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# II 型糖尿病并发抑郁症与 NLRP3 炎症小体关系的研究进展

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[摘 要] II型糖尿病并发抑郁症是自杀率最高的慢性并发症之一。NLRP3炎症小体是先天免疫系统中的胞内传感器，能够介导下游caspase-1及IL-1β等炎症因子成熟与分泌。研究发现NLRP3炎症小体在II型糖尿病患者中持续激活，同时NLRP3炎症小体的激活在抑郁症发病中扮演重要角色，因此推测NLRP3炎症小体的激活可能介导了II型糖尿病并发抑郁症。

[关键词] II型糖尿病；抑郁症；NLRP3炎症小体；IL-1β

# Research progress in type 2 diabetes with depression and NLRP3 inflammasome

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**Abstract** Type 2 diabetes mellitus with depression is an important cause of suicide in patients. NLRP3 inflammasome is an intracellular sensor in the innate immune system which can mediate the maturation and secretion of caspase-1/IL-1β and other inflammatory factors. Recent studies have found that NLRP3 inflammasomes are continuously activated in patients with type 2 diabetes, and the activation of NLRP3 inflammasome plays an important role in the pathogenesis of depression, so we speculate that activation of the NLRP3 inflammasome may mediate the process of type 2 diabetes with depression.

**Keywords** type 2 diabetes; depression; NLRP3 inflammasome; IL-1β

近年来，随着 II 型糖尿病患病率逐年上升及对糖尿病的研究逐步深入，糖尿病并发抑郁症发病率显著高于正常人群。研究[1]表明：II 型糖尿病并发抑郁症与 NLRP3 炎症小体密切相关。本文主要就 II 型糖尿病并发抑郁症与 NLRP3 炎症小体关系作一综述，旨在加强对 II 型糖尿病并发抑郁

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症患者的治疗。

## 1 NLRP3 炎症小体

Nod 样受体蛋白 3(NOD-like receptor protein 3，NLRP3) 炎症小体是核苷酸结合寡聚化结构域样受体 (NOD-like receptor，NLR) 家族中最具特色的成员之一。NLRP3 能够募集凋亡相关斑点样蛋白 (apoptosis-associated speck-like protein containing CARD，ASC) 和半胱氨酸天冬氨酸蛋白酶 1(cysteine-requiring aspartate protease-1，caspase-1) 组合成蛋白复合体，即炎症小体 [2]。作为先天免疫系统中的胞内传感器，NLRP3 炎症小体是联系免疫系统和代谢信号的重要因子。NLRP3 能够感受代谢应激从而被上调和激活，其激活过程主要关联肿瘤坏死因子、活性氧等许多胞内胞外信号分子的刺激。正常激活 NLRP3，能够检测内源性危险信号，调节免疫系统；而过度激活 NLRP3，会促进 caspase-1 成熟、自我剪切并释放到胞外，而成熟的 caspase-1 能够进一步激活 IL-1β 等炎症因子的成熟与分泌 [3-5]。大量研究 [6] 表明：NLRP3 炎症小体过度激活导致 IL-1 活化可能是阿尔兹海默病、糖尿病、抑郁症等多种炎症疾病的共同致病因素。与其他炎症体不同，NLRP3 是联系炎症反应和代谢性疾病的核心，能被代谢信号激活并进一步调节各种危险信号分子 IL-1β 和 caspase-1 的激活及释放。因此，NLRP3 对研究糖尿病等代谢性疾病的治疗新靶点具有重要意义。

## 2 NLRP3 炎症小体与 II 型糖尿病的关系

II 型糖尿病是一种复杂的糖代谢性疾病，其发病机制主要为胰岛 β 细胞受损和胰岛素抵抗。此外，病程中炎症物质的增加及免疫系统的激活也被视为发生 II 型糖尿病的重要原因 [7-10]。研究 [11] 显示：高糖和高浓度脂肪酸皆可刺激胰岛细胞激活 NLRP3 炎症小体，高血糖症和高胰岛素血症皆表现出 IL-1β，IL-18，TNF-α，caspase-1，C 反应蛋白及皮质醇等促炎标志物增加。硫氧还蛋白结合蛋白 (thioredoxin interacting protein，TXNIP) 是糖尿病和高血糖症的早期应答基因，随着血糖的升高，TXNIP 表达显著上调。糖代谢紊乱及氧化应激诱导的炎症级联反应可直接或通过增多的 TXNIP 等介质间接激活 NLRP3 炎症小体 [12-13]。激活的 NLRP3 可活化及释放 IL-1β，过多的 IL-1β 则会诱导损伤胰岛 β 细胞，加剧 II 型糖尿病病程并引发一系列的并发症 [14]。

## 3 NLRP3 炎症小体与抑郁症的关系

抑郁症是一种严重危害人类身心健康的精神疾病，该病的发生发展是遗传、环境、神经内分泌改变等共同作用的结果。近年来，抑郁症与免疫炎症的密切关系成为抑郁症具体机制的研究热点 [15]。研究 [16] 表明：抑郁症病程常伴随 NLRP3 及 IL-1β 等促炎症物质的改变。NLRP3 炎症小体能够激活 caspase-1，IL-1β 和 IL-18 前体释放到胞外并透过血脑屏障进入脑内。炎症介质进入大脑后，会影响谷氨酸释放、小胶质细胞和星形胶质细胞摄取、犬尿氨酸途径神经活性代谢物产生等多种神经传递机制，增加神经兴奋性，从而导致水肿、抑郁症等神经炎症 [17-19]。动物实验 [20-21] 证明：过度激活 NLRP3 导致 IL-1β 成熟与大量分泌，而 IL-1β 过剩会引起一系列抑郁症状，如抑制运动活动、抑制社会、快感缺失等；而当抑制 NLRP3 时能够有效减少小鼠的条件反射恐惧、习得性无助行为及焦虑行为，表明 NLRP3 可能参与小鼠抑郁发生发展的过程。临床研究 [22-23] 表明：抑郁症患者脑组织和血清中 NLRP3 炎症小体、IL-1β 和 IL-18 表达皆显著高于正常人。综上，NLRP3 可能在抑郁症发病机制中起重要作用。

## 4 NLRP3 炎症小体与 II 型糖尿病并发抑郁症的关系

II 型糖尿病是一种与行为、精神障碍密切相关的慢性疾病，与抑郁症有非常高的共病性。临床数据 [24-26] 表明：II 型糖尿病的发生发展往往伴随抑郁症等心理并发症；约 42% 的糖尿病患者有不同程度的抑郁症症状。Khan 等 [27] 报道：II 型糖尿病患者患抑郁症的发生率是普通人的 2.8 倍，复发率是非 II 型糖尿病患者的 8 倍；且 II 型糖尿病并发抑郁症的严重程度，并发症的发生率及病死率皆显著高于单纯 II 型糖尿病或抑郁症发生的概率。

最新研究 [28-29] 表明：II 型糖尿病可能通过异常调节下丘脑 - 垂体 - 肾上腺轴 (HPA) 轴及过度激活先天免疫系统，引发慢性细胞因子介导的炎症反应，从而并发抑郁症。II 型糖尿病激活的先天免疫系统与交感神经系统的下丘脑 - 垂体 - 肾上腺轴和蓝斑 - 去甲肾上腺素等内分泌系统结合时，炎症部位的巨噬细胞释放的细胞因子作用于大脑会进一步

加剧大脑炎症反应；促炎细胞因子可刺激下丘脑 - 垂体 - 肾上腺轴释放糖皮质激素，从而产生或进一步导致神经炎症。同时，机体慢性炎症反应诱导的皮质类固醇显著上升可导致海马损伤。海马神经元在情绪和行为调控中发挥重要作用，其损伤可能是导致动物行为和情绪障碍的重要原因[30]。研究[31-33]表明：在慢性应激诱导的抑郁小鼠和脂多糖诱导的抑郁小鼠的脑内皆检测到胰岛素信号转导障碍和 NLRP3 炎症小体的激活。Su 等[34]发现：轻度应激小鼠模型中，抑郁样行为与胰岛素分泌异常同时发生，推测这种并发症可能是由 NLRP3 炎症小体的激活诱导下丘脑 - 垂体 - 肾上腺轴失调引发。急性束缚应激后小鼠海马中的 NLRP3 水平显著升高，基因敲除 NLRP3 或使用抗糖尿病药物，如格列本脲，能够有效抑制 NLRP3 诱导 IL-1β 水平升高及小鼠的抑郁症状[35]。提示抑制 II 型糖尿病患者 NLRP3 炎症小体的激活，或许能够减少海马及神经损伤，从而缓解糖尿病患者的抑郁症。NLRP3 可能介导代谢失调导致的应激反应，参与 II 型糖尿病并发抑郁症的炎症过程[36]。

## 5 NLRP3 炎症小体对于 II 型糖尿病并发抑郁症的潜在治疗作用

目前，临床上常使用降糖药或抗抑郁类药物治疗 II 型糖尿病并发抑郁症。而降糖药可能导致患者血糖控制不良或低血糖[37]。另外，抗抑郁类药的使用与 II 型糖尿病患病率有密切联系，抗抑郁药在短期内可以改善血糖，但长期使用抗抑郁药会使 II 型糖尿病患病率提高两倍[38]。因此，探索 II 型糖尿病并发抑郁症的新型药物具有重要临床意义。

NLRP3炎症小体是诱导II型糖尿病并发抑郁症的关键因子。抑制NLRP3能有效逆转海马区IL-1β和caspase-1的活化，并改善II型糖尿病小鼠焦虑等抑郁样行为和认知功能障碍[39-40]，提示NLRP3抑制类药物可能成为II型糖尿病并发抑郁症的临床治疗手段。Liu等[41]研究表明：黄芩苷通过抑制大鼠前额叶皮质中NLRP3炎症小体的激活，能显著逆转大鼠抑郁样行为。体内和体外实验[42]皆证实玉米黄质具有抗炎作用，且已经应用于多种炎症引起的疾病模型。还有研究[43]表明：玉米黄质可以通过激活AKT通路降低葡萄糖水平，缓解II型糖尿病并发症及改善II型糖尿病动物的认知缺陷。此外，补充玉米黄质可以通过抑制炎症、氧化应激改善焦虑和抑郁导致的细胞凋亡[24]。5-羟色胺(5-hydroxytryptamine，5-HT)是脑内关键的神经递质之一，可以调节大脑的行为和情绪活动。临床研究[44]发现：使用5-HT再摄取抑制剂类药物能够明显改善II型糖尿病并发抑郁症和精神障碍患者脑中5-HT显著降低症状。该类药物能有效抑制NLRP3的活性、降低血糖、增加对胰岛素的敏感性、控制抑郁情绪并改善认知，且该药物的长期使用不会引起心律失常、尿潴留等不良影响[45]。临床上常用氟西汀、帕罗西汀等选择性5-羟色胺再摄取抑制剂(selective serotonin reuptake inhibitors，SSRIs)来治疗II型糖尿病并发抑郁症[46]。氟西汀是一种选择性5-HT再摄取抑制剂，能够阻断5-HT的再摄取，增加和延长5-HT的作用，从而具有降血糖和胰岛素增敏的作用。动物实验[47-49]表明：氟西汀能够恢复下丘脑室旁核中促肾上腺皮质激素释放激素水平，上调动物海马齿状回区域的神经性营养因子表达，从而改善抑郁症状。同时，氟西汀能够通过下调活性氧-蛋白激酶-NLRP3信号通路，抑制慢性应激小鼠海马中的NLRP3炎症小体的激活，缓解慢性应激诱发的抑郁样行为[45]。提示氟西汀在NLRP3炎症小体诱导的抑郁症等炎症性疾病的临床治疗中可能具有一定的作用。多项临床试验[50-51]表明：经氟西汀治疗的II型糖尿病并发抑郁症患者，其抑郁症状比安慰剂组显著降低，并且该药物对控制血糖没有不利影响。

近年来，II型糖尿病并发抑郁症患病率不断上升，严重危害人类身体健康。NLRP3炎症小体作为固有免疫的重要部分，与II型糖尿病并发抑郁症的发生发展密切相关。以NLRP3炎症小体作为药物治疗靶点，或许成为II型糖尿病并发抑郁症的有效治疗方法。

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