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三七中高含量皂苷与肠道菌群的相互作用研究进展

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摘 要

口服三七后，其中的主要活性成分－皂苷与肠道菌群之间存在双向相互作用。三七皂苷(PNS)会经肠道内特定细菌所编码的糖苷水解酶催化，转化为稀有次级皂苷。另一方面，PNS及其代谢产物作为“类益生元”物质，能够显著改变肠道菌群的组成与结构。本文综述了三七中高含量皂苷干预肠道菌群结构的现有研究进展，结果显示皂苷对菌群的调节效应在不同疾病模型、干预剂量及宿主生理状态下呈现显著差异，同种皂苷对菌群*α*多样性及*Lactobacillus*、*Bifidobacterium*等菌属的调控方向常有反转。针对现有研究的局限性，后续研究可结合体外菌群与PNS的共培养体系、关键功能基因鉴定等方法，并重点关注菌群代谢产物对宿主的反馈。这将为阐明PNS调节宿主健康的核心作用路径提供参考，并为其基于肠道菌群特征的个体化应用奠定理论基础。

关键词

肠道细菌群落，三七皂苷，相互作用

Research Progress on the Interaction between High-Content Saponins of *Panax notoginseng* and Gut Microbiota

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Abstract

After oral administration of *Panax notoginseng*, there is a bidirectional interaction between its main active components—saponins—and the gut microbiota. *P. notoginseng* saponins (PNS) are catalyzed by glycoside hydrolases encoded by specific gut bacteria and converted into rare secondary saponins. On the other hand, PNS and their metabolites act as prebiotic-like substances, significantly altering the composition and structure of the gut microbiota. This article reviews current research progress on the intervention of high-content saponins from *P. notoginseng* in gut microbiota structure. The results show that the regulatory effects of saponins on microbiota vary significantly across different disease models, intervention doses, and host physiological states. The direction of regulation by the same saponin on *α* diversity and genera such as *Lactobacillus* and *Bifidobacterium* is often reversed. Given the limitations of existing studies, future research may combine methods such as *in vitro* co-culture systems of gut microbiota and PNS, identification of key functional genes, with a focus on the feedback effects of microbial metabolites on the host. This will provide a reference for elucidating the core pathways by which PNS regulate host health, and lay a theoretical foundation for their individualized application based on gut microbiota characteristics.

Keywords

Gut Bacterial Community, *Panax notoginseng* Saponins, Interaction

1. 引言

三七(*Panax notoginseng* (Burkill) F. H. Chen)为五加科人参属植物，是中国传统中药材，享有“止血神草”“南国神草”美誉。现代研究表明三七中含有皂苷、多糖、黄酮、挥发油等多种活性成分[1]，其中皂苷被认为是三七中的主要活性成分。2025 年，在被赋予“世界三七之都”称号的云南省文山州，以三七为主的生物医药产业实现综合产值 190 亿元，其产业规模显著。目前，三七是血塞通、复方丹参片、丹七片等多种中成药的主要成分，三七总皂苷制剂(如三七总苷片、三七总苷胶囊)已实现商品化，广泛应用于临床。除药用外，三七作为药食同源植物，还以保健食品的形式大量融入日常膳食与健康管理之中。

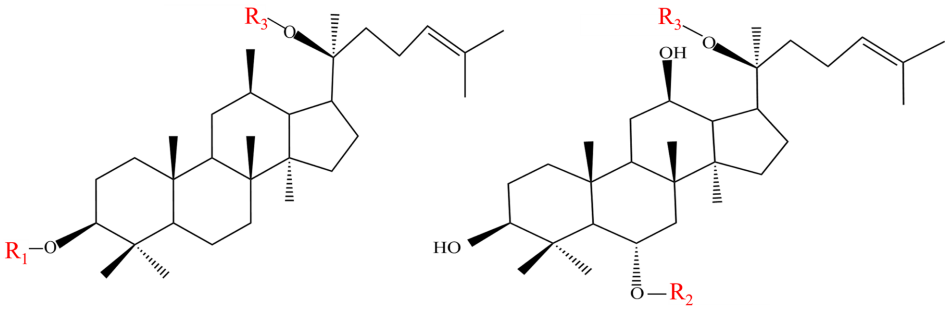
2. 三七中的高含量皂苷及其药理作用

2.1. 三七中的皂苷活性成分

目前研究者已从三七的根、茎、叶、花和果实中分离鉴定出含有人参皂苷、三七皂苷、绞股蓝皂苷等 270 种皂苷类化合物[2]。皂苷作为一类植物次生代谢产物，其结构分类主要基于非糖基部分的皂苷元，可分为三萜皂苷和甾体皂苷两大类别。三萜皂苷的苷元由 30 个碳原子构成，根据环的数目进一步分为四环三萜和五环三萜。其中，三七中的皂苷主要为达玛烷型四环三萜皂苷，可依据 C-6 位是否取代羟基，细分为 20(*S*)-原人参二醇型[20(*S*)-protopanaxadiol, PPD]，20(*S*)-原人参三醇型[20(*S*)-protopanaxatriol, PPT]，其化学结构见图 1。皂苷的糖基部分主要由葡萄糖(Glc)、阿拉伯糖(Arap)、木糖(Xyl)和鼠李糖(Rha)等单糖构成，它们通过氧苷键(-O-)特异性连接于苷元的 C-3/C-6/C-20 位。皂苷的糖基组成不仅决定了皂苷的

极性、溶解性与空间构象，也直接影响其生物利用度和药理活性，从而构成皂苷结构多样性与功能特异性的关键[3]。

三七皂苷(*P. notoginseng* saponins, PNS)可根据其相对含量分为高含量皂苷与稀有皂苷，高含量皂苷主要包括 PPD 型皂苷(Rb_1、Rd)和 PPT 型皂苷(Re、Rg_1、R_1)。云南不同地域所种植的三七中这五种高含量皂苷总量之和占比均大于 7.0% [4]，占 PNS 总含量的 90%以上[2]，其糖基组成及在三七主根中的含量见图 1。稀有皂苷种类众多，分子量较小、所含糖基较少，可以通过高含量皂苷脱糖或脱水获得。稀有皂苷通常生物活性更强、吸收率更高，且表现出多样的药理作用[5]。



20(*S*)原人参二醇型（PPD）型皂苷　　20(*S*)原人参三醇（PPT）型皂苷

皂苷	R_1（C-3）	R_2（C-6）	R_3（C-20）	三七主根中含量
Rb_1	Glc-Glc	H	Glc-Glc	29.20~112.70 mg/g
Rd	Glc-Glc	H	Glc	3.41~20.09 mg/g
Re	H	Glc	Glc-Rha	0.89~12.20mg/g
R_1	H	Glc	Glc-Xyl	3.69~59.76 mg/g
Rg_1	H	Glc	Glc	17.05~98.79 mg/g

Figure 1. High-content saponins in *Panax notoginseng* [2] [6]
图 1. 三七中的高含量皂苷[2] [6]

2.2. 三七中高含量皂苷的药理作用

三七中的高含量皂苷，尤以人参皂苷 Rg_1、Rb_1 为代表的高含量皂苷和三七皂苷 R_1 为代表的特征性皂苷成分为主，近年来在多方面开展了其药理作用的研究。

PNS 中的高含量皂苷通过多种机制发挥心血管保护作用。PNS 中的高含量皂苷可以通过改善脂质代谢[7]、抑制炎症反应、调节一氧化氮(NO)信号[8] [9]、促血管新生[10]、改善内皮细胞的形态特征[11]等机制，对动脉粥样硬化[12]、缺血性中风[13]、心肌缺血/再灌注损伤[14]-[16]等多类心血管疾病具有治疗潜力。皂苷类物质的抗炎效应也被广泛证实，其机制主要涉及抑制核因子 κB 等炎症信号通路，并降低促炎因子的释放[17]。在神经退行性疾病领域，人参皂苷 Rb_1、Rg_1、Rd 和 Re 作为主要研究对象，已在帕金森病(PD)中展现出神经保护潜力[18]。Rg_1 在阿尔茨海默病树鼩模型中可改善认知功能并调控肠道菌群[19]，Rb_1 在多种中枢神经系统疾病中的神经保护作用也被广泛证实[20] [21]。除心血管与神经系统外，PNS 还具有其他广泛的药理活性：可通过减少脂质合成、抑制脂肪生成、促进白色脂肪组织褐变和增加能量消耗等途径发挥抗肥胖作用[22]；改善以肠道炎症为特征的消化系统疾病，如炎症性肠病(IBD)等[23]。研究表明，PNS 能够重塑肠道微生物群，促进胆汁酸生成，从而改善 Th17/Treg 细胞平衡，发挥抗 IBD 作用[24]。

在皂苷药理作用研究中发现，由于皂苷具有高溶血活性，静脉给药途径受限[25]。皂苷可以与红细胞膜胆固醇结合生成不溶性分子复合物，破坏红细胞的渗透性，导致细胞膜去稳定，细胞膜溶解而导致溶血现象发生。口服给药后，皂苷进入肠道并与肠道菌群发生相互作用，Rb_1、Rg_1 等高含量皂苷会被分解为稀有次级皂苷后被人体吸收，而皂苷也会反过来影响肠道菌群。近年研究揭示肠道菌群在 PNS 代谢转化及其系统药效发挥中的关键作用，提示了“成分－菌群－宿主”互作的新机制维度。

3. 三七皂苷与肠道菌群的相互作用研究现状

肠道微生物包括细菌、古细菌、真菌、病毒，其中在个体肠道内有 150~170 种细菌占据主导地位，主要隶属于厚壁菌门、拟杆菌门、放线菌门、梭杆菌门、变形菌门、疣微菌门[26]。其中拟杆菌门和厚壁菌门占总数的 90%以上[27]，在不同的个体或者同一个体的不同生命阶段，它们之间的相对比例存在明显差异[28] [29]。大量研究表明，肠道菌群在炎症性肠病、肥胖、自闭症谱系障碍、免疫系统疾病等多种疾病中发挥关键作用。植物提取物能通过调节肠道菌群来发挥其健康益处[30]。而肠道益生菌如 *Escherichia coli* Nissle 1917 的定殖可有助于胃肠功能障碍性疾病的治疗[31]。

3.1. 肠道菌群对 PNS 的生物转化

口服三七后，其中的药理活性成分会受到肠道菌群的影响，改变其生物利用度和代谢途径。三七中的高含量皂苷进入人体后，因相对分子质量较大、脂溶性差，不易被人体直接吸收[32]。这些高含量原型皂苷会被特定的肠道菌群代谢，进行脱糖、脱氢、氧化和脱水等反应生成稀有次级皂苷或其他代谢产物[33] [34]。

PNS 进入人体后依赖肠道菌群进行代谢，否则无法有效实现生物转化。例如，在常规小鼠中灌胃 PNS，可检测到稀有次级皂苷 F_1、Rh_2、CK、aPPT；而在无菌大鼠血浆中则未检测到[35]。皂苷在体内的主要代谢方式是脱糖反应，是主要由微生物 *β*-糖苷酶催化的寡糖链水解的去糖基化反应，基本规律为四糖苷→三糖苷→二糖苷→单糖苷[36]。以人参皂苷 Rb_1 为例，Rb_1 在体外肠道菌群模型中，通过脱去糖基的代谢过程为 Rb_1→Rd→F_2→CK [37]。除了逐级脱糖反应，也存在其他次要的代谢途径，研究者发现在低脂肪和富含植物纤维饮食模式的肠道菌群中，PNS 主要发生脱糖反应，而在高脂肪和高蛋白饮食模式的肠道菌群中，PNS 主要发生脱氢、羟基化反应生成脱氢－原人参三醇[38]。

为探究哪些菌群参与皂苷代谢，研究者比较了皂苷代谢效率不同的小鼠肠道菌群。通过比较高效率和低效率皂苷代谢效率的小鼠肠道菌群，发现高效率组中 S24-7 科，产碱菌科(Alcaligenaceae)和丹毒丝菌科(Erysipelotrichaceae)的丰度显著高于低效率组[39]。人和小鼠肠道菌群介导的皂苷生物转化存在显著的物种特异性差异[40]，考虑到物种差异，研究者直接使用了人体粪便样品进行体外筛选。通过比对能将人参皂苷 Rb_1 有效代谢为 CK 的粪便样品与不能代谢的样品，发现 *Ruminococcus*、*Bacteroides* 和 *Bifidobacterium* 等属群水平显著增加[41]。

对于不同个体而言，肠道菌群的组成不同，皂苷的代谢活动存在显着差异[42]。Wenjing Wei 等人使用 50 名健康志愿者的肠道菌群在体外与人参皂苷 Rb_1 共培养，发现了显著的个体间差异，与高生物转化能力显著正相关的明确菌属包括：*Eubacterium_hallii_group*、*Anaerostipes* 等菌属[40]。在益生元干预增强肠道菌群转化皂苷的研究中，益生元干预可显著提高 *Prevotella* 丰度，推测其通过选择性促进糖苷水解菌(如 *Prevotella*)增殖，从而增强皂苷的生物转化和生物利用度[43]。将可能参与皂苷转化的候选菌群整理于图 2，发现其主要集中于拟杆菌门和厚壁菌门。在探究肠道细菌群落结构与 PNS 代谢谱之间的关系时，发现变形菌门可能通过调节糖苷酶活性，而拟杆菌门可能通过调节氧化还原代谢酶活性，二者共同影响 PNS 的代谢[44]。

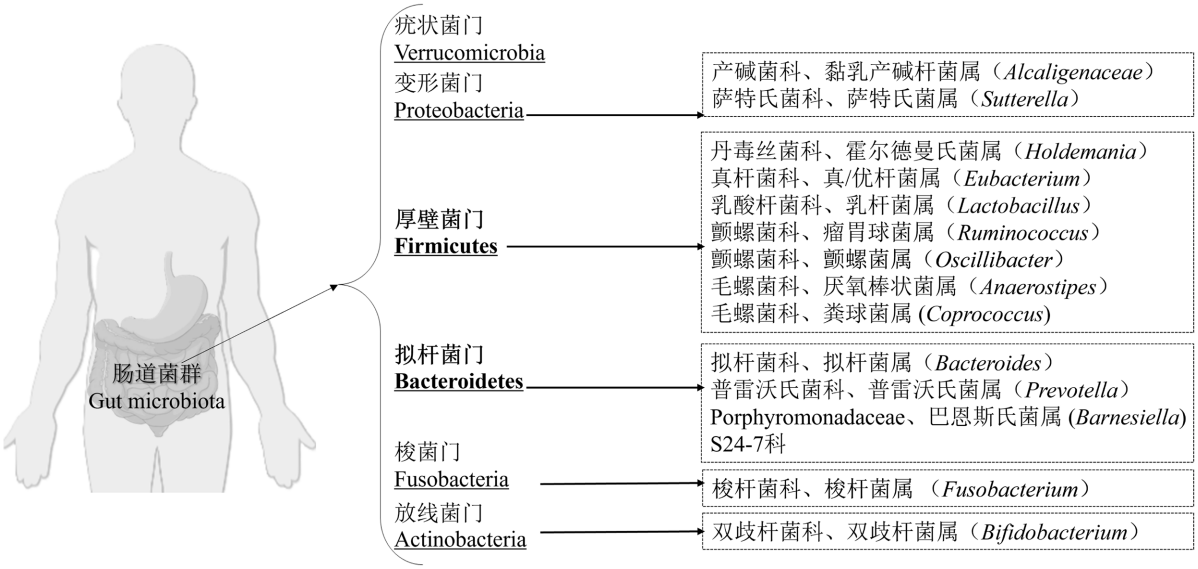


Figure 2. Potential candidate gut bacterial genera involved in saponin conversion
图 2. 可能参与皂苷转化的候选肠道菌属

因肠道菌群的组成受到环境、饮食[45]等多种因素的动态调节，且在不同个体间乃至同一个体的不同生命阶段均呈现出高度异质性，因此仅基于总体肠道菌群进行皂苷转化的比较分析存在一定的局限性。为更精确地阐释特定微生物在皂苷转化中的作用，采用分离培养法，针对单一菌种展开皂苷转化的功能研究。目前乳杆菌属，双歧杆菌属和梭杆菌属都有已分离出的菌株进行皂苷转化研究，其中双歧杆菌属的占比最多(表 1)。从肠道菌群宏基因组或某一菌株的基因组中挖掘相关糖苷水解酶序列进行异源表达，来进行皂苷转化研究。如研究者从短双歧杆菌(*Bifidobacterium breve*) ATCC15700 中挖掘的糖苷水解酶家族(Glycoside hydrolases, GH) *β*-木糖苷酶可以将人参皂苷 Rd 转化为人参皂苷 F_2 和 CK [46]；*β*-葡萄糖苷酶 BaBgl1A 可以将 Rb_1 和 Rd 的转化为人参皂苷 Gyp17 和 F_2；BaBgl3A 将 Rb_1 转化为 Gyp17 和 Rd [47]。整理发现，能够转化皂苷的酶主要属于 GH1、GH3、GH30、GH51 家族[48]。

Table 1. Transformation of saponins by isolated and cultured intestinal bacteria [39] [48]
表 1. 分离培养肠道菌对皂苷的转化[39] [48]

菌种		菌株	皂苷转化途径
乳双歧杆菌	*Bifidobacterium lactis*	Bi-07	Rb_1 and Rc → F_2
长双歧杆菌	*Bifidobacterium longum*	KCCM11953	Rb_2/Rb_1 and Rc → Rd
		RD47	Rb_2 and Rc → Rd
双歧杆菌	*Bifidobacterium* sp.	K-525	Rg_1 and Re → Rh_1
动物双歧杆菌	*Bifidobacterium animalis*	GM1	Rb_1 → Rd
梭杆菌	*Fusobacterium* sp.	K-60	Gyp17 → CK
发酵乳杆菌	*Lactobacillus fermentum*	KCCM40401	Rb_2/Rb_1 and Rc → Rd

3.2. PNS 对肠道菌群结构和功能的影响

口服三七后，所含皂苷在肠道菌群作用下发生代谢转化，其代谢产物又可反过来调节肠道菌群的结构与功能。通过宏基因组测序技术，分析口服皂苷前后肠道内容物或粪便中肠道菌的变化，可以了解其对体内肠道菌群组成造成的影响。目前研究认为皂苷是“类益生元”化合物，主要作用是促进肠道的有益菌种的生长，同时抑制有害菌种的丰度[32] [49]。皂苷的摄入可以通过改善肠道菌群结构，进而治疗肠道疾病或其他相关疾病[33]。

通过梳理不同研究中三七高含量皂苷干预后肠道菌群 α 多样性及丰度发生显著变化的菌属(表 2)，可以发现：皂苷在不同模型或剂量下引起的 α 多样性变化截然相反，其对菌属的调节作用也呈现出高度异质性。其中，在菌属丰度变化层面，调节方向的分歧表现得更为突出。同一菌属在不同条件下的应答常出现反转，统计中出现频率最高的菌属为乳酸杆菌属(*Lactobacillus*)。乳酸杆菌属在 Rb_1 动脉粥样硬化模型(20 mg/kg)和健康小鼠中被富集等 11 项研究中其丰度上升，却在 Rb_1 高脂饮食模型(60 mg/kg)、Rg_1 糖尿病模型及 R_1 脑缺血模型中研究中则被显著抑制。*Bifidobacterium* 亦在健康小鼠和衰老模型中被上调，却在 Rg_1 抑郁模型和高剂量 Rb_1 高脂饮食模型中下降。被视为有益菌的 *Akkermansia* 虽在多组 Rb_1干预中被富集，但也并非普遍存在。这些证据表明，皂苷对肠道菌群的调节远非简单的“促益抑害”线性模式，而是皂苷类型、剂量、模型状态与初始菌群结构多重因素交织形成的复杂网络效应。只观测皂苷和肠道微生物的关系，无法直接判断皂苷对肠道微生物的影响而对针对的症状产生稳定效果，因此难以形成稳定的结果。

Table 2. The impact of high levels of saponins from *Panax notoginseng* the gut microbiota at the genus level
表 2. 三七中的高含量皂苷对肠道菌群的影响

皂苷类型	肠道菌群来源	α 多样性	肠道菌属水平的显著变化	
Rb_1 (200 mg/Kg)	高脂饮食－鼠	Shannon↓ Pielou_E↓	↑*Akkermansia* ↓*Allobaculum*, *Reyranella*, *Eubacertium*	[53]
Rb_1 (60 mg/kg)	高脂饮食－鼠	Chao1↑ Shannon↑ Simpson↓	↑*Blautia*, *Eisenbergiella*, *Anaerostipes* ↓*Clostridium subcluster XIVa*, *Clostridium cluster XVIII*, *Eubacterium*, *Lactobacillus*, *Turicibacter*, *Bifidobacterium*, *Clostridium-sensu-stricto*	[54]
Rb_1 (120 mg/kg)	高脂饮食－鼠	Shannon↓ Pielou_E↓	↑*Akkermansia*, *Parasutterella*, *Bacteroides* ↓*Allobaculum*, *Intestinimonas*, *Oscillibacter*, *Alistipes*, *Helicobacter*	[55]
Rb_1 (20 mg/kg)	动脉粥样硬化模型－鼠	Chao1↑ Shannon↑	↑*Lactobacillus*, *Prevotella*, *Oscillospira*, *Blautia*	[56]
Rb_1 (600 μg)	接种 H1N1 流感疫苗－鼠	NR	↑*Akkermansia*	[57]
Rb_1 (30 mg/kg)	衰老模型－鼠	Shannon↑ Ace↑	↑*Helicobacter*, *Lactobacillus*, *Mycoplasma*, *Bifidobacterium* ↓*Streptococcus*	[58]
Rb_1 (50 mg/kg)	衰老模型－鼠	Shannon↓	↑*Stenotrophomonas*, *Faecalibaculum*, *Oligella*, *Facklamia*, *Paenalcallgenes* ↓*Anaeroplasma*, *Blautia*, *Lactobacillus*, *Ruminicidstridium*	[59]

续表

Rb_1 (50 mg/kg)	健康－鼠	NR	↑*Bifidobacterium*, *Lactobacillus*	[60]
R_1 (80 mg/kg)	脑缺血再灌注损伤及药物干预－鼠	Shannon↑ Simpson↑	↓*Escherichia*, *Shigella*, *Lactobacillus*, *Morganella*	[61]
R_1 (25 mg/kg)	动脉粥样硬化模型－鼠	Chao1↓ Shannon↓ Simpson↓	↑*Akkermansia*, *Bacteroides*, *Lactobacillus*, *Prevotella*, *Clostridium* ↓*Desulfovibrio*, *Ruminococcus*	[62]
Re (15 mg/kg)	衰老模型－鼠	Shannon↑ Ace↑	↑*Helicobacter*, *Lactobacillus*, *Mycoplasma*, *Bifidobacterium* ↓*Streptococcus*	[58]
Rg_1 (100 mg/kg)	糖尿病－鼠	Shannon↑	↑*Lachnoclostridium* ↓*Lactobacillus*	[63]
Rg_1 (200 mg/kg)	吗啡依赖性模型－鼠	Chao1↑	↑*Bacteroides*	[64]
Rg_1 (40 mg/kg)	酒精性肝病模型－鼠	Chao1↓ Shannon↓	↑*Lactobacillus*, *Oscillibacter*, *Blautia*, *Alistipes*, *Ruminiclostridium*, *Alloprevotella*, *Lachnospiraceae_NK4A136_group*	[65]
Rg_1 (40 mg/kg)	抑郁模型－鼠	Shannon↑	↑*Lactobacillus*, *Bacteroides*, *Allobaculum*, *norank_f_Muribaculaceae* ↓*Bifidobacterium*	[66]
Rg_1 (200 mg/kg)	急性溃疡性结肠炎模型－鼠	Chao1↑ Ace↑	↑*norank_f_Muribaculaceae*, *Lactobacillus*, *Allobaculum*, *Akkermansia*, *Turicibacter* ↓*Odoribacter*, *Clostridia_UCG*-014, *Bacteroides*, *Turicibacter*	[67]
Rg_1 (30 mg/kg)	阿尔茨海默症模型－树鼩	NR	↑*Morganella*, *Lactobacillus*, *Escherichia-Shigella*	[68]
Rd (20 mg/kg)	糖尿病鼠模型－鼠	Chao1↑ Simpson↑ Shannon↑ Pielou_E↑	↑*SMB53*, *Ruminococcus*, *rc4-4* ↓*Lactobacillus*, *Clostridium*	[69]
Rd/Rb_3 (分别加入 20 mg/kg)	结肠息肉模型－鼠	NR	↑*Lactobacillus*, *Bifidobacterium*, *Ruminococcus*, *Prevotella*, *Blautia* ↓*Butyricimonas*, *Campylobacter*, *Dysgonomonas*, *Fusobacterium*, *Helicobacter*	[70]
Rb_1、Rb_2、Rb_3、Rc (分别加入)	人源粪便 GAM 培养	NR	↑*Escherichia*, *Streptococcus*, *Ruminococcus* ↓*Dorea*, *Sutterella*, *Prevotella*, *Megasphaera*	[71]
人参二醇型皂苷 (10 mg/kg)	肥胖小鼠－模型	NR	↑*Bacteroides*, *Lactobacillus*, *Akkermansia*, *Alloprevotella*, *Lachnospiraceae-UCG*-006	[72]
人参二醇型皂苷	人类粪便 GAM 培养	NR	↑*Escherichia-Shigella* ↓*Dorea*, *Prevotella*_9, *Megasphaera*	[73]

4. 讨论

口服三七后，其富含的原型皂苷进入肠道，被具有皂苷代谢能力的“初级代谢菌群”转化为稀有的次级皂苷，这些次级皂苷随后又作用于肠道中的“次级利用菌群”。由于不同个体的肠道菌群组成存在差异，同一原型皂苷可能引发截然不同的菌群重组方向。皂苷在调节菌群的同时，自身也被菌群代谢转化，这种双向互作使整个过程极为复杂[42]。

资源竞争与交叉哺育的相互作用是决定微生物群落组装的核心机制之一。在研究聚糖与肠道菌群的互作中，已发现肠道菌群间存在大量的交叉喂养，一种生物体的副产品可以成为另一种生物体的重要资源[50]。例如，拟杆菌属菌株能够降解并消耗 β-葡聚糖，其释放的寡糖可被其他肠道微生物(如双歧杆菌和植物乳杆菌)利用，从而形成有序的代谢接力[51]。这种代谢分工模式也适用于皂苷的多糖基结构，不同菌株仅拥有部分去糖基化所需的酶。

同时疾病本身会重塑了肠道菌群的组成和代谢能力。当相应疾病导致具备糖苷酶关键编码基因的菌群，如某些拟杆菌属和双歧杆菌属物种减少时，皂苷的去糖基化效率大幅下降，稀有次级皂苷生成不足，原本在健康状态下应该被富集的菌属便无法获得代谢底物的支持。这提示，皂苷并非独立的药物分子，而是一个需要肠道菌群“活化”的前体物质；其治疗潜力取决于宿主肠道菌群的代谢活化能力，而疾病状态恰恰可能削弱这一能力。So Yeon Jeon 等人通过将皂苷提取物与益生菌 *Bifidobacterium longum* CBT BG7 和 *Lactiplantibacillus plantarum* CBT LP3 PM 混合，定向增强了皂苷的去糖基化转化，提高了其利用率[52]。这一策略表明，通过靶向补充具有特定代谢功能的菌株，有望恢复疾病状态下受损的皂苷活化效率。在此基础上，未来可发展基于肠道菌群代谢潜力的个体化用药方案，以最大化三七的临床疗效。

5. 总结与展望

PNS 的体内药理活性的发挥高度依赖于特定肠道细菌及其编码的糖苷水解酶介导的去糖基化代谢过程，从而转化为生物利用度更高的稀有次级皂苷。同时，PNS 及其代谢产物能够重塑肠道菌群结构，然而调节效应呈现显著的矛盾性与条件依赖性：不仅同种皂苷在不同疾病模型及剂量下对菌群 α 多样性的影响方向截然相反，且 PNS 对 *Lactobacillus*、*Bifidobacterium*、*Akkermansia* 等关键菌属丰度的调控效应随宿主背景与实验条件而异。这种复杂网络效应表明，单纯依赖菌群测序比较，难以厘清 PNS 在复杂宿主环境下对特定菌群的直接作用靶点与分子机制。

针对现有研究局限，可以筛选或构建特征性肠道菌群，借助大量、可重复的体外共培养体系，在错综复杂的网络关系中寻找共性规律。其次可以将研究重心前移至皂苷生物转化的关键酶及其编码基因。尽管肠道菌群的物种组成存在显著个体异质性，但执行特定代谢功能的基因簇相对保守，因此可借助宏基因组学策略系统鉴定不同人群肠道中负责皂苷去糖基化的核心糖苷水解酶基因，以挖掘 PNS 代谢的分子基础。最后，除观测干预前后的组成差异外，更应整合宏转录组学与宏代谢组学等多组学技术，追踪 PNS 干预进程中菌群的响应特征，解菌群代谢皂苷产生的短链脂肪酸、次级胆汁酸等物质如何反过来影响宿主。通过上述多层次、多维度研究体系的整合，有望更为精准地揭示 PNS 调节宿主健康的共性作用机制，进而为其基于肠道菌群特征的个体化用药与精准营养干预策略提供坚实的理论依据。

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