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Review 综述

共生微生物对昆虫脂质代谢的影响

王争艳*，张洁，张闪，周丽贞，罗琼

河南工业大学 粮食和物资储备学院，河南 郑州

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摘　要：共生微生物与昆虫之间的相互作用对昆虫的生长、发育和繁殖具有至关重要的作用。本文重点阐述共生微生物如何通过复杂的信号通路来调控昆虫的脂质代谢。共生微生物通过多种机制影响昆虫的脂质代谢，不仅为宿主提供类固醇等脂质或脂质前体，还通过产生短链脂肪酸和激活免疫信号通路，来间接影响宿主的胰岛素信号通路，进而改变昆虫体内的脂质含量。此外，共生微生物还能通过激活雷帕霉素靶标蛋白和激脂激素信号通路来调节昆虫的脂质代谢过程。深入研究这些信号通路在不同昆虫种类中的共性与差异，对于理解昆虫的生态适应性和繁殖策略以及开发新的害虫治理策略具有重要意义。

关键词：昆虫；共生微生物；脂质代谢；信号通路

Effects of microbial symbionts on lipid metabolism in insects

WANG Zhengyan*, ZHANG Jie, ZHANG Shan, ZHOU Lizhen, LUO Qiong

School of Food and Strategic Reserves, Henan University of Technology, Zhengzhou, Henan, China

Abstract: Interactions between microbial symbionts and insects are essential for the growth, development, and reproduction of insects. This review focuses on how microbial symbionts regulate lipid metabolism in insects *via* signaling pathways. Microbial symbionts affect lipid metabolism in insects through a variety of mechanisms. Microbial symbionts provide lipids or lipid precursors such as steroids to their insect hosts. Microbial symbionts can also modulate host insulin signaling pathway by producing short-chain fatty acids or activating immune signaling pathways, thereby changing the lipid content of insects. In addition, microbial symbionts can activate target of

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*Corresponding author. E-mail: zywang@haut.edu.cn
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rapamycin and adipokinetic hormone signaling pathways to regulate lipid metabolism in insects. Further research in the similarities and differences of these signaling pathways in different insect species is of great significance for comprehension of insect ecological adaptability and reproductive strategies, and development of new pest management strategies.
Keywords: insect; microbial symbiont; lipid metabolism; signaling pathway

脂质是脂肪和类脂及其衍生物的总称。脂肪是指由甘油和脂肪酸化合而成的甘油三酯；类脂是指结构或物理性质与脂肪相似的物质，主要包括磷脂、糖脂以及类固醇[其中类固醇涵盖麦角固醇、固醇(也被称为甾醇)、24-亚甲基胆固醇和胆固醇][1]。脂质在昆虫的生命活动中扮演着至关重要的角色，它们不仅是昆虫能量贮存的主要形式，还是合成激素的前体物质，同时也是细胞膜和卵黄原蛋白的重要构成成分，广泛参与昆虫体内的多种生理生化过程[2]。

共生微生物广泛存在于昆虫体内及体表，涵盖了细菌、真菌及古细菌等多个类群[3]。在长期的协同进化历程中，昆虫与这些共生微生物之间建立起了稳定的共生关系[4]。昆虫为共生微生物提供营养，而共生微生物则通过直接或间接的方式参与昆虫的生理生化过程[5-6]。其中，共生微生物与昆虫脂质代谢之间的关联主要体现在为昆虫提供营养供给和调节其代谢过程2个方面。例如，共生类酵母菌或酵母菌能够向昆虫提供包括脂肪酸、胆固醇、二氢胆固醇、7-脱氢胆固醇和麦角固醇在内的多种关键脂质或脂质前体[7-8]；共生蓝变菌(*Sporothrix* sp.) 1提供的棕榈油酸能够提升松材线虫(*Bursaphelenchus xylophilus*)的繁殖能力[9]；而豌豆蚜(*Acyrthosiphon pisum*)在感染共生沙雷氏菌(*Serratia symbiotica*)后，其脂肪体中的脂肪酸合成酶和二酰基甘油酰基转移酶表达水平显著上调，进而合成更多的甘油三酯，增强了豌豆蚜对冷热胁迫的耐受性[10]。

目前，已有研究初步揭示了共生微生物能够通过调控胰岛素信号通路和激活免疫通路来影响黑腹果蝇(*Drosophila melanogaster*)的脂质代谢。当果蝇感染发光光杆状菌(*Photorhabdus luminescens*)后，其胰岛素信号通路中的真核翻译起始因子4E结合蛋白(eukaryotic initiation factor 4 binding protein, 4E-BP)基因和蜕皮激素诱导基因*Impl2* (*ecdysone-inducible gene l2*)的表达水平显著上调，这导致转录因子FoxO发生核易位，并促进脂肪酶的表达，从而降低果蝇体内的脂质含量[11]。粪肠球菌(*Enterococcus faecalis*)会激活果蝇的Toll信号通路，导致果蝇脂肪体中脂肪酸合成酶和二酰甘油酰基转移酶的表达水平下调，进而减少脂肪体内的甘油三酯含量[12]。然而，共生微生物调控宿主脂质代谢的机制因共生体系的不同而有所差异，因此需要更加深入的研究来全面解析这些相互作用[13]。本文旨在综述共生微生物与昆虫脂质代谢之间的联系及其潜在机制，以期为深入研究共生微生物对昆虫生理功能的影响提供参考。

1 共生微生物为宿主提供脂质

共生微生物能够为昆虫提供脂质或脂质前体，如类固醇和脂肪酸等，这些物质在昆虫的生长、发育及繁殖过程中发挥着至关重要的营养供给和信号传导作用[14-15]。甾醇是昆虫进行蜕皮、生长和发育所不可或缺的营养素，然而，昆虫自身并不具备合成甾醇的能力，因此必须从食物或共生微生物中获取这一关键营养素[16]。烟草甲(*Lasioderma serricorne*)、药材甲(*Stegobium paniceum*)、褐飞虱(*Nilaparvata*

lugens)和无刺蜜蜂(*Scaptotrigona depilis*)体内的类酵母菌或酵母菌能够为宿主提供麦角固醇[8]。进一步地，褐飞虱体内的类酵母菌还能将麦角固醇转化为 24-亚甲基胆固醇，并最终合成胆固醇[17]。此外，在白蚁的肠道中，拟杆菌和厚壁菌负责将多糖降解为单糖，并将其转运至细胞内[18]。这些单糖在细胞内进一步被降解为丙酮酸，随后通过乙酰辅酶 A 和 Wood-Ljungdahl 途径产生乙酸，为宿主合成胆固醇和脂肪酸提供底物[19]。

2 共生微生物调控昆虫脂质代谢

2.1 IIS 通路介导的调控

在胰岛素/胰岛素样生长因子信号(insulin/insulin-like growth factor signaling, IIS)通路中，胰岛素分泌细胞产生的胰岛素样肽(insulin-like peptides, ILPs)与细胞膜上的胰岛素受体结合，引发细胞内的级联反应，激活下游激酶如磷脂酰肌醇 3-激酶(phosphatidylinositol 3-kinase, PI3K)和蛋白激酶 B (protein kinase B, Akt)，从而影响下游过程，包括抑制叉头转录因子 O 家族(fork head transcription factor O, FoxO)的核易位、激活固醇调节元件结合蛋白(sterol regulatory element-binding protein, SREBP)，进而诱导果蝇脂肪体合成甘油三酯[20]。当 IIS 通路减弱时，FoxO 被去磷酸化并进入核内，激活下游靶基因，启动 *brummer* 的表达，进而催化甘油三酯分解[21] (图 1)。

共生微生物能激活昆虫的 IIS 通路[22]。当黑腹果蝇感染果实醋杆菌(*Acetobacter pomorum*)、植物乳杆菌(*Lactobacillus plantarum*)或嗜昆虫假单胞菌(*Pseudomonas entomophila*)后，其脂肪体内的 IIS 通路被激活，Akt 磷酸化水平增加，同时 FoxO 靶基因表达水平降低[23-24]。然而，一些研究得出了相反的结论。例如，当黑腹果蝇感染海分枝杆菌(*Mycobacterium marinum*)或球孢白僵菌(*Beauveria bassiana*)后，其脂肪体内的 Akt 磷酸化水平降低，而 FoxO 靶基因的表达水平上调[25-26]。这些结果说明，共生微生物对宿主 IIS 通路的影响因微生物种类的不同而有所差异，也意味着共生微生物能通过调控 IIS 通路来影响宿主的脂质代谢。

进一步研究发现，共生微生物通过产生乙酸、丙酸和丁酸等短链脂肪酸来激活 IIS 通路，进而影响昆虫体内的脂质代谢[24,27]。例如，黑腹果蝇肠道内的路氏肠杆菌(*Enterobacter ludwigii*)、果实醋杆菌和植物乳杆菌产生的乙酸，可以促进 DILP2 和 DILP5 的表达，从而激活 IIS 通路，导致果蝇体内甘油三酯含量增加[28-29]。与此相反，病原菌可以刺激宿主消耗乙酸，进而抑制 IIS 通路。例如，当黑腹果蝇感染病原菌霍乱弧菌(*Vibrio cholerae*)后，其乙酰辅酶 A 合成酶-1 基因的表达水平上调[30]，导致乙酸消耗量增大，肠道中乙酸水平下降，从而抑制 IIS 通路，干扰宿主的肠道脂质代谢[31-32] (图 2)。

2.2 IMD/IMD-IIS 信号通路介导的调控

免疫缺陷(immune deficiency, IMD)信号通路在黑腹果蝇体内(包括肠道和脂肪体)发挥着重要的免疫调节作用，参与抵御革兰氏阴性菌以及部分革兰氏阳性菌的感染[33]。当细菌细胞壁中的二氨基庚二酸型肽聚糖与宿主细胞膜上的肽聚糖识别受体(peptidoglycan recognition protein LC, PGRP-LC)结合后，IMD 蛋白开始募集 FADD 蛋白(fas-associated death-domain-containing protein)和 dREDD 蛋白(death-related ced-3/Nedd2-like protein)形成复合体，并通过蛋白激酶 TAK1 (transforming growth factor-activated kinase 1)来激活 IκB 激酶(inhibitor of κB kinase, IKK)复合体，活化的 IKK 复合体激活

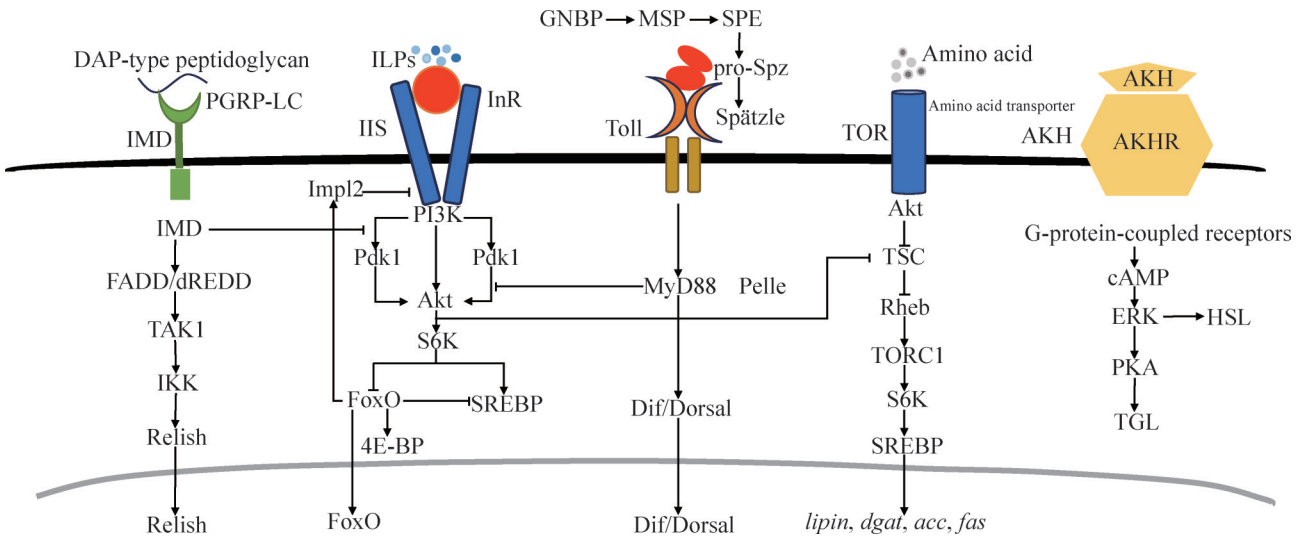

图1　昆虫主要的免疫信号通路和胰岛素信号通路及其相互作用

Figure 1　Major immune and insulin pathway signaling in insects and their interactions. Arrows depict activation while bars represent suppression in molecular interactions. 4E-BP: Eukaryotic initiation factor 4 binding protein; AKH: Adipokinetic hormone; AKHR: Adipokinetic hormone receptor; Akt: Protein kinase B; cAMP: Cyclic adenosine monophosphate; Dif/Dorsal: Dorsal-related immunity factor; dREDD: Death-related ced-3/Nedd2-like protein; ERK: Extracellular signal-regulated kinase; FADD: Fas-associated death-domain-containing protein; FoxO: Fork head transcription factor O; GNBP: Gram-negative binding protein; HDAC4: Histone deacetylase 4; HSL: Hormone-sensitive triglyceride lipase; IIS: Insulin/insulin-like growth factor signaling; IKK: Inhibitor of κB kinase; ILPs: Insulin-like peptides; IMD: Immune deficiency; *Impl2*: Ecdysone-inducible gene l2; InR: Insulin receptor; MSP: Modular serine protease; MyD88: Myeloid differentiation factor 88; Pdk1: Serine/threonine kinase 3-phosphoinositide-dependent protein kinase 1; PI3K: Phosphatidylinositol 3-kinase; PKA: Protein kinase A; Rheb: Ras homolog enriched in brain; pro-Spz: Pro spätzle; S6K: Ribosomal S6 kinase; SPE: Spätzle-processing enzyme; SREBP: Sterol regulatory element-binding protein; TAK1: Transforming growth factor-activated kinase 1; TGL: Triglyceride lipase; TOR: Target of rapamycin; TORC1: TOR complex 1; TSC: Tuberous sclerosis tumor suppressor.

转录因子 Relish，促使 Relish 发生核易位，并启动抗菌肽的表达[34] (图 1)。

共生微生物可以通过激活昆虫体内的 IMD 信号通路来调控速激肽(tachykinin, Tk)的表达。例如，当黑腹果蝇感染果实醋杆菌后会激活 IMD 信号通路，促进果蝇中肠内分泌细胞合成速激肽[35]。速激肽会与 G 蛋白偶联受体 TKR99D/PKA (protein kinase A)信号传导途径相互作用，抑制肠壁细胞中 SREBP 的表达，从而抑制肠道内脂肪酸合成酶和乙酰辅酶 A 羧化酶的表达，进而抑制脂肪合成[36]。据此推测，共生微生物可以通过 IMD-Tk 途径来调控宿主的脂质代谢。此外，共生微生物还能通过激活 IMD 信号通路来调控 IIS 通路，影响昆虫体内脂质代谢。例如，当黑腹果蝇感染病原菌霍乱弧菌或海分枝杆菌后，激活的 IMD 信号通路会通过抑制 Pdk1 (serine/threonine kinase 3-phosphoinositide-dependent protein kinase 1)来降低 IIS 通路的活性，导致 FoxO 的关键靶基因 *Impl2* 和 *4E-BP* 的表达上调，而 *Impl2* 的过表达会进一步抑制 IIS

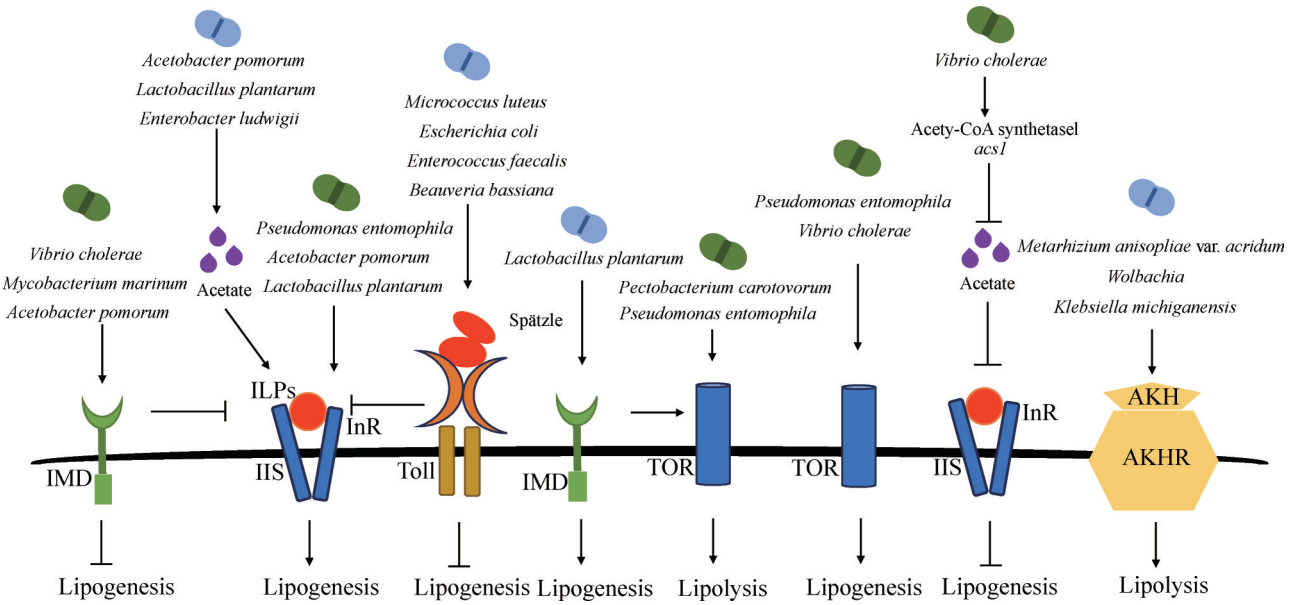

图2　共生微生物对昆虫脂质代谢的影响

Figure 2　Effects of microbial symbionts on lipid metabolism in insects. Arrows depict activation while bars represent suppression in molecular interactions. AKH: Adipokinetic hormone; AKHR: Adipokinetic hormone receptor; IIS: Insulin/insulin-like growth factor signaling; ILPs: Insulin-like peptides; IMD: Immune deficiency; InR: Insulin receptor; TOR: Target of rapamycin.

通路的活性，从而导致果蝇脂肪体中的脂质含量下降[20,25,37] (图 2)。

2.3　Toll/Toll-IIS 信号通路介导的调控

在昆虫肠道和脂肪体中，真菌和革兰氏阳性菌感染会激活 Toll 信号通路。革兰氏阴性菌结合蛋白(Gram-negative binding protein, GNBP)受体识别病原菌细胞壁中的赖氨酸型肽聚糖，进而激活模块化丝氨酸蛋白酶(modular serine protease, MSP)的级联反应[38]。这一级联反应通过由 Spätzle 激活酶和 Spätzle 加工蛋白酶介导的裂解过程，使下游配体 Spätzle 产生活性，随后配体 Spätzle 与 Toll 受体结合[39]，进而激活接头蛋白 MyD88 和 Pelle，促进转录因子 Dif/Dorsal 的核易位，最终启动载脂蛋白基因表达[40] (图 1)。例如，家蚕(*Bombyx mori*)和黄粉虫(*Tenebrio molitor*)在感染病原菌如球孢白僵菌、单核增生李斯特氏菌(*Listeria monocytogenes*)或大肠埃希氏菌(*Escherichia coli*)后，可以激活 Toll 信号通路，启动载脂蛋白 III 转录，从而促进宿主脂质的转运[38,41-42]。

共生微生物还可以通过激活 Toll 信号通路来抑制 IIS 通路，进而影响昆虫体内的脂质代谢。黑腹果蝇感染藤黄微球菌(*Micrococcus luteus*)、大肠埃希氏菌[26]、粪肠球菌[43]或球孢白僵菌后，会激活脂肪体中 Toll 信号通路中的接头蛋白 MyD88[44]，MyD88 通过作用于 PI3K 或 PI3K 下游蛋白激酶 Pdk1 来降低 Akt 的磷酸化水平，同时 Toll 信号通路还会降低 DILP6 的表达。IIS 通路的减弱会活化 FoxO，进而促进 *brummer* 的表达，导致昆虫脂肪体中脂质含量降低[21,26,45] (图 2)。

2.4　TOR/TOR-IIS 信号通路介导的调控

雷帕霉素靶标蛋白(target of rapamycin, TOR)

信号通路在调节昆虫的营养和能量可用性、生长因子信号以及免疫反应等方面起着关键作用，通过促进昆虫体内的脂质合成进而调控宿主细胞的生长、增殖和新陈代谢[46]。TOR 信号通路可以被氨基酸激活。结节性硬化症蛋白质复合体(tuberous sclerosis complex, TSC)会诱导 Ras 蛋白脑组织同源类似物(Ras homolog enriched in brain, Rheb)失活，进而抑制 TOR 复合体 TORC1 的形成，而 TORC1 可以激活 S6 激酶(ribosomal S6 kinase, S6K)和 SREBP，从而促进脂质合成[47] (图 1)。此外，IIS 通路中活化的 Akt 会抑制 TSC 复合体的形成，进而上调 TOR 信号通路。

共生微生物能够通过调控 TOR 信号通路来影响宿主体内的脂质代谢。例如，黑腹果蝇感染胡萝卜软腐坚固杆菌(*Pectobacterium carotovorum*)或嗜昆虫假单胞菌后，会激活肿瘤坏死因子受体相关因子蛋白 3/Warts 激酶信号传导途径，抑制黑腹果蝇体内的 Akt 磷酸化水平和 TOR 信号通路，进而抑制 S6K 以及 SREBP，并激活宿主体内的丝氨酸/苏氨酸激酶 ATG1，从而促进昆虫肠道的脂质分解[48]。一些研究得出了相反的结论，如当黑腹果蝇感染霍乱弧菌或嗜昆虫假单胞菌后会激活昆虫的 TOR 信号通路，导致 S6K 和 SREBP 被激活，使肠道中的脂质合成基因如 *lipin*、*dgat*、*acc* 和 *fas* 的表达水平上调[23,49]。

另一项研究发现，黑腹果蝇感染植物乳杆菌后会激活 IMD 信号通路，导致转录因子 Relish 激活肠壁细胞中肽酶基因 *jon66Ci* 和 *jon66Cii* 的表达，导致肠道肽酶活性升高，从而促进宿主对蛋白质的消化来增加体内游离氨基酸的含量，增强了 TOR 信号通路，激活 S6K 和 SREBP，同时也会进一步上调 DILP 的表达，共同促进宿主的脂质合成[24,50-52] (图 2)。

2.5 AKH 信号通路介导的调控

昆虫的脂质代谢受激脂激素(adipokinetic hormone, AKH)信号通路控制[53]。AKH 由昆虫心侧体腺细胞合成、储存，并释放到血淋巴中，当与激脂激素受体(adipokinetic hormone receptor, AKHR)结合后，AKHR 与 G 蛋白偶联增强，进而增强腺苷酸环化酶的活性，导致细胞内第二信使环磷酸腺苷(cyclic adenosine monophosphate, cAMP)的含量增加，引起细胞外调节蛋白激酶(extracellular signal-regulated kinase, ERK)发生磷酸化，从而激活 PKA[54]，使下游的甘油三酯酯酶(triglyceride lipase, TGL)发生磷酸化，从而催化脂肪体内的甘油三酯降解为甘油二酯，甘油二酯进入血淋巴后，经由载脂蛋白运输至昆虫的飞行肌，最终氧化产生能量[55] (图 1)。

共生微生物能够调控 AKH 信号通路促进昆虫体内的脂质分解。例如，当沙漠蝗(*Schistocerca gregaria*)感染金龟子绿僵菌(*Metarhizium anisopliae* var. *acridum*)或灰飞虱(*Laodelphax striatellus*)感染 *Wolbachia* 后，会通过激活 AKH 信号通路来促进脂肪体和血淋巴中的甘油三酯降解为甘油二酯[56-57]。进一步的研究发现，当橘小实蝇的肠道感染密歇根克雷伯氏菌(*Klebsiella michiganensis*)后，会导致宿主神经肽抑咽侧体素 A 型(allatostatin, Ast-A)基因的表达水平下降[58]，而 *Ast-A* 基因通常通过刺激昆虫的 AKH 分泌细胞来产生 AKH[59]，在 AKH 信号通路中，激活的 ERK 会导致激素敏感性脂肪酶(hormone-sensitive triglyceride lipase, HSL)的 Ser600 位点发生磷酸化，从而催化宿主脂肪体中的脂质分解[60] (图 2)。然而，目前关于共生微生物如何调控神经肽表达的机制尚不明确。

3 总结与展望

昆虫的脂质代谢与其环境适应性和繁殖能力密切相关，共生微生物在昆虫宿主的脂质代谢中扮演着至关重要的角色。这些微生物能够为昆虫提供类固醇、脂肪酸等物质，以满足其生存和繁殖的需求。此外，共生微生物还通过产生短链脂肪酸激活 IIS 通路，通过 IMD 和 Toll 信号通路调控 Akt 的表达水平，通过 TOR 信号通路调节脂质基因表达水平以及通过调控神经肽基因表达水平影响 AKH 信号通路来影响昆虫的脂质代谢。深入研究这些信号通路在不同昆虫种类中的共性与差异，有助于揭示昆虫脂质代谢的调控机制。

在未来的研究中，可以进一步探索共生微生物产生的脂质代谢物在建立和维持共生关系中的作用，如进一步明确棕榈油酸在松材线虫与蓝变菌共生关系中的作用[9]。在微观层面，共生微生物如何调控宿主信号通路也存在很多未知，如尚需进一步研究共生微生物产生的乙酸如何调控宿主的 IIS 通路[31]，以及感染共生微生物后宿主免疫信号通路如何调控脂质代谢[45]。因此，对共生微生物如何调控昆虫脂质代谢仍有许多未知，需要进一步深入研究。

深入理解共生微生物对宿主脂质代谢的影响及其机制，对于开发新型病虫害防治策略至关重要。可以直接利用影响宿主脂质代谢的共生微生物及其产生的次级代谢物，开发新型农药或生物制剂[61]，以干扰昆虫脂质代谢来影响其繁殖能力。例如，利用肠道微生物调节昆虫卵黄原蛋白合成可以有效抑制害虫的繁殖，从而达到防治害虫的目的[62]。此外，共生微生物的研究涉及生物学、医学和环境科学等多个领域[63]，深入理解其在昆虫脂质代谢中的作用，不仅能丰富基础生物学知识，还能为医学研究和环境保护提供新思路。

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作者利益冲突公开声明

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