3-羟基犬尿氨酸与IL-8也表现出正相关性^[16]。综上,尽管已有大量针对青少年抑郁障碍与炎症因子的相关研究,但两者之间的确切关系尚不明确,需进一步探索具体作用机制及临床应用价值。

1.4 遗传

既往研究显示,抑郁障碍患者后代罹患儿童精神障碍的风险显著增加,提示抑郁障碍具有较高的遗传易感性^[17]。在青少年群体的相关研究中,发现

血清素转运蛋白基因启动子区的多态性等位基因表达差异可能促进抑郁障碍的发生,尤其对青少年女性影响显著^[9],该结果可能部分解释了青少年期男女抑郁障碍患病率存在差异的原因。Zhao等^[18]的研究表明,在青少年抑郁障碍患者中,CNTNAP3、IL1RAP、MEGF9、UBE2W、UBE2D1等5种基因与免疫、炎症因子及神经系统密切相关。另有研究指出,在抑郁症状较重的青少年中,在促炎转录因子活性增强、核因子活性提升、糖皮质激素受体活性降低及干扰素反应因子作用下,炎症相关基因上调,而抗病毒相关基因则下调^[19]。此外,抑郁障碍的全基因组关联分析发现,大脑和全血中的HACE1基因与抑郁障碍密切相关^[20],尽管该研究对象为成年人,但为儿童青少年抑郁障碍的遗传学研究提供了新方向。

1.5 心理社会因素

青少年抑郁障碍的发生与多种心理社会因素密切相关。社会压力与排斥反应可通过调节神经通路,影响炎症相关的免疫系统,进而诱发抑郁障碍^[21]。研究证实,童年逆境经历显著增加抑郁障碍和焦虑障碍的发生风险^[22]。童年逆境主要包括情感虐待、家庭功能失调、暴力及低社会经济地位4类,其中情感虐待组的抑郁风险最高,低自尊在情感虐待与抑郁症状之间起中介作用^[23]。青少年抑郁障碍的发病风险还与童年时期心理压力事件的数量有关,压力事件越多则患病风险越高,这可能与压力对IL-6水平的影响有关^[24]。研究发现,拥有较高共情能力的青少年更易受到人际脆弱性与同伴关系的影响,这也是青春期抑郁障碍的重要心理危险因素之一^[25]。此外,人际关系与家庭关系显著影响大脑皮质下和体感运动网络等脑区的功能连接,因此存在家庭或人际困难的青少年更易罹患抑郁障碍^[26]。

1.6 其他

近年来,肠道菌群紊乱可通过激活免疫反应、影响HPA轴功能、调节脑源性神经营养因子等途径促进抑郁障碍的发生^[27]。青少年抑郁障碍患者事件相关电位与健康对照组相比具有明显差异,可能是青少年抑郁障碍的潜在生物标志物^[28]。此外,人格特征、铁蛋白水平异常及镁缺乏等均是青少年抑郁障碍的预测因素。青少年抑郁障碍的发病机制极为复杂,涉及青春期特殊的生长发育、遗传背景、神经递质失衡以及心理社会因素等多重因素的相互作用,多种机制交织共同推动疾病的发展。

2 青少年抑郁障碍NSSI的独特发病机制

2.1 脑影像学 and 脑功能连接

青少年抑郁障碍NSSI患者在脑影像学方面表现出特异性变化。Guo等^[6]的研究证实青少年抑郁障碍NSSI患者的前额叶皮质功能连接减少,其中右眶额叶皮层与右岛叶之间的连接下降与NSSI发生频率呈负相关。静息态功能磁共振成像提示存在NSSI的患者默认模式网络及基于岛叶的突显网络相关性较低^[29]。青少年抑郁障碍已被证实与相关脑区体积减少有关,而关于青春期NSSI行为的研究同样发现NSSI青少年的灰质体积下降^[30],提示青少年抑郁障碍与青春期的NSSI可能存在相同的神经影像学基础。神经元活动的研究表明,青少年抑郁障碍合并NSSI患者的右侧额上回和右侧舌回的低频波动值与NSSI的频率和严重程度呈正相关^[31]。此外,青少年抑郁障碍患者NSSI行为与脑内包括双侧伏隔核、右壳核及双侧扣带回在内的奖赏通路有关,提示青少年抑郁障碍NSSI可能是一种成瘾模式,为理解青少年抑郁障碍NSSI及其干预策略提供了依据^[32]。脑电图研究表明,抑郁障碍合并NSSI的青少年右半球相对活化程度增加,而左半球激活则与自杀风险呈正相关^[33]。然而,目前青少年抑郁障碍NSSI的大部分研究为横断面设计,未来需追踪随访其大脑发育轨迹,进一步阐明其神经演化规律,为该群体的早识别、早诊断及早期干预提供影像学支持。

2.2 神经递质及激素

青少年NSSI患者相比健康对照者,血浆 β -内啡肽水平显著降低,且疼痛阈值升高^[34],而有研究表明血浆 β -内啡肽与青少年的抑郁症状存在负相关^[35],提示血浆 β -内啡肽水平与青少年抑郁障碍患者NSSI相关。青少年抑郁障碍及NSSI的发生也与下丘脑-垂体-甲状腺轴的改变相关,低甲状腺激素及高睾酮水平是男性青少年抑郁障碍患者发生NSSI的独立危险因素^[36]。另有研究指出,青少年抑郁障碍患者通常会反复出现NSSI,这与该过程可促进内源性阿片肽的释放有关,反复实施NSSI行为不仅能减轻自伤带来的疼痛感,还能增强抑郁障碍患者的愉悦感和欣快感^[37],此外,NSSI的严重程度与HPA轴的反应性增强、自主神经系统交感神经活动减少及副交感神经活动增加相关^[38]。目前已有大量关于抑郁障碍及NSSI与神经递质及激素水平的相关研究,但针对青少年抑郁障碍NSSI研究不足,未来需继续增加针对该特殊人群的研究。

2.3 炎症因子

青春期抑郁障碍合并NSSI患者的免疫功能改变与童年逆境、抑郁症状的严重程度密切相关,而非仅由自伤本身引起的炎性反应。最新研究表明,青少年抑郁障碍合并NSSI患者相比对照组,外周白细胞计数及白细胞/皮质醇比值显著升高,提示存在免疫激活状态。因此,白细胞计数升高可能作为青春期抑郁障碍合并NSSI的免疫激活和慢性社会应激的生物学标志,有助于揭示早期炎症机制对NSSI及其临床共病的作用^[39]。Qiao等^[40]研究发现,合并NSSI的青少年抑郁障碍患者的外周CRP、IL-1、IL-6等促炎因子水平升高,而抗炎因子IL-10降低,并且这些炎症水平与NSSI行为频率呈正相关。我国一项研究则发现,炎症因子中的IL-1 β 和TNF- α 水平升高与青少年抑郁障碍合并NSSI显著相关,其中IL-1 β 的水平与NSSI的相关性最大^[41]。此外,炎症水平升高还与自杀意念和自杀行为存在显著相关性^[42]。因此,炎症细胞因子不仅可作为青少年抑郁障碍合并NSSI的潜在生物标志物,也为制定个体化防治策略提供了理论依据。

2.4 遗传

Wang等^[43]探讨了SIRT1基因甲基化及其表达在青少年抑郁障碍合并NSSI患者中的作用。结果显示,抑郁障碍合并NSSI组的SIRT1基因启动子区CpG5位点甲基化水平显著高于健康对照组,同时其外周血SIRT1蛋白表达明显降低。这些结果提示SIRT1基因甲基化升高与其表达下调可能参与青少年抑郁障碍合并NSSI的发生机制。国内研究发现,携带Val/Val基因型的青少年在遭遇父母冲突并产生抑郁症状后,更易出现NSSI行为,而Met/Met或Val/Met基因型的个体表现出较弱的风险效应^[44]。此外,5-HTR2A基因rs6313多态性对同伴欺凌-抑郁症状-NSSI的关系起到调节作用,CC基因型的个体在面对同伴欺凌时,抑郁症状更容易导致NSSI,而CT基因型和TT基因型的携带者更具抗逆能力,NSSI风险较低^[45]。另有研究表明,OXTR基因rs53576多态性与青少年抑郁障碍合并NSSI之间存在一定的相关性^[46]。基因遗传与青少年抑郁障碍合并NSSI密切相关,未来需开展大样本量的随机对照实验,以进一步阐明基因与青少年抑郁障碍合并NSSI的关系。

2.5 心理社会因素

Nock等^[47]提出的NSSI四功能模型(个体正强化、个体负强化、人际正强化和人际负强化)中,个

体负强化被视为核心机制。青少年NSSI常见的原因包括引起他人关注、控制局势及情绪调节,这在共病抑郁障碍的青少年中更易出现^[48]。经历童年创伤增加了个体低自尊及消极情绪,与患有抑郁障碍的青少年NSSI行为密切相关^[49]。一项关于青少年抑郁障碍的研究发现,青少年抑郁障碍合并NSSI者常经历更多童年虐待、不良同伴关系及低心理弹性^[50]。年龄较小或年级较低的抑郁障碍患者更易出现NSSI行为,且与留守儿童身份及创伤事件密切相关。互联网背景下,抑郁症状是NSSI与网络欺凌间最强中介因素,压力应对和负面情绪反应亦起到重要作用^[51]。不健康的互联网使用增加抑郁障碍的青少年患者NSSI的风险^[52]。国内研究显示,存在焦虑情绪及对母亲的依恋回避是青少年抑郁障碍患者发生NSSI行为的危险因素^[53]。既往研究揭示了青少年NSSI行为与抑郁障碍、童年创伤、不良人际关系、互联网环境等因素的关联,但对各因素的交互作用及长期影响研究不足,未来需进一步研究多种因素交互机制。

2.6 其他

青少年抑郁障碍患者中,女性比男性更容易出现NSSI,这可能是多因素共同作用的结果。存在NSSI行为的女性青少年抑郁障碍患者在面对需要响应人际关系的刺激(如愤怒面孔)时,表现出更显著的注意力偏差^[54]。在抗氧化防御系统中,尿酸水平与青少年女性抑郁障碍患者NSSI的发生呈负相关,这与雌激素对尿酸合成及代谢的影响密切相关^[55]。青少年女性抑郁障碍患者更容易存在述情障碍,而述情障碍已被证实与NSSI的发生与维持有关^[56]。此外,抑郁障碍NSSI行为可能与肠道微生物、饮食及宗教文化等相关,未来需深入探究其相互作用机制,为精准干预提供依据。

3 NSSI与自杀的关系

NSSI与自杀行为之间存在复杂的大脑功能改变,神经网络模型显示,有NSSI行为的青少年更易出现剧烈自杀企图^[57]。此外,NSSI行为的次数与自杀行为的概率呈正相关^[37]。研究指出,纹状体激活与自杀及NSSI相关,且有自杀意念及自杀未遂者的前额叶边缘连接减少^[58]。Li等^[59]的研究发现,NSSI和自杀未遂者的脑功能异常主要体现在边缘网络与感觉注意网络、控制网络、默认模式网络三大核心认知网络间静息态功能连接的变化。脑电图研究证实,自杀组左半球激活增强,而NSSI组以右半

球更为活跃^[33]。自杀行为还与血清色氨酸水平降低、3-羟基犬尿氨酸水平升高相关^[6],并在眶额叶皮层的情绪与认知功能连接上存在不平衡的交互模式^[60]。Kraus等^[61]发现,住院青少年抑郁障碍患者常以NSSI避免自杀行为,NSSI亦被证实为情绪和人际功能失调的一种现实应对手段。人格研究指出,NSSI与自杀的强预测因素之一为人格测评的自杀量表分数,提示二者可能存在相同的人格特质^[62]。此外,压力事件数量增加与NSSI持续存在均会提升自杀风险,冲动性及情绪失调也同样影响自杀风险,并对NSSI行为和压力性生活事件的出现有调控作用^[63]。

虽然研究发现NSSI患者与自杀未遂者在遗传特征上有部分重叠,但目前尚无明确方法揭示其遗传差异的具体性质。青少年NSSI与自杀行为之间有多种共同风险因素,其中以抑郁障碍共病最常见,家庭功能障碍、冲动性、气质类型等也介导NSSI向自杀的演变。长期NSSI可导致个体对疼痛的恐惧减弱、获得习得性自杀能力以及情绪调节受损,从而增加自杀行为发生的风险。然而,现有研究多为横断面设计,NSSI与自杀常在同一受试者中并存,难以明确二者因果关系与具体机制。此外,测量工具多为自我报告,可能存在偏差,不同文化背景下的关系也存在异质性。因此,未来研究需增加多样性,并结合神经影像、生物标志物、心理社会等多维度因素,明确NSSI与自杀的关键转折点,利用生态瞬时评估和人工智能数字追踪等技术,动态捕捉NSSI和自杀的变化过程。青少年NSSI与自杀的研究亟需多学科整合,联合学校及家庭,探索有效干预路径,以期降低青少年NSSI和自杀行为的发生率。

4 针对青少年抑郁障碍NSSI的干预

4.1 心理治疗方面

青少年抑郁障碍合并NSSI的治疗主要以心理治疗为核心,不同心理治疗模式对NSSI的干预效果存在差异。研究显示,认知行为疗法联合药物治疗在减少NSSI行为、减轻抑郁症状和药物不良反应、降低1年内复发率等方面均优于单纯药物治疗^[64]。接受与承诺疗法作为第三波认知行为疗法,强调提升个体对情绪体验的接受与感受能力、认知情绪调节能力及心理灵活性,通过促进有价值的目标行为和减少经验回避,减少自伤行为的发生^[65]。辩证行为疗法同属第三波认知行为疗法,逐步推广用于抑郁障碍、双相障碍、进食障碍及有冲动性攻击行为

的青少年群体,且辩证行为疗法已被证实是降低青少年NSSI及自杀风险的有效干预方案^[66]。人际心理治疗作为结构化心理治疗方法,侧重于增强青少年的人际交往能力,可有效减轻抑郁症状并改善人际关系^[67]。叙事疗法通过倾听患者的自我叙述,挖掘其过往解决问题的积极经验,协助患者构建正向人生故事,进而减少NSSI的发生频率与程度^[68]。基于依恋的家庭治疗强调家庭系统功能修复,通过增强家庭成员间的依恋关系,减轻青少年抑郁症状及NSSI风险^[69]。团体心理治疗作为低成本干预方法,对NSSI也有一定作用,但成员间受益程度差异较大^[70]。简短心理治疗不仅能降低青少年NSSI的当前发生率,还对未来3~4年NSSI的发生有持续影响^[71]。此外,为期12周的正念能减轻青少年的NSSI行为及改善其与父母的关系,且能改善抑郁症状^[72]。目前,青少年抑郁障碍合并NSSI的心理干预方案日益多样化,治疗周期与效果存在一定差异^[65,69,71-77](表1)。

4.2 药物治疗方面

针对青少年抑郁障碍,不同国家的药物治疗指南不尽相同。美国食品药品监督管理局批准选择性血清素再摄取抑制剂(selective serotonin reuptake inhibitor, SSRI)中的氟西汀和艾司西酞普兰用于青少年的短期及维持治疗^[78]。韩国神经精神药理学学会推荐氟西汀、艾司西酞普兰和舍曲林为青少年抑郁障碍的一线药物^[79]。世界生物精神病学联合会制定的单相抑郁障碍生物学指南建议使用氟西汀、舍曲林和文拉法辛^[80]。中国自2021年起,国家药品监督管理局首次批准氟西汀用于8岁及以上儿童和青少年的中重度抑郁障碍药物治疗^[81]。值得注意的是,几乎所有抗抑郁药物均可能增加青少年NSSI及自杀风险,精神科医生在用药前应进行全面评估。美国食品药品监督管理局、英国药品和保健品监管局、欧洲药品局均已发出警告,提示使用抗抑郁药治疗儿童及青少年时,自杀风险可能增加^[82]。目前,一般不推荐对青少年抑郁障碍合并NSSI患者进行单一药物治疗,舍曲林联合心理治疗能减少青少年抑郁障碍患者的NSSI行为^[83]。综上所述,现有关于青少年NSSI药物干预的证据仍较为有限^[84-89](表2)。

几乎所有SSRI都会增加青春期抑郁障碍患者出现NSSI的可能性,但在一项非对照研究中证实氟西汀在降低青少年NSSI的频率方面有一定的作用^[90],抗精神病药物氯氮平、奥氮平、利培酮可能比

表1 不同心理治疗对青少年抑郁障碍NSSI的比较

Table 1 Comparison of Different Psychotherapies for NSSI in Adolescents with Depressive Disorders

Study	Sample	Psychotherapy	Format	Combined with other treatment	Effectiveness
Bockting et al. ^[69]	Attachment-based family therapy group(n=142)	Attachment-based family therapy	16 weeks, 1 session/week	Received all standard treatments Sertraline	Reduced suicidality; more cost-effective; improved family functioning
Liu et al. ^[73]	DBT group(n=50) CBT group(n=50)	DBT	12 weeks: 1 individual session (60 min)/week; 1 family skills training (120 min)/week; 1 brief phone coaching (10 min)/week		Higher NSSI remission rate in DBT group; but depression, anxiety, and social functioning scores lower compared to control
Yuan et al. ^[65]	ACT group(n=36) Control group(n=36)	ACT	9 sessions over 6 weeks, 60 min/session	Received standard psychotherapy	Improved emotion regulation; alleviated depressive mood
Zhang et al. ^[74]	NT group(n=26) Control group(n=29)	NT	3 weeks, 2 sessions/week, 60 min/session	Received standard psychotherapy	Significant reduction in NSSI and depression scale scores
Rockstroh et al. ^[71]	Brief psychotherapy group(n=74)	Brief psychotherapy	10 sessions	No	Improved NSSI; positively associated with long-term outcomes in depression and borderline personality disorder
Van Den Heuvel et al. ^[75]	CBT group(n=282)	CBT	12 sessions, 45-60 min each, 1-2 times/week	No	Reduced depressive symptoms; sequence of CBT modules did not affect outcomes
Katsuki et al. ^[76]	IPT group(n=2)	IPT	12 sessions, 5 times/week; 4 follow-up emails after intervention	No	Reduced depressive symptoms post-intervention; no further self-injury behaviors
Liu et al. ^[72]	Mindfulness group (n=29)	Mindful breathing or loving-kindness meditation	12 weeks, 1.5 h/session; follow-ups at week 6, week 12, and 3 months	No	Effective in reducing NSSI behaviors; lower scores on children's depression inventory
Gratz et al. ^[77]	Group psychotherapy group(n=23)	Group psychotherapy	14 weeks, 90 min/week, 4-6 patients/group	Received standard treatment	Possibly effective in reducing NSSI behaviors

DBT: dialectical behavior therapy; CBT: cognitive behavioral therapy; ACT: acceptance and commitment therapy; NT: narrative therapy; IPT: interpersonal psychotherapy.

其他抗精神病药更能降低自伤自杀风险^[91], 喹硫平、阿立哌唑可能对减轻自杀意念有效^[92]。苯二氮卓类药物短期可能使NSSI行为减少, 长期使用会使冲动行为脱抑制而导致NSSI发生率增加, 此外共病注意力缺陷与多动障碍的青少年抑郁障碍患者服用哌醋甲酯、托莫西汀、安非他明治疗会增加NSSI^[93]。目前关于青少年精神药物使用的研究取得一定进展, 但仍存在样本量小、研究范围不全面等问题, 未来需继续深入探索青少年使用精神药物减少抑郁情绪及自伤自杀行为的相关研究。

4.3 物理治疗

非侵入性治疗在青少年抑郁障碍及NSSI的干预中显示出一定疗效。部分研究证实, 电休克治疗

可显著降低青少年抑郁障碍患者的IL-1 β 、IL-6水平并提高IL-10水平, 减少自伤自杀意念及自杀行为, 且其治疗效应大于不良反应^[94], 目前关于电休克治疗在儿童青少年中的应用仍存在较大争议, 或许与电休克治疗可能导致记忆障碍相关。重复经颅磁刺激是目前治疗青少年重度抑郁障碍的有效非侵入性方法之一。研究表明, 重复经颅磁刺激不仅可以改善首次发作青少年抑郁障碍患者的抑郁症状及认知功能^[95], 降低NSSI行为的发生频率, 还可与药物联用增加疗效, 对青少年患者均具有较高的安全性^[96]。经皮耳廓迷走神经刺激作为新兴神经调节疗法, 能通过调控海马体、前扣带皮层和内侧前额叶皮层的基因表达, 改善青少年的抑郁症状^[97]。

表2 关于NSSI和自杀的药物治疗效果比较
Table 2 Comparison of pharmacotherapy effects on NSSI and suicide

Study	Sample size(<i>n</i>)	Treatment duration	Effectiveness	Study type
Zhou et al. ^[84]	MDD with suicidal ideation Esketamine group(<i>n</i> =27) Midazolam group(<i>n</i> =27)	4 weeks	Esketamine: anti-suicidal and antidepressant response rates=69.2% and 61.5%, Midazolam: 52.5% and 52.5%	RCT
Dervic et al. ^[85]	Mood disorder with suicide attempt Lithium group(<i>n</i> =94) Valproate group(<i>n</i> =94)	2.5 years	Lithium group had fewer suicide attempts compared to valproate group	RCT
Yovell et al. ^[86]	Suicidal ideation buprenorphine group(<i>n</i> =57) Non-buprenorphine group(<i>n</i> =31)	4 weeks	Very low - dose buprenorphine reduced suicidal ideation	RCT
Madse et al. ^[87]	Moderate-to-severe MDD Escitalopram group(<i>n</i> =450) Nortriptyline group(<i>n</i> =346)	12 weeks	Suicidal ideation reduced in both groups. In males, escitalopram reduced suicide risk more than nortriptyline, which increased suicidal ideation	Retrospective study
Canuso et al. ^[88]	MDD with suicidal ideation Intranasal esketamine group(<i>n</i> =36) Traditional antidepressants group(<i>n</i> =32)	4 weeks	Esketamine showed stronger effects in improving depressive symptoms and suicidal ideation within 4-24 hours compared to control	RCT
Shamseddeen et al. ^[89]	MDD Trazodone group(<i>n</i> =33) Control group(<i>n</i> =276)	12 weeks	Trazodone increased risk of NSSI	RCT

NSSI: nonsuicidal self-injury; MDD: major depressive disorder.

此外,经颅直流电刺激和光照疗法耐受性好、不良反应小,在成人抑郁障碍治疗中临床证据丰富,尽管目前针对青少年的研究有限,但前景良好^[98]。除上述神经调控治疗外,针灸、运动疗法及冲浪疗法等非药物干预也对青少年抑郁障碍及NSSI具有积极作用。

5 小结与展望

本综述系统梳理了青少年抑郁障碍NSSI的流行病学特征、神经生物学机制、遗传学风险因素与心理社会环境的交互作用,着重强调了神经递质失衡、炎症反应、神经激素变化以及SIRT1、5-HTR2A等基因多态性在NSSI发生发展过程中的作用,而童年创伤、家庭功能障碍、同伴欺凌等社会环境压力通过影响青少年的情绪调节和抑郁水平,进一步加剧了NSSI的风险。多项研究也揭示,遗传易感性不仅影响个体对外部压力的反应,还在一定程度上决定了干预措施的效果和预后。现有的心理治疗、药物治疗及新型物理干预手段在改善情绪症状和降低NSSI行为方面虽显示出一定疗效,但依从性和远

期复发率问题尚未有效解决,特别是在识别和干预高危人群、实现个体化干预方面仍有明显不足。未来,应深入开展多组学整合、纵向大样本追踪及神经影像结合分子遗传机制研究,明确影响NSSI及自杀风险的关键生物标志物和心理社会风险模型,推动精准分层和动态风险评估体系的建立。临床实践中应积极推进生物-心理-社会一体化治疗路径,结合遗传分型、发病机制和环境应激源有针对性地制定干预策略,实现早筛查、早干预、持续跟踪与复发预防。同时,需加强家庭、学校与社会三方协作,通过心理健康教育、能力训练和支持性资源建设提升青少年的情绪调节、社会适应和问题应对能力,减少NSSI及其相关不良结局。通过多学科协作和持续创新,进一步提升青少年抑郁障碍合并NSSI患者的整体预后和社会功能,为精神健康防治工作提供更加科学、系统和可持续的发展路径。

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