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瘦型非酒精性脂肪性肝病的发病机制研究进展

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摘　要

瘦型非酒精性脂肪肝病(Lean Non-Alcoholic Fatty Liver Disease, lean NAFLD)是指在体重指数(Body Mass Index, BMI)正常或偏低人群中发生的非酒精性脂肪肝病(NAFLD)。尽管传统观点认为NAFLD主要与肥胖相关，但近年来发现，并非所有NAFLD患者都伴随体重超标，这一部分患者日益受到关注。而lean NAFLD的发病机制复杂，呈现出代谢－免疫－遗传多维度交互特征，涉及脂质代谢异常、胰岛素抵抗、肠道微生物群失调、遗传易感性及炎症反应等多方面因素。本文旨在综述当前关于lean NAFLD发病机制的研究进展，分析其与肥胖相关NAFLD (obese NAFLD)的异同，并探讨未来研究方向。

关键词

非酒精性脂肪性肝病，发病机制，瘦型

Research Progress on the Pathogenesis of Lean Non-Alcoholic Fatty Liver Disease

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Abstract

Lean Non-alcoholic Fatty Liver Disease (lean NAFLD) refers to non-alcoholic fatty liver disease (NAFLD) that occurs in individuals with normal or low body mass index (BMI). Although traditional views suggest that NAFLD is mainly associated with obesity, it has been found in recent years that not all NAFLD patients are accompanied by overweight, and this group of patients is receiving increasing attention. The pathogenesis of lean NAFLD is complex, exhibiting multidimensional interactions of metabolism, immunity, and genetics, involving various factors such as abnormal lipid metabolism, insulin resistance, dysbiosis of gut microbiota, genetic susceptibility, and inflammatory response. This article

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aims to review the current research progress on the pathogenesis of lean NAFLD, analyze its similarities and differences with obesity related NAFLD, and explore future research directions.

Keywords

Non-Alcoholic Fatty Liver Disease, Pathogenesis, Lean

1. 引言

非酒精性脂肪肝病(Non-Alcoholic Fatty Liver Disease, NAFLD) [1]是全球最常见的慢性肝病之一，其患病率随着肥胖和代谢综合征的流行而显著上升。据报道全球在 1990 年至 2019 年之间，NAFLD 的发生率增加了 95.4%，由 88,177 例增加到 172,330 例[2]。目前全球 NAFLD 的总患病率约为 32.4%，且患病率逐年增加，其中男性的 NAFLD 的总体患病率高于女性(39.7% vs 25.6%) [3] [4]。超重与肥胖群体中 NAFLD、非酒精性脂肪肝(non-alcoholic fatty liver, NAFL)、非酒精性脂肪性肝炎(non-alcoholic steatohepatitis, NASH)、显著纤维化(≥F2)、进展期纤维化(≥F3)的总体患病率相似(分别为 69.99% vs 75.27%, 42.49% vs 43.05, 33.50% vs 33.67%, 20.27% vs 21.60%, 6.65% vs 6.85%) [5]。然而，与传统认为 NAFLD 主要与肥胖相关的观点不同的是，研究中逐渐发现有部分 NAFLD 患者为 BMI 正常甚至偏低，在全球 NAFLD 中约 40%的患者被归类为非肥胖人群，约 19.8%为低体重指数患者[6]。因此，lean NAFLD 患者的比例并不低，应得到重视，有必要进一步了解此类患者的发病机制以加强对此类疾病的了解。

2. Lean NAFLD 的定义与临床意义

Lean NAFLD 的定义目前尚存争议，但通常指 BMI < 25 kg/m²或< 23 kg/m² (亚洲人群)且无过量饮酒的患者中出现肝脏脂肪变性[7] [8]。根据影像学(如超声或 MRI)或组织学(肝活检)诊断的 NAFLD 患者中，约 10%~20%属于 lean NAFLD。尽管这些患者体重正常，但其与正常人群相比的话，胰岛素抵抗、代谢合并症、内脏脂肪沉积的比例会更高，并且肌肉量会减少[9] [10]。这些现象提示 NAFLD 的发病机制可能并非单纯由体重决定，而是涉及更复杂的代谢和病理生理过程。且部分 lean NAFLD 患者可能进展为 NASH 或肝纤维化，在非肥胖或 lean NAFLD 患者中，29.2%患有显著纤维化(≥F2 期)，3.2%患有肝硬化[6]，提示其疾病进程与肥胖相关 NAFLD 相似，但进展速度可能更快。因此，深入研究 lean NAFLD 的发病机制不仅有助于完善 NAFLD 的分类和诊断标准，还能为非肥胖人群的 NAFLD 预防和治疗提供新思路。

3. Lean NAFLD 的发病机制研究

3.1. 代谢重构假说

1、肝脏脂质动态失衡：肝脏是脂质代谢的核心器官，其脂肪积累与脂质合成(如脂肪酸从头合成)和分解(如 β-氧化)的失衡密切相关。在 lean NAFLD 中，肝脏中乙酰辅酶 A 羧化酶和脂肪酸合酶的活性可能升高[11]，导致脂肪酸过度合成。其次，尽管体重正常，部分 lean NAFLD 患者可能因线粒体功能异常或过氧化物酶体增殖物激活受体 α 活性降低，导致脂肪酸 β-氧化能力下降，从而促进脂质堆积[12] [13]。

2、胰岛素抵抗：obese NAFLD 患者通常伴有明显的全身性肥胖，而肥胖是导致胰岛素抵抗(Insulin Resistance, IR)的主要因素之一，IR 会导致肝脏脂肪堆积和炎症反应，进而促进 NAFLD 的发展，因此全身性肥

胖驱动的 IR 在 obese NAFLD 中占主导地位。但有研究表明，IR 似乎也是 lean NAFLD 的潜在因素，即使体重正常，lean NAFLD 患者可能因肌肉或脂肪组织的胰岛素信号通路异常而出现胰岛素抵抗，且瘦人与普通人相比 IR 更常见[14] [15]。此类患者肝内甘油三酯含量升高与骨骼肌、肝细胞和脂肪组织 IR 升高有关，与体重指数无关。此外，有研究发现高加索 lean NAFLD 人群中的糖耐量受损比例与肥胖患者相似[16]，这也从侧面证实了这一观点。肝脏对胰岛素的敏感性降低，导致糖异生增加和脂肪合成增强，进而促进脂肪变性。

3、脂肪组织释放的游离脂肪酸增多：尽管 lean NAFLD 患者体脂率较低，但瘦型人群的脂肪细胞可能因遗传或环境因素(如饮食、压力)发生功能紊乱，表现脂肪储存能力下降和游离脂肪酸(Free Fatty Acids, FAAs)释放增加，导致 FAAs 循环浓度升高，从而促进肝脏的脂肪变性。研究表明，在肝内脂肪变性和 IR 的病例中，脂肪酸转位酶 CD36 (一种控制血浆中游离脂肪酸摄取的蛋白质)在骨骼肌和肝脏组织中增强，在脂肪组织中减弱[17]。另有研究发现与健康对照组相比，lean NAFLD 受试者的肝内 FAAs 流入增加，门静脉 FAAs 流量增加[18]，这些发现也进一步证实了这一观点。

3.2. 遗传－表观调控网络

1、基因多态性：Lean NAFLD 患者通常不伴有明显的肥胖，但其肝脏脂肪堆积和炎症反应却较为显著，但根据现有研究表明这类患者可能具有更高的遗传易感性。目前已知某些基因变异(如 PNPLA3、TM6SF2、MBOAT7、LIPA、SAMM50)与 NAFLD 的易感性相关，且在瘦型人群中可能表现出更强的效应[19]-[22]。根据这些研究可以发现这些等位基因的变化会影响脂质代谢，降低相关酶的活性，进而促进肝脂肪变的发生，且基因的改变会对患者肝纤维化的进展以及长远预后造成影响。

2、表观遗传调控及家族聚集性：DNA 甲基化、组蛋白修饰和非编码 RNA 可能通过调控脂质代谢相关基因的表达，影响肝脏脂肪变性。如载脂蛋白 B (Apolipoprotein B, ApoB)是脂蛋白中的关键成分，而脂蛋白又与脂肪代谢息息相关。载脂蛋白突变可导致脂肪吸收不良、脂溶性维生素缺乏和肝脏脂蛋白分泌减少，肝脏甘油三酯积累导致脂肪变性和纤维化[23]。家族性低脂蛋白血症为一种常染色体不完全显性遗传病，其特征是血浆低密度脂蛋白胆固醇(low-density lipoprotein-cholesterol, LDL-C)和 ApoB 降低。由于 ApoB 的减少，他们可以表现出肝脂肪变性和肝肿大[24]。

这些研究进一步提示了遗传因素在 lean NAFLD 发病机制中的重要作用。遗传易感性对 lean NAFLD 的影响呈现多维度、多层次的特征，基因多态性和表观遗传标记的发现一方面为深入理解 lean NAFLD 的分子机制提供了依据，也为开发未来个体化诊疗指明了方向。

3.3. 肠－肝轴异常、肠屏障功能障碍

肠－肝轴用于描述肠道微生物群组成和功能背景下肠道上皮、血管和免疫屏障与肝脏循环之间复杂的相互作用。肠道生态失调的特征是肠道微生物群的组成和多样性的改变，目前大多数研究表明 NAFLD 患者革兰氏阴性拟杆菌门的丰度增加，导致厚壁菌门与拟杆菌门的比值降低[20] [25]。而在 lean NAFLD 中同样可以观察到肠菌群的总体丰富度和多样性降低，有益菌的减少[26] [27]，肠屏障功能的受损，从而进一步促进细胞因子的分泌，肠道通透性增加可能使细菌内毒素进入门静脉，激活肝脏的炎症反应，促进脂肪变性和纤维化，参与 lean NAFLD 的发病。且也有研究表明瘦型 NASH 患者拟杆菌门/厚壁菌门比值降低[28]。这也进一步说明肠道菌群结构的改变进一步促进 lean NAFLD 的发生与发展，与此同时也为该病的治疗提供了新思路，通过对肠道菌群的干预(补充益生菌或特定菌株的粪菌移植)对 lean NAFLD 患者的治疗作用，但目前仍有待于进一步研究与验证。

3.4. 炎症与氧化应激

慢性炎症和氧化应激是 NAFLD 进展的关键因素[29]，尤其是在 obese NAFLD 患者中更为明显，肥

胖状态下，脂肪组织释放的炎症因子(如 TNF-α、IL-6)和氧化应激产物会进一步加剧肝脏的炎症反应和纤维化进程。但在 lean NAFLD 中可能具有一些独特表现，此前有研究发现非肥胖 NAFLD 患者和健康对照者的瘦素、TNF-α 和 IL-6 水平无显著差异[16]。此外，Huang [30]等人发现巨噬细胞固醇调节元素结合蛋白裂解激活蛋白(Sterol regulatory element binding protein cleavage-activating protein, SCAP)可以通过激活 STING-NF-κB 信号传导途径来充当巨噬细胞炎症反应的新调节剂和 lean NAFLD 的发病机理。因此，lean NAFLD 患者的炎症与氧化应激水平可能较低，目前该机制并未在 lean NAFLD 患者中完全阐明，有待于进一步通过 lean NAFLD 动物模型研究炎症微环境对 lean NAFLD 的影响。

3.5. 其他可能的机制

有研究发现血清瘦素水平与 NAFLD 的严重程度有关，瘦素水平越高，纤维化程度越重，尤其是在肥胖型 NAFLD 中更为显著[31]，因此血清瘦素水平的高低是否与 lean NAFLD 的严重程度相关仍需要进一步进行验证。Lee 等人[32]的研究指出 NAFLD 的严重程度与亚洲人群中的内脏脂肪积累呈正相关，内脏肥胖可能是潜在 lean NAFLD 的危险因素，可以通过进一步研究来证实。此外，过量饮用含糖饮料，尤其是含有高果糖玉米糖浆的，有罹患 NAFLD 的可能[33]。一项针对非肥胖型 NAFLD 病例(平均年龄 30 ± 13 岁，平均 BMI 25.6 ± 2.6，无高血压、高脂血症或 2 型糖尿病等潜在危险因素)的研究支持了这一结论，该研究显示，共 31 例非肥胖型 NAFLD 患者，其中 80%的患者每天摄入超过 50 克的添加糖，而健康对照组为 20% [34]。

4. 总结与展望

Lean NAFLD 的发病机制研究近年来取得显著进展，揭示了代谢紊乱、遗传易感性、肠肝轴失调等多维度的复杂网络。尽管研究已取得重要突破，仍存在诸多未解问题。例如，基因–环境互作机制的系统性解析仍需深化，尤其是各种因素中何种因素为主导，不同种族、性别及代谢表型的异质性；多组学整合分析(如基因组、表观组、代谢组)对疾病分型和预后预测的价值尚待验证；此外，动物模型与临床转化之间的差距也限制了治疗靶点的开发；与此同时，急需建立全球统一的 lean NAFLD 诊断标准。

未来研究仍需聚焦以下方向：精准医学：构建基于上述基因组学、转录组学、蛋白质组学及代谢组学等多组学数据的个体化风险评估模型以及整合高通量测序、生物信息学和人工智能技术，构建涵盖基因突变、表观遗传修饰及分子标志物等方面的精准诊断体系的完善，以提高 lean NAFLD 预测的准确性和早期干预的针对性。新型治疗策略：针对 lean NAFLD 相关特定基因变异或关键表观遗传通路(如 PNPLA3、TM6SF2、MBOAT7 等)，开展靶向药物的研发，旨在精准抑制疾病进展。以及基于个体化能量消耗特征，设计个性化运动处方，结合肠道微生态调节策略，如补充益生菌或粪菌移植等干预手段，共同促进肝脏脂肪含量的有效下降。长期随访研究：通过长期随访队列研究，深入探究 lean NAFLD 的肝纤维化、肝癌等终点事件的病程进展(如各个阶段的临床特征、生化指标及影像学变化)、严重程度(如肝纤维化评分、Child-Pugh 评分等)及治疗策略(包括药物治疗、生活方式干预、手术等方法的反应性和疗效)与 obese NAFLD 是否一致，从而探讨 NAFLD 的不同亚型在肝纤维化、肝癌等终点事件的发生率、生存率及预后情况，揭示不同亚型在长期预后上的异同。随着技术进步和跨学科合作的深化，lean NAFLD 的机制研究有望为临床诊疗提供更精准的理论支持和干预手段。

利益冲突

本文章不存在利益冲突，特此声明。

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