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Review 综 述

细菌半乳糖代谢通路及对细菌毒力的影响研究进展

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摘 要：半乳糖是自然界中广泛存在的一种单糖，不仅能作为碳源参与生物能量代谢，还能作为生物合成前体参与生物各类生理反应。在真核细胞中，半乳糖或相关衍生物能作为信号分子参与细胞间通讯。近期的研究发现，半乳糖能调控细菌内信号传导影响细菌毒力，半乳糖因此被认为是细菌致病过程中低估的重要环境调控因子，但具体调控机制还未被充分揭示。本文结合最新研究数据，对细菌半乳糖代谢通路以及半乳糖代谢在细菌毒力、细菌-宿主互作等方面发挥的生物学功能进行综述，并对半乳糖代谢途径中关键蛋白(酶)作为开发新型疫苗潜在靶点进行讨论，为深入理解细菌致病机制和挖掘新型抗菌策略的相关研究提供新的思路和参考借鉴。

关键词：半乳糖；细菌；代谢通路；毒力调控

Research progress in bacterial galactose metabolic pathway and its impacts on bacterial virulence

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Abstract: Galactose is a ubiquitous monosaccharide in nature, serving not only as a primary carbon source for bioenergy metabolism but also as a precursor for various biological synthesis

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reactions. In eukaryotic cells, galactose or its derivatives can act as signaling molecules to participate in intercellular communication. Recent studies have revealed that galactose can modulate bacterial virulence by regulating intracellular signal transduction. Accordingly, galactose is considered an underappreciated environmental regulator in bacterial infection. However, the specific regulatory mechanisms remain incompletely elucidated. This review integrates the latest research findings to summarize the bacterial galactose metabolic pathway, the biological implications of galactose metabolism in bacterial virulence and interactions with hosts, and the key proteins (enzymes) in the galactose metabolism pathway as potential targets for developing novel vaccines. It offers new insights and reference for comprehending bacterial infection mechanisms and exploring innovative antibacterial strategies.

Keywords: galactose; bacteria; metabolic pathway; virulence regulation

半乳糖是自然界中广泛存在且能被所有生物利用的一种常见单糖，不仅作为乳糖分子的亚单位存在乳制品中，在谷物、水果、豆类、坚果、肉类和蔬菜中也含量较高[1-2]。半乳糖通常以游离单糖的形式存在，也可以通过各类糖苷键(如 α-1,6、β-1,3 和 β-1,4)与其他碳水化合物(如低聚糖、多糖)结合[3]。半乳糖同时还是糖蛋白、糖脂、含糖代谢产物的组成成分[1]。自然界中的半乳糖通常以 D-半乳糖为主，L-半乳糖也天然存在[4]。

在真核细胞中，半乳糖在多种生理过程中扮演重要角色，既作为细胞新陈代谢的必需碳水化合物参与细胞中能量的产生及储存，又作为信号分子参与细胞间的通讯[5-6]。半乳糖也是糖基化的前体，在糖基化过程中，半乳糖通过与蛋白质和脂质的结合，形成结构复杂的糖蛋白、蛋白聚糖、糖脂等糖共轭物，这类分子不仅赋予细胞表面特定的识别标记，还参与细胞信号传导、细胞黏附、细胞免疫应答和细胞骨架形成等生理反应[7-8]。半乳糖还可以通过参与鞘糖脂的合成影响溶酶体的功能[9]。此外，半乳糖代谢与线粒体功能密切相关，代谢的中间体和产物可以作为各种线粒体过程的底物或辅因子，从而潜在地影响线粒体活性和整体细胞能量稳态[10]。在特定的细胞(如原代肌肉细胞)中，半乳糖还被发现可以通过促进有氧代谢增强线粒体的能量产生效率[11]。半乳糖代谢异常通常会对生物体健康造成严重的影响，例如半乳糖血症会造成机体发育迟缓，严重者导致死亡[5]。

在细菌中，半乳糖也在能量获取及特定生理反应调节过程中扮演着多种角色。除了能够为细菌提供能量和生物合成的前体物质，例如双歧杆菌(*Bifidobacterium*)和乳杆菌(*Lactobacillus*)可利用宿主体内具有益生作用的低聚半乳糖(galactooligosaccharides, GOS)促进其在肠道中的生长及正常定殖[12-13]，半乳糖代谢还能够通过调节细菌内部的信号传导途径，影响环境适应性的改变[14]，因此被认为是细菌致病过程中被低估的重要环境调控因子。在许多致病细菌中，半乳糖代谢途径的活性还与细菌的毒力密切相关。目前研究较为深入的几种调控方式包括：(1) 作为碳源，致病菌可以通过增强半乳糖代谢效率促进细菌的生存能力提高细菌对宿主的感染效率；(2) 在大肠杆菌(*Escherichia coli*)等细菌中，内源半乳糖可以通

过调控某些毒力因子[如特定类型的黏附素(adhesin)、内毒素(toxin)]的表达增强细菌对宿主细胞的黏附和在宿主体内的免疫逃避;(3) 半乳糖可以促进细菌生物被膜的形成效率，通过协助细菌抵御宿主系统的攻击和抗生素在生物被膜中的渗透，增强细菌在宿主体内的生存及感染能力;(4) 半乳糖还能通过改变细胞表面特定结构(例如拓扑形貌结构)影响细菌与宿主细胞受体的结合[15]。此外，半乳糖代谢过程中产生的中间代谢产物，如半乳糖酸和半乳糖醛酸等，也可能直接或间接地影响部分细菌[如大肠杆菌、啮齿枸橼酸杆菌(*Citrobacter rodentium*)]的致病性[16-17]。这些代谢产物可能通过改变细菌的生理状态或调节毒力基因的表达，从而影响细菌的致病力。研究表明，在某些肠道致病菌中，如空肠弯曲杆菌(*Campylobacter jejuni*)，细菌能量代谢可以通过重塑细胞膜结构促进特定毒力因子(短链溶血磷脂酰乙醇胺)的分泌提高细菌毒力[18-21]，以及提高细菌在宿主体内的铁离子转运效率、支链氨基酸代谢效率，抑制具有细胞毒性的活性氧在细菌内的积累，使细菌在类肠道内环境中具有更高的增殖效率[22-23]。半乳糖可以通过糖基化修饰改变细菌细胞膜上受体的活性及定位[15]，因此对细菌特定细胞结构的修饰也可能是半乳糖影响细菌毒力的一种调控方式。

细菌如何通过半乳糖代谢途径调节其毒力基因的表达，以及这一过程如何受到宿主环境因素的影响，是当前研究的热点问题。虽然半乳糖代谢已被大量报道与细菌致病性密切相关，但其对细菌毒力调控的具体分子机制还存在大量未知。深入研究这些机制，不仅可以揭示细菌致病性的分子基础，还可以为开发新的抗菌药物和疫苗提供潜在的靶点。本文重点对细菌半乳糖代谢通路以及半乳糖代谢与不同种细菌毒力之间的关系进行详细探讨，以期为细菌性疾病的防治提供新的视角和思路。

1 细菌半乳糖代谢的通路

大部分细菌的半乳糖代谢是通过 Leloir 途径完成的，通过 Leloir 途径半乳糖可转化为葡萄糖-1-磷酸，随后进入糖酵解途径产生能量或合成糖原(图 1)[1,24]。在此过程中，半乳糖首先通过半乳糖渗透酶(galactose permease, GalP)或磷酸转移酶系统(phosphotransferase system PTS, LacEF)将胞外的半乳糖转运进菌体中[1,25]，随后半乳糖变旋酶(galactose mutarotase, GalM)将进入胞内的 β-半乳糖异构化为 α-半乳糖[26-27]，半乳糖激酶(galactokinase, GalK)进一步将 α-半乳糖磷酸化为半乳糖-1-磷酸(galactose-1P)[28-29]，半乳糖-1-磷酸再与半乳糖-1-磷酸尿苷酰转移酶(galactose-1-phosphate uridylyltransferase, GalT)在尿苷二磷酸葡萄糖-4-差向异构酶(UDP-galactose 4-epimerase, GalE)的催化下与二磷酸尿苷葡糖-葡萄糖(uridine diphosphate glucose, UDP-glucose)反应生成 UDP-半乳糖(UDP-galactose)和葡萄糖-1-磷酸(glucose-1P)[30-32]。葡萄糖-1-磷酸最后在磷酸葡萄糖变位酶(phosphoglucomutase, Pgm)的作用下被进一步转化为葡萄糖-6-磷酸(glucose-6P)后进入糖酵解途径产生 ATP[33]。

此外，在乳杆菌属(*Lactobacillus*)和链球菌属(*Streptococcus*)的一些细菌中，还存在塔格糖-6-磷酸(tagatose-6-phosphate)代谢途径[1]，通过此途径半乳糖被由 *lacEF* 编码的转运系统(phosphotransferase system PTS, LacEF)转入细胞后[34]，可被由 *lacAB* 编码的 6-磷酸-半乳糖异构酶(galactose-6-P isomerse, LacAB)、*lacC* 编码的 6-磷酸塔格糖激酶(tagatose-6-P kinase, LacC)以及 *lacD* 编码的 1,6-塔格糖-二磷酸醛缩酶(tagatose-1,6-bP aldolase, LacD)进一步裂解为 3-

磷酸-甘油醛(glyceraldehyde 3 phosphate, GPDH)及磷酸二羟丙酮(dihydroxyacetone phosphate, DHAP)[35-36], 随后进入糖酵解途径被细菌利用(图 1)。取决于遗传特性和环境适应能力，不同的细菌可能只拥有上述 2 种途径中的一条或两者皆有。

2 半乳糖代谢对细菌毒力的影响

2.1 链球菌(*Streptococcus*)

链球菌是一类广泛存在于人和动物口腔、皮肤、肠道等处的革兰氏阳性菌，包括化脓性链球菌(*Streptococcus pyogenes*) [又称为 A 群链球菌(group A *Streptococcus*, GAS)]和肺炎链球菌(*Streptococcus pneumonia*) 2 种主要致病菌[37]，其他病原菌还有无乳症链球菌(*Streptococcus agalactiae*) [又称为 B 群链球菌(group A *Streptococcus*, GBS)]、猪链球菌(*Streptococcus suis*)、变形链球菌(*Streptococcus mutans*)、肝炎链球菌(*S. mitis*)[38]。半乳糖代谢对链球菌毒力的调控主要通过调节毒力因子的表达以及影响生物合成前体的产生而完成。

在化脓性链球菌(*S. pyogenes*)中，适应性代谢酶 LacD 被报道可以通过改变半乳糖代谢的效率调控包括分泌型半胱氨酸蛋白酶 SpeB 在内的多种毒力因子的表达调控细菌毒力[1,39-41]。半乳糖还能以供体依赖的方式干扰 *S. pyogenes* 与靶标细胞表面相关结构(如口腔上皮细胞的 A、B、H 抗原结构)的相互作用，从而影响 *S. pyogenes* 在宿主体内的定殖[42]。此外，在感染宿主过程中，*S. pyogenes* 的重要毒力因子成孔毒素链球菌溶血素(streptolysin O, SLO)能结合到宿主细胞膜表面并在细胞膜上形成孔洞造成细胞毒性，而含半乳糖的糖类共轭物是 SLO 准确膜定位的重要识别靶点[43-44]。

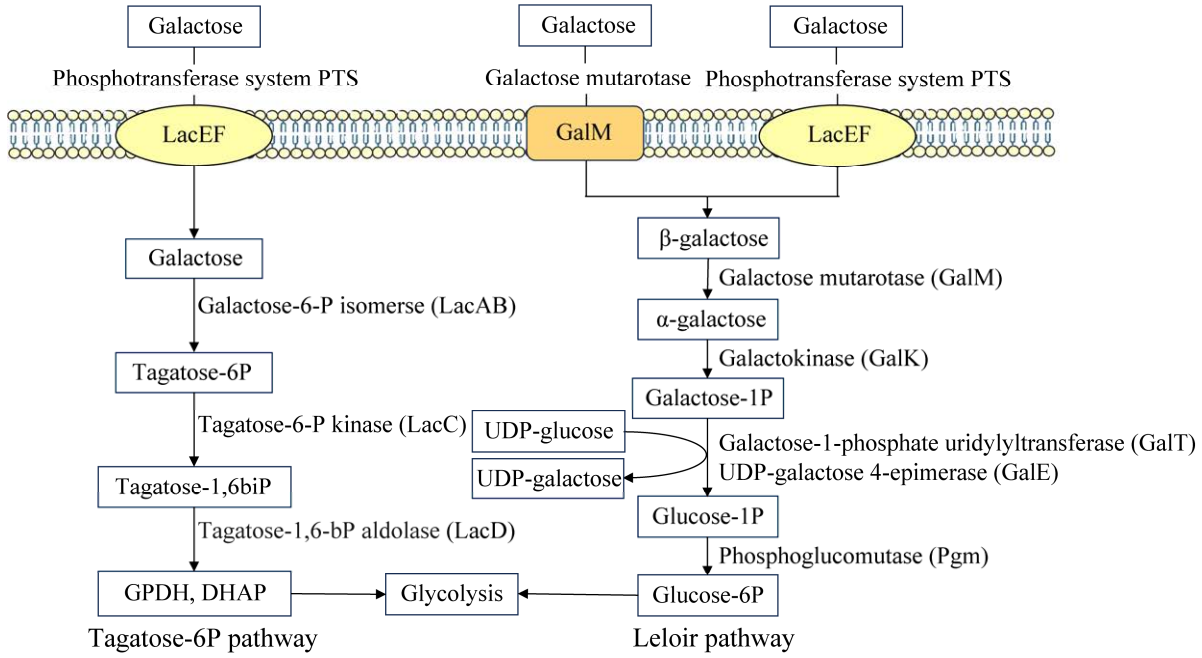

图 1 细菌半乳糖代谢 Tagatose-6-phosphate 及 Leloir 途径

Figure 1 Tagatose-6-phosphate and Leloir galactose metabolic pathways in bacteria. The enzymes involved in the different catalyzed reactions are indicated in the text.

在肺炎链球菌 *S. pneumoniae* 中，半乳糖能通过激活 TprA/PhrA 及 Rgg1518/SHP1518 群体感应系统(quorum sensing, QS)，作用于 LacI 家族糖代谢转录因子 CcpA 引起糖代谢抑制，利用半乳糖积累丙酮酸甲酸酶(pyruvate formate lyase, PFL)并通过谷氨酰胺代谢转录调控因子 GlnR、环境及营养感知系统转录调控因子 GntR，以及介导细胞壁最小肽聚糖(minimal peptidoglycan, PG)周转率调控 *S. pneumoniae* 的毒力及定殖能力[45-49]。同时半乳糖还能分别从 Leloir 和塔格糖代谢 2 条途径显著调控 *S. pneumoniae* CPS 及生物被膜的生成效率，因此半乳糖被认为是 *S. pneumoniae* 转录调控、种群行为和毒力的关键决定因子，同时半乳糖代谢途径中关键蛋白(酶)还被视为开发抗 *S. pneumoniae* 感染药物及疫苗的潜在作用靶点[50-53]。当肺炎链球菌利用半乳糖而不是葡萄糖作为唯一的碳源时，经鼻感染小鼠时它们表现出更强的毒力[51]。此外，在由甲型流感病毒(influenza A virus, IAV)引起的 *S. pneumoniae* 继发性细菌感染过程中，IAV 被发现能通过影响 *S. pneumoniae* 半乳糖代谢关键蛋白的正常磷酸化调控细菌的生长能力及毒力[54]。

在 B 群链球菌(GBS)中，半乳糖代谢被报道与细菌毒力及环境适应性密切相关[55]。GBS 和猪链球菌 *S. suis* 是唯一能合成唾液酸化荚膜多糖的革兰氏阳性菌，唾液酸通过 α-2,3 与半乳糖相连形成 GBS 的荚膜多糖(capsular polysaccharide, CPS)[56]，这种连接方式可以在细菌感染过程中通过调控免疫细胞(如树突状细胞)的活化，影响细胞免疫反应[57]。

在猪链球菌(*S. suis*)中，*S. suis* 菌膜表面受体磷酸转移酶(phosphotransferase, FruA)可激活 AI-2 QS 系统，上调半乳糖代谢效率，促进荚膜多糖 CPS 生成并增强 *S. suis* 毒力[58]，表明半乳糖代谢在 *S. suis* 致病过程中发挥着重要作用。我国学者 Jiang 等也于 2022 年首次报道半乳糖代谢途径中的适应性代谢酶 LacD 是介导 *S. suis* 代谢、应激和毒力的一种重要多功能蛋白[59]。然而，以上研究中，半乳糖对 *S. suis* 应激和毒力的调控是由半乳糖代谢的中间产物还是最终产物发挥作用，以及具体的分子机制还未被充分揭示。

在变形链球菌(*S. mutans*)中，半乳糖被报道与 *S. mutans* 生物被膜形成密切相关，虽然在缓症链球菌(*S. mitis*)和口腔链球菌(*S. oralis*)半乳糖均被认为能促进细菌生物被膜的形成，但半乳糖却能显著地抑制 *S. mutans* 生物被膜的形成[60]，表明半乳糖对链球菌生物被膜的影响具有种属选择性。在半乳糖对 *S. mutans* 生物被膜调控中，半乳糖作用于反义长链 RNA (antisense vicK RNA, ASvicK)转录后调控 VicRK 转录系统，抑制胞外多糖(exopolysaccharides, EPS)及生物膜的生成效率，并改变细菌毒力[61]。此外，*S. mutans* 还可以通过改变 LacR 与 6-磷酸-半乳糖形成的 LacR-DNA 复合体的生成效率调控对细菌活力和致病性至关重要的各种碳水化合物的代谢，以及通过转录调控因子 NagR 调控 *nagB* (编码葡糖胺-6-磷酸脱氨酶)与 *glmS* (编码谷氨酰胺-果糖-6-磷酸氨基转移酶)的表达并进一步通过介导包括葡萄糖转移酶(glucosyltransferase, GTF)、细胞表面蛋白抗原(cell surface protein antigen, Pac)、葡聚糖结合蛋白(glucan binding protein, Gbp)和乳酸在内的毒力因子的合成影响 *S. mutans* 的毒力[62-63]。近期的研究表明，*S. mutans* 半乳糖代谢还与细胞分裂密切相关[60,64]，但具体机制尚不清晰。

2.2 芽孢杆菌(*Bacillus*)

芽孢杆菌是一种自然界中常见的革兰氏阳性菌，对包括高温、高盐、酸碱、动物免疫等多种环境压力具有极强的抵御能力[65]。常见的致病性芽孢杆菌包括炭疽杆菌(*Bacillus*

anthracis)、蜡样芽孢杆菌(*Bacillus cereus*)、苏云金芽孢杆菌(*Bacillus thuringiensi*)[66]。半乳糖对芽孢杆菌的毒力调控主要通过影响细菌毒力相关分子的糖基化及生物被膜的生成 2 条途径完成[67-68]。半乳糖代谢异常还被报道会引起芽孢杆菌的自溶[69]。

如表 1 所示，在炭疽杆菌(*B. anthracis*)中，半乳糖能通过调控次生细胞壁多糖(secondary cell wall polysaccharide, SCWP)的半乳糖基化促进 *B. anthracis* 的增殖与细胞分裂[67,70]。敲除编码半乳糖代谢关键酶尿苷二磷酸葡萄糖-4-表异构酶的基因 *gelE* 后，*B. anthracis gelE* 突变株将无法利用 D 聚 γ 谷氨酸(poly-D-γ-glutamic acid, PDGA)合成荚膜，从而导致 *B. anthracis* 毒力的下降[71]。以上报道表明，正确的半乳糖基化是 *B. anthracis* 致病的关键因素。

表 1　半乳糖代谢对细菌毒力的影响及主要调控机制

Table 1　The effects of galactose metabolism on bacteria virulence and the main mechanism

Bacteria	Effect on virulence	Mechanism	References
Streptococcus pyogenes	Facilitation	(1) Inducing the expression of virulence genes (2) Supplying binding site for attachment (3) Supplying identification targets on host cells for the membrane localization of virulence factors	[39-44]
S. pneumonia	Facilitation	(1) Inducing the expression of transcriptional regulators (2) Activating QS system (3) Promoting CPS and biofilm formation	[45-54]
S. agalactiae	Facilitation	Promoting formation of α-2,3 CPS	[56-57]
S. suis	Facilitation	Promoting CPS formation	[58-59]
S. mutans	Suppression	Inhibiting biofilm and EPS formation	[60-61]
Bacillus anthracis	Facilitation	Maintaining precise glycosylation of SCWP and capsule	[67,70-71]
B. cereus	Suppression (strain specificity)	(1) Antagonizing the oligomerization and assembly of toxins (2) Antagonizing the dimerization of F-actin	[72-74]
B. subtilis *B. velezensis* *B. licheniformis*	Facilitation	Promoting biofilm formation	[68,75-76]
EHEC *Citrobacter rodentium*	Facilitation	Inducing the expression LEE pathogenicity island	[16]
Listeria monocytogene	Facilitation	(1) Maintaining precise assembly of WTA (2) Reconstructing the surface structure of cell membrane	[77-78]
Yersinia pestis	Facilitation	Promoting LPS formation	[79-80]
S. epidermidis	Suppression	Inhibiting biofilm formation	[81]
Idiomarina fontislapidosi	Facilitation	Promoting LPS formation	[82]
Erwinia amylovora	Facilitation	(1) Inducing the expression of transcriptional regulators (2) Promoting the process of amylovoran synthesis (3) Maintaining precise assembly of LPS	[83]
Ralstonia solanacearum	Facilitation	Activating QS system	[84]
Xanthomonas oryzae	Facilitation	Inducing the expression of transcriptional regulators	[85]
Salmonella spp.	Facilitation	Maintaining precise assembly of LPS	[86]
Pseudomonas aeruginosa	Facilitation	Activating QS system	[87]

在蜡样芽孢杆菌(*B. cereus*)中，半乳糖能抑制 *B. cereus* 毒力，但这种抑制作用具有显著的菌株选择性，例如对 *B. cereus* B10502 及 M2 菌株有毒力调控作用，但对 *B. cereus* T1 和 T2 菌株的生物活性无影响[88]。半乳糖对 *B. cereus* 毒力的抑制作用被认为可能是通过拮抗 *B. cereus* 毒素单体同型聚集体的寡聚化和组装导致在感染过程中 *B. cereus* 无法在靶标细胞膜表面成孔，或者是通过拮抗 *B. cereus* 对靶标细胞 F-肌动蛋白(F-actin)的去二聚化导致细菌效应物质无法有效地运输到细胞中[72-74]。

在枯草芽孢杆菌(*Bacillus subtilis*)、贝莱斯芽孢杆菌(*Bacillus velezensis*)、地衣芽孢杆菌(*Bacillus licheniformis*)中，半乳糖都被发现能显著促进生物被膜的生成效率并调控细菌毒力，但半乳糖并不参与苏云金芽孢杆菌(*Bacillus thuringiensis*)生物被膜的合成过程[68,75-76]。

2.3 其他细菌

如表 1 所示，在肠出血性大肠杆菌(enterohemorrhagic-*Escherichia coli*, EHEC)及啮齿枸橼酸杆菌(*C. rodentium*)中[16]，半乳糖代谢中间产物半乳糖酸和半乳糖醛酸能通过 ExuR 转录调控因子调控肠细胞脱落位点(locus of enterocyte effacement, LEE)毒力岛的表达影响 EHEC 及 *C. rodentium* 的毒力。在单核增生李斯特氏菌(*Listeria monocytogenes*)中，半乳糖通过调控壁磷壁酸(wall teichoic acid, WTA)的正确组装影响 *L. monocytogenes* 毒力及细胞间物质传播效应[77]，还可以通过调节半乳糖代谢酶的表达，改变细菌表面结构，从而增强 *L. monocytogenes* 对宿主免疫系统的逃避能力[78]。在鼠疫耶尔森氏菌(*Yersinia pestis*)中，半乳糖转运及代谢被发现与细菌的脂多糖(lipopolysaccharide, LPS)合成及在哺乳动物中的致病性有直接关联[79-80]。在表皮葡萄球菌(*Staphylococcus epidermidis*)中，半乳糖能抑制生物被膜的生成，因此被认为是抗生物膜药物的作用靶点[81]。在海洋细菌石泉海源菌(*Idiomarina fontislapidosi*)中，半乳糖能通过参与脂多糖 LPS 的合成参与 *I. fontislapidosi* 毒力的调控[82]。在梨火疫病菌解淀粉欧文氏菌(*Erwinia amylovora*)中，半乳糖代谢被报道能通过调控 GalE 的表达影响细菌的毒力及荚膜代谢[83]。在引起植物细菌性青枯病的重要病原茄科罗尔斯通氏菌(*Ralstonia solanacearum*)中，半乳糖通过 *phc* QS 系统激活环脂肽类分子拉呋喃酮及拉斯托宁的合成分泌提高 *R. solanacearum* 毒力[84]。在水稻黄单胞菌(*Xanthomonas oryzae*)中，半乳糖通过 LysR 型转录调控因子 GamR 激活编码 III 型分泌系统的关键基因 *hrpG*、*hrpX* 的表达调控 *X. oryzae* 的毒力[85]。在沙门氏菌(*Salmonella*)中，由于半乳糖是 LPS O 抗原上的第一个糖基，因此半乳糖代谢能显著影响 *Salmonella* 毒力[86]。在布鲁氏菌属(*Brucella*)中，半乳糖代谢也被推测与细菌毒力密切相关[87]。在铜绿假单胞菌(*Pseudomonas aeruginosa*)中，半乳糖代谢的改变影响了其群体感应系统。当半乳糖代谢受到抑制时，群体感应信号分子的产生减少，导致生物膜形成受阻，细菌毒力下降[89]。此外，半乳糖代谢产物还被报道可通过抑制细菌果胶甲基酯酶(pectin methylesterase, PME)的表达影响多种细菌的致病性[90]。

3 总结与展望

半乳糖是一种能被生物利用的常见单糖，不仅作为重要碳源参与细菌生长和增殖，而且其代谢过程与细菌的毒力密切相关。不同细菌种的半乳糖代谢途径差异性也在细菌的致病性及宿主适应性中起着决定性作用。近年来，新的半乳糖代谢中间产物和调控机制被相继报

道，这些发现补充了现有细菌代谢网络中的空白，也为开发新的抗菌策略提供了可能。然而在这个快速发展的领域，由于代谢通路的复杂性和调控的精细性，半乳糖在细菌生理学及细菌-宿主互作过程中的功能还存在大量未知，例如是由代谢中间产物还是终产物发挥作用，以及调控毒力的具体分子机制并未被全面研究。尽管目前尚未有研究直接表明半乳糖代谢途径可以被用作细菌疫苗的开发靶点，但在多种细菌中半乳糖代谢被报道能直接影响细菌的增殖、侵袭、毒力及环境适应力，因此通过对不同细菌代谢途径中对细菌生长及毒力关键蛋白质或酶的筛选，可能为开发针对细菌代谢途径的抗菌疫苗提供新思路，但针对细菌代谢途径的疫苗开发需要精准验证作用靶点应与宿主细胞中充分不同，以避免交叉反应和对宿主的潜在危害。本综述阐述了半乳糖在细菌中普遍存在的代谢通路及在部分代表菌中对细菌毒力调控的分子机制，部分揭示了代谢通路在细菌致病性中扮演的多面性角色，以及如何影响细菌对宿主的适应性和生存策略。未来的研究应当集中在半乳糖代谢与其他生理过程的相互作用，以及代谢通路与毒力基因表达的详细联系。此外，随着抗生素耐药性的日益严重，深入了解细菌代谢通路对于开发针对特定代谢环节的替代疗法和疫苗具有重要的实际意义。这些研究将有助于加强对细菌感染的防治，提高公共卫生水平，为全球健康事业做出贡献。

作者贡献声明

郑佳佳：数据收集和处理及论文撰写；薛松、杨晴晴：数据收集和处理及参与论文讨论；方仁东：论文构思和设计、论文修改及参与论文讨论；曹雪峰：论文构思和设计、数据收集和处理、论文修改及参与论文讨论。

作者利益冲突公开声明

作者声明不存在任何可能会影响本文所报告工作的已知经济利益或个人关系。

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