



蜜蜂寿命可塑性与衰老调控机制研究进展*

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摘要 蜜蜂作为典型的社会性昆虫, 其同源基因组的蜂王和工蜂表现出显著寿命差异, 为研究寿命可塑性及衰老调控机制提供了独特模型。本文系统综述了端粒长度与端粒酶活性、表观遗传调控机制、胰岛素信号通路、能量代谢和氧化应激、肠道微生物、环境压力等在蜜蜂寿命可塑性及衰老调控中的最新研究进展。与工蜂相比, 蜂王的端粒酶活性、卵黄蛋白原水平更高, H3K27ac 修饰更多, 线粒体功能更稳定, 保幼激素水平、甲基化水平、胰岛素信号通路活性更低, 同时其特有的肠道菌群通过抑制胰岛素信号通路, 激活抗氧化防御机制, 使蜂王寿命得到显著延长。未来可应用最新测序技术挖掘蜜蜂寿命可塑性与衰老新型调控节点和生物标志物, 探讨环境压力与内在衰老机制的交互作用, 为深入理解衰老的生物学本质提供新的理论视角。

关键词 蜜蜂; 端粒; 表观遗传调控; 胰岛素信号通路; 肠道微生物

Progress in research on lifespan plasticity and aging regulation in honeybees

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Abstract As in other social insects, there are significant differences in the lifespans of honeybee queens and workers, which provide a unique model for studying lifespan plasticity and aging regulation mechanisms in insects. This paper systematically reviews recent advances in research on the lifespan plasticity and age regulation of honeybees, focusing on telomere length, telomerase activity, epigenetic mechanisms, insulin signaling pathways, energy metabolism and oxidative stress, gut microbiota, and environmental stressors. Compared to workers, queen bees have higher telomerase activity and vitellogenin levels, increased H3K27ac modifications, and more stable mitochondrial function. They also have lower levels of juvenile hormone, methylation, and insulin signal pathway activity. In addition, the unique gut microbiota of queen honeybees significantly extends their lifespan by suppressing the IIS pathway and activating antioxidant defense mechanisms. The latest sequencing technology can allow the investigation of new regulatory nodes and biomarkers of honeybee lifespan plasticity and aging, the interaction between environmental stress and intrinsic aging mechanisms, and provide a new theoretical perspective for a deeper understanding of the biology of aging.

Key words honeybee; telomere; epigenetic regulation; insulin signaling pathway; gut microbiota

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蜜蜂作为重要授粉昆虫,在促进农作物增产和维持生态平衡方面发挥关键作用(曾志将, 2020; 江慧枝等, 2025)。在蜜蜂社会中,蜂王寿命可达 2-3 年,而工蜂在春夏季繁殖期寿命仅为 3-6 周,越冬期则可延长至数月(Page and Peng, 2001; Corona *et al.*, 2005)。蜂王与工蜂两个雌性级型间寿命差异显著,同时工蜂寿命也有高度可塑性。具有生殖能力的蜂王与主要承担非繁殖任务的工蜂,均由相同的受精卵发育而来,其寿命具有弹性,为研究生物体在细胞、个体和超级个体三个层级的衰老过程提供了独特范本(Aamodt, 2009; 金梦杰和曾志将, 2023)。

衰老是生物体随时间推移而发生的功能渐进性衰退过程,统计学上体现为死亡风险升高和繁殖成功率降低(Čapková Frydrychová, 2023; Min *et al.*, 2024; Kroemer *et al.*, 2025)。衰老分为内在衰老和外在衰老,两种机制相互关联。内在衰老指生物标志物的自然变化,包括细胞与分子层面的改变、遗传因素及激素变化等,外在衰老则由环境压力、饮食习惯、氧化应激等加速生理衰老的因素所调控(Min *et al.*, 2024),其中氧化应激不仅是对外部环境损伤的被动反应,更是活性氧作为细胞内信号分子参与多种生理调控过程的内生分子机制(Finkel and Holbrook, 2000; Zhu *et al.*, 2022)。随着分子生物学技术的革新与发展,发现蜜蜂显著的寿命二态性受内在机制和外在因素共同影响(Amdam and Omholt, 2002; Corona *et al.*, 2005, 2007; Remolina *et al.*, 2007; Münch *et al.*, 2008; Koubová *et al.*, 2021)。本文从蜜蜂衰老理论的发展、内在调控机制、外在影响因素等方面系统梳理蜜蜂寿命可塑性与衰老研究领域的最新进展,并展望未来的研究方向与策略。

1 蜜蜂衰老理论

蜜蜂衰老研究起源于对其社会组织结构的观察。早期研究建立了“营养-内分泌-基因表达”的级型分化理论框架(陈伟轩等, 2024),雌性幼虫持续摄入蜂王浆可诱导其发育为蜂王,形成长寿命、高繁殖力表型,而摄入 3 d 蜂王浆后转

为摄入天然蜂粮,则发育为短寿命的工蜂(Page and Peng, 2001; Chittka and Chittka, 2010)。蜂王浆中的保幼激素(Juvenile hormone, JH)是关键调节因子(Page and Peng, 2001; Amdam and Omholt, 2003),与蜜蜂寿命呈负相关。蜂王维持较低 JH 水平伴随着较长的寿命。采集蜂 JH 浓度较哺育蜂显著升高。JH 与卵黄蛋白原(Vitellogenin, Vg)之间也存在互相抑制的关系,高 Vg 水平对应低 JH 水平。此外, JH 对胰岛素信号(Insulin insulin-like signaling, IIS)通路的基因表达有正向影响,蜂王在 IIS 信号下调时还能保持高生殖力(Corona *et al.*, 2007; Remolina and Hughes, 2008)。

目前对蜜蜂级型分化发育的分子机制已有较为深入的研究,然而,对其寿命可塑性调控机制的认识仍相对有限。早期有 3 种主要理论对衰老现象的成因提供了互补性解释,突变积累理论(The mutation accumulation theory)指出,中性或弱有害突变可在世代间积累,这些突变在个体发育及成年早期阶段不造成显著影响,在繁殖期后才开始显现有害影响(Amdam and Omholt, 2002; Rando and Wyss-Coray, 2021; Li *et al.*, 2023);拮抗多效性理论(Antagonistic pleiotropy theory)强调,某些基因能同时调控多个性状,在生命早期能促进繁殖、增强生存能力,在老年阶段则会加速衰老、诱发疾病(Williams, 1957; Amdam and Omholt, 2002),由于自然选择更倾向于早期繁殖成功率而非长期生存,所以这些早期有益晚期可能有害的基因得以在群体中保留;一次性体细胞理论(Disposable soma theory)认为生物体在生殖细胞与体细胞修复之间存在资源权衡,繁殖投入的增加会减少体细胞维护,从而缩短寿命(Kirkwood, 1977; Amdam and Omholt, 2002; Čapková Frydrychová, 2023; Li *et al.*, 2023)。

突变积累理论和拮抗多效性理论无法解释同一基因型却拥有不同寿命的兼性性状,将工蜂在群体层面视为体细胞,一次性体细胞理论可解释工蜂寿命的可塑性(Amdam and Omholt, 2002)。然而,单个蜜蜂的平均寿命对蜂群的生存至关重要,工蜂寿命缩短会导致蜂群群势和蜂

蜜产量下降 (Nearman and VanEngelsdorp, 2022)。

2 蜜蜂衰老的内在调控机制

2.1 端粒长度与端粒酶活性

蜜蜂寿命缩短可能与端粒长度和端粒酶活性密切相关。在多数脊椎动物中, 端粒长度随年龄增长而缩短 (Grula *et al.*, 2024), 最短端粒会触发 DNA 损伤反应 (Lai *et al.*, 2018)。在蜜蜂中, 细胞中少数缩短的端粒就可能导致衰老, 造成工蜂的“端粒早衰综合征”, 进而引发蜂群崩溃 (Aubert and Lansdorp, 2008; Stindl and Stindl, 2010)。西方蜜蜂 *Apis mellifera* 的端粒重复序列为 (TTAGG)_n (Robertson and Gordon, 2006; Zhou *et al.*, 2022), 其长度从 2.3 kb 到超过 48.0 kb 不等 (Koubová *et al.*, 2021), 在蜂群的不同个体间 (无论是同一级型还是不同级型) 存在显著差异 (Korandová and Frydrychová, 2015; Koubová *et al.*, 2021), 但同一工蜂的大脑、腿部肌肉、马氏管和胸部等多组织端粒长度一致 (Korandová and Frydrychová, 2015), 在滋养细胞和脂肪细胞这类非增殖细胞中, 年轻蜂王与年老蜂王、新羽化工蜂与年老工蜂的端粒长度无显著差异 (Hsieh and Hsu, 2011a, 2011b)。此前研究只能反映平均或相对端粒长度 (Korandová and Frydrychová, 2015; Lai *et al.*, 2018; Koubová *et al.*, 2021; Grula *et al.*, 2024), 其主要受个体差异影响, 蜂王和工蜂端粒长度并无统计学上的显著差异 (Korandová and Frydrychová, 2015; Koubová *et al.*, 2021), 因此, 要明确端粒长度对蜜蜂衰老的指示作用仍需深入研究。

蜜蜂的端粒酶活性在不同发育阶段、不同级型和季节存在差异。端粒酶活性在蜜蜂胚胎期最高, 工蜂和雄蜂体细胞中的活性会随发育下降至较低水平, 蜂王的 3 日龄幼虫端粒酶活性恢复至胚胎期水平, 蛹末期蜂王卵巢和雄蜂精巢端粒酶活性显著上调。蜂王的端粒酶活性显著高于工蜂和雄蜂, 蜂王头部端粒酶活性比工蜂约高 15-70 倍 (Korandová and Frydrychová, 2015; Koubová and Čapková Frydrychová, 2021)。与其他季节相比, 冬季工蜂的端粒酶活性最高, 其脂肪体组织中

的端粒酶活性较夏季高出约 10 倍, 大脑和头部也分别有 2.5 和 3.0 倍的升高 (Koubová *et al.*, 2021)。年轻蜂王与年老蜂王、新羽化工蜂与年老工蜂的滋养细胞和脂肪细胞中端粒酶活性无显著差异, 表明这些细胞在成年后不再分裂 (Hsieh and Hsu, 2011a, 2011b)。同样是高端粒酶活性的蜂王卵巢和头部, 卵巢的 DNA 复制活性也高, 头部则不然, 证明端粒酶可能参与端粒维持之外的功能, 作为多效性调控因子参与线粒体功能调节或氧化应激防御, 帮助规避繁殖代价带来的寿命缩短 (Koubová *et al.*, 2021; Koubová and Čapková Frydrychová, 2021; Čapková Frydrychová, 2023)。

2.2 表观遗传调控机制

影响蜜蜂寿命可塑性与衰老的主要表观遗传机制包括 DNA 甲基化、非编码 RNA 和组蛋白修饰 (Vaiserman, 2015; Yan *et al.*, 2015; Hu *et al.*, 2025)。蜜蜂的 DNA 甲基化基因表达具有组织特异性 (Cardoso-Júnior *et al.*, 2018)。蜂王与工蜂大脑存在超过 550 多个差异甲基化基因 (Lyko *et al.*, 2010), 蜜蜂大脑中多个基因的 DNA 甲基化模式多样性较低, 年龄对甲基化模式的影响大于行为差异 (Kucharski and Maleszka, 2020)。蜂王幼虫甲基化水平先上升后下降, 工蜂幼虫则随年龄增长而上升 (Shi *et al.*, 2013)。与此相似, 反叛工蜂 DNA 甲基化水平也随年龄增长而升高, 整体 DNA 甲基化水平显著低于正常工蜂, 表现出类似蜂王的特征 (Paleolog *et al.*, 2021)。使用抑制剂 RG108 去甲基化可显著延长工蜂寿命, 该效应独立于保幼激素通路, DNA 甲基化可能与经典内分泌通路并行调控寿命 (Cardoso-Júnior *et al.*, 2018)。蜜蜂母体效应通过调节产卵策略, 改变卵中的营养储备, 进而调控后代 DNA 甲基化水平和模式, 特别是调控发育关键基因和信号通路上的甲基化, 实现对蜂王发育和级型分化的表观遗传调控 (He *et al.*, 2021)。其 DNA 甲基化还存在表观遗传印迹现象, 使用 2 日龄及以上幼虫人工育王会造成蜂王和后代雄蜂获得更多的高甲基化位点, 影响培育蜂王的质量, 进而降低整个蜂群繁衍 (李震等,

2022)。西方蜜蜂蜂王和工蜂的甲基化 CpG 位点主要集中在受可变剪接影响的基因外显子区域 (Lyko *et al.*, 2010)。Vg 基因的 DNA 甲基化可调控 Vg 基因表达, 进而调控蜜蜂寿命 (Cardoso-Júnior *et al.*, 2018)。

长链非编码 RNA (lncRNA)、微 RNA (miRNA) 等非编码 RNA 能在转录和翻译层面精细调控基因表达, 影响蜜蜂级型分化、学习记忆及环境应激反应等生理过程 (Hu *et al.*, 2025), *lncov1*、*lncov2* 调控卵巢发育和级型分化 (Humann *et al.*, 2013), *Nb-1* 调控工蜂劳动转型 (Tadano *et al.*, 2024), LOC102654625 在蜜蜂暴露于噻虫啉时参与调控毒性反应 (Fent *et al.*, 2020), lncRNA kakusei 调控热防御行为中的产热与温度感知抵御黄蜂攻击 (Kiya *et al.*, 2012)。长度为 22 nt 的 miRNA 在蜂王与工蜂间存在显著差异, 在 61 种已知 miRNAs 中, 17 种在蜂王幼虫中表达显著上调, 20 种显著下调 (Shi *et al.*, 2015)。

组蛋白修饰通过影响蜜蜂级型分化调控其寿命, 在 H2A、H2B、H3、H4 4 种核心组蛋白修饰类型中, H3 对基因表达的调控研究较为广泛 (Jin *et al.*, 2023)。组蛋白 H3 上的第 27 位赖氨酸残基乙酰化 (H3K27ac) (Wojciechowski *et al.*, 2018)、第 4 位赖氨酸残基甲基化 (H3K4me1) (Zhang *et al.*, 2023) 可能是蜜蜂级型分化的重要调控因子 (陈伟轩等, 2024), 新羽化蜂王的 H3K27ac 修饰数量远超工蜂, 工蜂则表现出更高的 H3K4me1 修饰水平 (Jin *et al.*, 2023)。

其他表观遗传调控机制包括可变剪接、基因表达等 (Vaiserman, 2015)。中华蜜蜂 *Apis cerana cerana* 的可变剪接富集在生长信号通路和免疫通路, 通过调控关键基因 *Vg6*、*CYP15A1*、*PTPRR* 的转录本多样性, 影响工蜂的发育进程、免疫状态和功能转换, 从而实现蜜蜂劳动分工的精细调控 (Yao *et al.*, 2025)。在幼虫早期敲除 *Vg* 基因显著抑制了其发育为成年蜂王 (Zhang *et al.*, 2022)。抑制 *Dnmt3* (DNA 甲基转移酶) 基因能使幼虫发育为类蜂王表型 (Kucharski *et al.*, 2008)。*Map3k15* (Jin *et al.*, 2023)、*MRJP3*、*SV2A*、*APD3* (Ma *et al.*, 2025) 等 16 个关键衰老基因

可为以后的蜜蜂衰老研究提供潜在分子标记。

以上表观遗传调控机制的协同作用尚未系统阐明, 如 DNA 甲基化时钟等在揭示衰老分子机制方面仍然存在局限, 构建针对特定组织、年龄段或表型的专用生物钟可能更有效 (Porter *et al.*, 2021)。

2.3 胰岛素信号通路

营养差异能通过胰岛素信号通路调控蜜蜂寿命, 老年蜂王头部胰岛素样肽及其潜在受体的表达量低于老年工蜂, 高营养状态时, IIS 活性降低, JH 水平下降, Vg 水平升高, 蜂王寿命延长, JH 对 IIS 的影响被假定通过 Vg 介导 (Corona *et al.*, 2007)。蜂王的 IIS 通路关键基因 *ILPI* 和 *InR* 表达比工蜂低, 同时, Vg 持续高表达 (Wang *et al.*, 2022)。Vg 含量随年龄增加但无显著季节差异 (Spremo *et al.*, 2024)。胰岛素信号传导是一个复杂的级联反应, 调控不同节点对通路输出强度和特异性的影响不同, 抑制其上游的受体底物 *IRS* 基因表达会促进工蜂行为成熟加快, 提前开始采集行为, 导致寿命缩短 (Ihle *et al.*, 2019), 抑制其下游的 TOR 通路, 可以延迟工蜂从哺育蜂向采集蜂转变的年龄 (Ament *et al.*, 2008), 进而延长寿命。同时抑制 *IRS* 和 *TOR* 基因表达, 幼虫体内 JH 水平显著降低, 双基因抑制组死亡率显著升高, 相关基因表达、蛋白质丰度及 DNA 甲基化水平显著变化, 导致典型工蜂表型。外源 JH 处理能够恢复蜂王特征, 确认 JH 位于 IIS/TOR 信号通路下游 (Mutti *et al.*, 2011)。Vg-JH 调控模块位于胰岛素样肽 1、AKHR 和 PKG 的上游, 主要调控碳水化合物代谢, 从而控制工蜂从哺育行为向采集行为转变 (Wang *et al.*, 2012)。目前胰岛素信号通路的分子调控网络和反馈机制并不完全明确。

2.4 能量代谢和氧化应激

蜂王具有更高的线粒体能量利用、能量调节、细胞降解与代谢活性。蜂王滋养细胞和绛色细胞中 cAMP/PKA (Cyclic AMP/Protein kinase A)、RAS/MAPK (Ras GTPase/Mitogen-activated protein kinase) 通路相关分子表达均明显高于工

蜂 (Hsu *et al.*, 2022), 并且老年蜂王这两类细胞的能量代谢状态与年轻蜂王相似 (Lu *et al.*, 2018)。此外, 蜂王滋养细胞和脂肪细胞中的能量调节分子如 AMPK 活性, ATP、ADP、AMP 浓度, ADP/ATP 和 AMP/ATP 比值, cAMP 浓度, FoxO 表达, SirT1 的表达及活性, PPAR- α 表达等能在衰老过程中保持年轻状态 (Hsu and Hu, 2014)。但工蜂衰老会伴随着这些细胞能量状态的显著衰退 (Hsu and Chuang, 2014)。自然衰老工蜂与瓦螨 *Varroa destructor* 感染的早衰工蜂, 在血淋巴及脂肪体不同节段的能量代谢关键环节上, 前者三羧酸循环 (Krebs 循环) 与呼吸链中代谢物浓度及酶活性高于后者, 早衰工蜂代谢效率更低 (Kunat-Budzyńska *et al.*, 2025)。冬季蜜蜂更倾向于利用琥珀酸和甘油-3-磷酸等线粒体底物产热, 而非夏季蜜蜂主要依赖的复合物 I (NADH 相关底物) 途径 (Cormier *et al.*, 2022)。

蜂王滋养细胞和绛色细胞中的线粒体功能维持比工蜂稳定。新蜂王和老蜂王的线粒体密度、线粒体膜电位 ($\Delta\Psi_m$)、 NAD^+ 、NADH、ATP 浓度、 NAD^+ /NADH 比值、ND1 和 ATP 合酶相对表达量均无显著差异 (Hsu and Lu, 2015)。相比之下, 工蜂的线粒体功能和能量代谢效率随年龄增长明显下降 (Chuang and Hsu, 2013)。蜂王通过维持较高 *COX-1*/*CytC* 比值优化呼吸链蛋白组成, 可能通过提高电子传递效率减少活性氧产生而实现长寿 (Corona *et al.*, 2005)。东方蜜蜂 *Apis cerana* 工蜂的线粒体损伤程度随年龄增长而加剧, 自噬体数量先增后减, 老年工蜂自噬效率下降导致异常线粒体积累, 从而加速衰老进程 (Ma *et al.*, 2025)。

年龄会影响蜜蜂的抗氧化能力, 蜂王与工蜂、野生蜂群和人工饲养蜂群的氧化应激防御机制不同。蜂王衰老过程中, 其抗氧化系统的调控较为复杂, 年轻蜂王抗氧化基因表达较高但随年龄增长快速下降 (Corona *et al.*, 2005), 年老蜂王滋养细胞和脂肪细胞中超氧化物歧化酶、过氧化氢酶等抗氧化酶活性随年龄显著上升, 推测代谢活动增强与氧化应激水平升高同步发生, 进而引发应激防御机制的强化, 促进蜂王长寿 (Hsieh

and Hsu, 2013)。工蜂存在内在的生理衰老 (Remolina *et al.*, 2007), 工蜂翅膀肌肉中与线粒体 DNA 氧化损伤修复相关的 *mrp23S/mth1* 融合基因和 *nth1* 基因在衰老过程中显著下调, 导致采集蜂线粒体 DNA 完整性减少约 50% (Aamodt, 2009)。年轻采集蜂经过飞行后, 飞行肌中的总抗氧化能力和过氧化氢酶活性显著提升, 且 Hsp70 水平增加, 显示飞行产生大量活性氧激活抗氧化反应, 年老采集蜂则失去这种抗氧化提升能力 (Williams *et al.*, 2008; Margotta *et al.*, 2018)。早期研究认为抗氧化反应主要发生于高代谢的飞行肌, 而不是头部组织 (Williams *et al.*, 2008), 但后期研究检测到采集蜂飞行肌和脑组织中的 DNA 氧化损伤标志物 8-羟基脱氧鸟苷和脂质过氧化产物丙二醛 (Malondialdehyde, MDA) 水平显著高于巢内工蜂 (Margotta *et al.*, 2018)。野生蜂群采集蜂寿命、脂质损伤水平显著高于人工饲养蜂群, 但蛋白质羰基损伤无显著差异, 野生蜜蜂可能通过耐受氧化应激机制, 而不是修复机制来维持较长寿命 (Ward *et al.*, 2022)。JH 和 Vg 也会对蜜蜂氧化应激产生影响, 高 JH 水平的蜜蜂易受氧化应激伤害, 但 Vg 基因的高表达能保护工蜂免受氧化应激损伤 (Seehuus *et al.*, 2006)。

从以上研究可以看出, 目前的研究对于滋养细胞、脂肪细胞、绛色细胞研究较多, 其他类型的细胞相关研究较少; 对不同蜂种、社会角色 (如越冬蜂) 的系统性研究较少; 线粒体功能相关通路的内在分子机制、组织 DNA 修复动态等有待进一步探索。

2.5 肠道微生物

肠道菌群与免疫系统、抗氧化机制、IIS 信号通路共同作用, 促进蜂王长寿。与工蜂相比, 蜂王体内含有更高丰度的醋杆菌科 *Acetobacteraceae* (尤其是共生杆菌属 *Commensalibacter* 和大黄蜂菌属 *Bombella*)、乳杆菌属 *Lactobacillus* 和双歧杆菌属 *Bifidobacterium*。蜂王的肠道生物标志物为肠大黄蜂菌 *Bombella intestini* (Wang *et al.*, 2022), 其可以改善蜜蜂食欲, 促进采食, 增加

蜂体营养储备,促进养分的分解代谢(张维等, 2023),也可以在幼虫低营养情况下为幼虫提供色氨酸抵抗营养压力(Jin *et al.*, 2025)。将蜂王肠道菌群移植给无菌工蜂可以抑制胰岛素信号传导,增强抗氧化能力,从而显著延长其寿命(Wang *et al.*, 2024)。肠道微生物在蜜蜂体内起作用的分子机制及其在压力源下的群落变化对蜜蜂健康的深远影响等仍未可知(刘建辉等, 2025)。

3 蜜蜂衰老的外在影响因素

农药暴露可能加剧与年龄相关的线粒体功能和抗氧化防御能力的下降,从而加速衰老(Ma *et al.*, 2025)。常见农药如新烟碱、有机磷、拟除虫菊酯等和病原体如微孢子虫 *Nosema ceranae*、瓦螨等会诱导活性氧过量产生,导致抗氧化酶活性失调和解毒功能下降,卵黄蛋白原含量也发生变化(微孢子虫 *N. ceranae* 感染后工蜂的 Vg 表达下降、蜂王则升高),两者的协同作用显著增加蜜蜂氧化损伤和死亡率(Olgun *et al.*, 2020)。连续摄入植物源杀虫剂苦参碱超过 9 d 会显著降低蜜蜂存活率,干扰肠道优势菌群(如 *Gilliamella*、*Dorea*、*Lachnospirillum*)丰度,解毒、免疫和代谢相关基因表达受胁迫后响应,关键代谢物如精氨酸琥珀酸和亚精胺上调,花生四烯酸及 *LOC107998471* (*Aldh*) 下调(Yu *et al.*, 2025)。

季节变化通过调控蜜蜂解毒酶活性、免疫关键酶和免疫蛋白的表达,影响蜜蜂对环境压力的应对能力。冬季工蜂的酚氧化酶(Phenol oxidase, POx)、前酚氧化酶(Prophenol oxidase, proPOx)、葡萄糖氧化酶(Glucose oxidase, GOx)活性较高,Vg 和防御素-1 的转录丰度显著增加,吞噬反应更强,血细胞活力更高,工蜂的免疫防御和抗氧化系统得到提升,从而延长寿命。夏季工蜂的谷胱甘肽 S-转移酶(Glutathione S-transferase, GST)活性较高,乙酰胆碱酯酶(Acetylcholine esterase, AChE)活性呈周期性波动,在夏季先升后降,证明夏季工蜂主要以增强解毒能力来应对环境毒素和农药暴露,寿命较短(Cormier

et al., 2022; Spremo *et al.*, 2024)。

转地饲养会导致工蜂寿命缩短和氧化应激水平增加(Simone-Finstrom *et al.*, 2016)。在无蜂王环境下,反叛工蜂具有较高繁殖潜力和更长的寿命(Kuszevska *et al.*, 2017)。额外提供水源能显著延长蜜蜂寿命(Cormier *et al.*, 2022)。

4 结论与展望

蜜蜂寿命缩短会导致蜂群群势和蜂蜜产量下降,研究揭示蜜蜂衰老规律,不仅对传粉昆虫保护和生态系统维持具有迫切现实意义,也为理解人类衰老本质和探索健康老龄化策略提供重要启示。目前已初步揭示其衰老进程受众多内在机制和外在因素共同影响,但最短端粒对蜜蜂衰老的影响、表观遗传调控机制如何协同、胰岛素和线粒体功能通路分子机制、微生物-宿主互作途径、自然环境中多因素协同对衰老的影响等尚不明确,未来可加强在自然和半自然条件下的研究,应用最新测序技术研究蜜蜂衰老分子机制及协同作用,探讨环境压力(如农药、气候变化)与内在衰老机制的交互影响。

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