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烟酸调节动物肠道黏膜屏障功能的分子机制

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摘　要: 作为一种重要的维生素,烟酸在抑制肠道炎症反应及维持肠道健康等方面发挥重要作用。研究表明,烟酸可以直接结合烟酸受体 G 蛋白偶联受体 109A(GPR109A)发挥其生理作用,还能够以辅酶烟酰胺腺嘌呤二核苷酸(NAD^+)的形式参与细胞能量代谢。本文主要阐述了烟酸维护肠道黏膜屏障功能的作用机制,为维护断奶仔猪肠道健康提供科学理论依据。

关键词: 烟酸;G 蛋白偶联受体;烟酰胺腺嘌呤二核苷酸;肠道黏膜屏障;能量代谢

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肠道上皮是隔离外界环境的第 1 道防线,大量肠道微生物定植于肠道上皮细胞的黏液外层,以便于促进消化吸收和调节黏膜免疫系统的发育与功能。断奶应激会破坏仔猪肠道微生态稳态,导致肠道屏障功能受损,从而引起仔猪腹泻和生长停滞。饲料添加剂抗生素在预防断奶仔猪腹泻和促进仔猪生长等方面发挥重要的作用,然而长期不规范使用抗生素导致药物残留及抗生素耐药等负面问题日益凸显。随着全面禁止在猪饲料中添加抗生素政策的落实,开发有益的饲用抗生素替代品是畜牧业亟待解决的问题。烟酸是一种重要的 B 族维生素成员,主要作为饲料添加剂。近年来的研究表明,饲粮中添加烟酸有助于提高断奶仔猪的生产性能及减少仔猪腹泻[1-3]。此外,已有的研究发现显示,烟酸还能够抑制炎症反应及调节肠道健康[4]。因此,借鉴烟酸在人体疾病及小鼠等动物模型中的应用,本文阐述了烟酸调节肠道黏膜屏障功能的分子机制,为其在断奶仔猪生产中更广泛、更好地应用提供科学的理论依据。

1　烟酸及其代谢产物

1.1　烟酸的研究进展

烟酸也称为维生素 B_3,是一种水溶性维生素,主要通过胃肠道黏膜吸收。除了体外添加烟酸,动物体内还可通过犬尿氨酸途径将多余的色氨酸转化为烟酸[5]。烟酸是细胞代谢辅酶烟酰胺腺嘌呤二核苷酸(NAD^+)和烟酰胺腺嘌呤二核酸磷($NADP^+$)的前体,它在机体内以 NAD^+或 $NADP^+$形式参与脂质代谢、呼吸氧化反应和糖类无氧分解等细胞过程,并在细胞能量代谢方面发挥重要作用。近年来,烟酸在调控炎症反应、调节肠道稳态及维持肠道健康状态等方面受到国内外的广泛关注。在人类疾病研究过程中发现添加烟酸可缓解单核细胞介导的炎症反应[6-7],并降低 M1 型巨噬细胞的数量[8]。烟酸还可通过下调核转录因子-κB(NF-κB)的信号,缓解败血症引起的肺部炎症反应和提高患者的生存率[9]。Feng 等[10]研究发现,添加烟酸可下调肿瘤坏死因子-α(*TNF*-α)、白细胞介素-8(*IL*-8)、γ-干扰素(*IFN*-γ)及白细

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胞介素-1β(*IL*-1*β*)等促炎因子的表达量及增强草鱼肠道黏膜屏障功能的完整性。此外,Salem等[11]的研究表明,烟酸降低葡聚糖硫酸钠(DSS)诱导结肠炎小鼠的炎症因子髓过氧化物酶(*MPO*)和*TNF*-α的表达及改善病态的血管生成,从而实现改善结肠炎疾病。

1.2 烟酸在动物体内的代谢产物

烟酸具有多重的生物活性,在动物体内转化为辅酶 NAD^+,NAD^+ 随后可被磷酸化形成 $NADP^+$[12]。烟酸在动物体内主要以 NAD^+ 形式参与机体代谢,其在生物氧化过程中以氢和电子的传递体参与糖类、脂肪和蛋白质的代谢转化及能量释放。除此之外,NAD^+ 还作为去乙酰化酶、核糖聚合酶(PAR)和 CD38 等反应酶的底物。沉默信息调节因子 2 相关酶 1(SIRTl)是一种 NAD^+ 依赖的组蛋白去乙酰化酶,它在细胞分化、细胞代谢、细胞凋亡、DNA 修复及抑制炎症等过程起到十分重要的作用[13]。NAD^+ 调节 SIRT1 介导的去乙酰化作用,而蛋白质去乙酰化有助于转录因子和代谢酶调节线粒体氧化功能和增强细胞在应激条件下的存活率[14-15]。饲粮中添加烟酸,可增加 NAD^+ 的浓度,有利于改善应激条件下线粒体的功能及预防食物或年龄等因素引起的代谢疾病[16-18]。

2 烟酸调节肠道黏膜屏障功能的分子机制

2.1 烟酸直接结合 G 蛋白偶联受体 109A(GPR109A)调节肠道健康

GPR109A 是介导烟酸、丁酸和羟基丁酸等物质生物学效应的重要受体[19],其广泛表达于淋巴细胞、脂肪细胞、单核细胞、巨噬细胞、树突状细胞及肠道上皮细胞[20]。近年来的研究表明,GPR109A 与配体结合后,能够调节微生物代谢物、抑制肠道炎症及维持肠道黏膜屏障功能完整性[21-25]。GPR109A 作为烟酸的重要受体,能够保护小鼠抵抗三硝基苯磺酸(TNBS)诱导的肠道炎症反应,而敲除 *GPR*109*A* 后,烟酸无法发挥其保护作用[11]。Singh 等[22]发现烟酸结合 GPR109A 后,促进小鼠树突状细胞和巨噬细胞产生抗炎因子白细胞介素-10(IL-10),进而诱导调节性 T 细胞产生和抑制辅助性 T 细胞(Th)17,同时烟酸通过 GPR109A 受体提高肠道上皮细胞白细胞介素-18(*IL*-18)的表达,从而促进肠道黏膜屏障损伤修复。作为降低肠道通透性及肠道炎症反应、提高肠道屏障完整性的重要受体,GPR109A 还与肠道微生物的多样性和稳定性存在密切的联系[26]。Bhatt 等[27]研究发现,GPR109A 有效限制肠道微生物诱导白细胞介素-23(IL-23)的产生,抑制了 IL-23 介导的肠道炎症疾病。然而,尚未有研究报道烟酸是否直接结合 GPR109A 调控断奶仔猪肠道健康。

2.2 烟酸通过其代谢产物 NAD^+ 调节能量代谢信号改善肠道黏膜屏障功能

能量代谢是细胞最基本、最重要的活动之一。研究表明,多种信号通路调控能量代谢平衡,其中单磷酸腺苷依赖的蛋白激酶(AMPK)信号通路和雷帕霉素靶蛋白(mTOR)信号通路被广泛研究。近年来的研究显示,SIRT1 在细胞能量代谢过程也扮演重要角色。研究表明,在能量水平较低的条件下,烟酸可通过 SIRT1、AMPK 及 mTOR 等能量代谢信号通路,降低肠道上皮细胞促炎症因子的表达,而提高肠道上皮细胞紧密连接蛋白的表达,从而促进肠道黏膜屏障功能损伤修复。

*SIRT*1 广泛表达于小肠和结肠等肠上皮细胞。作为细胞超灵敏的能量传感器,SIRT1 作为 NAD^+ 依赖的组蛋白去乙酰化酶,可以通过调控细胞核内转录因子的去乙酰化进行调节能量代谢及缓解应激反应,在细胞增殖、衰老、凋亡、炎症和能量代谢等方面发挥重要作用[28-29]。研究表明,烟酸通过 SIRT1 抑制脂多糖(LPS)诱导巨噬细胞分泌 TNF-α、白细胞介素-6(IL-6)及一氧化氮(NO)[30],同时烟酸调控 NF-κB 和 MAPK 信号通路抑制角质上皮细胞 IL-8 的产生[31]。作为重要的脱乙酰基酶,SIRT1 能够调控组蛋白和非组蛋白的去乙酰化。已有报道发现 SIRT1 调节 p65 蛋白去乙酰化从而促进 NF-κB 泛素化,并抑制 *NF-κB* 及其下游目标基因的转录[32-34]。大量研究表明,炎症反应、机体新陈代谢及线粒体生物三者能形成密切的关系网,而 SIRT1 在这个关系网中扮演重要调节作用。细胞核 SIRT1 调动免疫细胞进行糖酵解及脂肪氧化反应,为机体对抗炎症提供能量[35]。此外,SIRT1 还可以刺激肠道干细胞生长[36]及上调紧密连接蛋白的表达量[37],从而增强肠道屏障功能。肠道上皮平衡依赖于肠道上皮细胞、肠道共生菌及肠道免疫细胞三者之间的相互作用,如果此平衡被打破将会引起肠道紊乱。研

究表明,肠道微生物通过代谢物调控细胞线粒体的能量,肠道微生物代谢产物胆汁酸通过调节与脂质、碳水化合物代谢有关的基因,例如胆汁酸受体(*FXR*)影响肠道线粒体的能量代谢[38],而 FXR 是 SIRT1 的目标去乙酰化分子[39]。肠上皮细胞缺乏 SIRT1 导致胆汁酸浓度的升高,随之抑制肠道革兰氏阳性菌(乳酸菌等益生菌)的繁殖生长,而对革兰氏阴性菌(大肠杆菌或沙门氏菌等病原菌)没有太大的影响,从而改变肠道菌群结构,导致肠道紊乱,引起肠炎病[40]。Wellman 等[41]研究表明,肠道上皮细胞的 SIRT1 通过调节胆汁酸的浓度影响肠道菌群组成,从而加强肠道屏障功能。

当肠道黏膜细胞处于饥饿的状态时,AMPK 信号被激活,随之关闭消耗 ATP 的合成代谢途径,并同时启动产生 ATP 的分解代谢途径。作为细胞能量调节器,AMPK 能够刺激 NAD^+ 合成酶(NAMPT)生成,提高 NAD^+ 的浓度从而加强 SIRT1 的去乙酰化作用,促进 SIRT1 下游目的蛋白的去乙酰化[42],抑制慢性炎症反应[33,43-44];同时,SIRT1 的浓度同样影响 AMPK 的活化[45]。Nin 等[46]的研究也发现,SIRT1 迅速被活化的情况下有助于进一步激活细胞核内 AMPK。研究表明,AMPK 和 SIRT1 不能与雷帕霉素靶蛋白复合物 1(mTORC1)在同一种细胞中同时被激活。mTORC1 信号通常在细胞能量水平充足的条件下被激活,而 AMPK 和 SIRT1 一般是在细胞能量水平比较低的情况下被活化,能量限制会降低 *mTORC*1 的表达量而上调 *AMPK* 和 *SIRT*1 的表达量[47-48]。然而,最近的研究表明,在能量限制时,肠道干细胞的 *mTORC*1 的表达量上调了,促进了肠道干细胞蛋白质的合成及增加肠道干细胞的数量;此外,此研究显示能量水平不足会降低肠道潘氏细胞中 *mTORC*1 的表达了而上调此细胞环腺苷二磷酸核糖(*cADPR*)的表达量,进而激活肠道干细胞钙离子(Ca^{2+})信号通路,Ca^{2+} 信号通路的活化有助于 AMPK 磷酸化;活化的 AMPK 促进 NAD^+ 的合成,上调 *SIRT*1 的表达,SIRT1 的活化有利于核糖体蛋白 S6 激酶(S6K1)的去乙酰化,从而促进肠道干细胞中 mTORC1 的磷酸化;饲粮中添加烟酸可增加机体内的 NAD^+ 浓度,增强肠道干细胞 SIRT1 的活性,从而上调肠道干细胞 *mTORC*1 的表达量,实现促进肠道干细胞的增殖[36]。

3　小　结

仔猪在断奶时,通常会产生大量炎症因子,引起肠道上皮细胞凋亡和紧密连接结构紊乱,损伤肠道屏障功能;同时,断奶应激引起仔猪采食量降低,导致出现肠道黏膜供能不足,损害肠道屏障功能。已有研究表明,烟酸改善了断奶仔猪生长性能,但是,尚未阐明烟酸调控断奶仔猪肠道健康的机制。本文借鉴人和其他研究的动物模型阐述了烟酸调控肠道屏障功能,为烟酸调控断奶应激仔猪肠道黏膜损伤修复提供理论依据,然而,还需要进一步验证烟酸改善肠道黏膜屏障功能究竟是直接结合 GPR109A 还是通过其代谢产物 NAD^+ 调节能量代谢通路进而发挥作用,或者这 2 条通路同时发挥作用。

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Molecule Mechanism of Niacin Regulate Intestinal Epithelial Barrier Function of Animals

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Abstract: As an important vitamin, niacin plays important effects on prevention intestinal inflammation and maintaining intestinal healthy and other aspects. Research shows that niacin can directly bind to niacin receptor G protein-coupled receptor 109A (GPR109A) to play its physiological role, and partakes in cell energy metabolism in form of coenzyme nicotinamide adenine dinucleotide (NAD^+). This review mainly expounded the mechanism of niacin maintaining intestinal mucosal barrier function, to provide the scientific theoretical basis for maintaining intestinal health of weaned piglets. [***Chinese Journal of Animal Nutrition***, **2020**, **32**(**2**): **481-486**]

Key words: niacin; GPR109A; NAD^+; intestinal mucosal barrier; energy metabolism

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