

蜂蜜抗氧化活性和抗衰老功能研究进展*

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摘 要 蜜蜂是最重要授粉昆虫, 也是人类生产活动最密切经济昆虫之一, 还是一种理想模式生物。蜂蜜是养蜂生产中一种主要蜂产品, 属于药食同源的食物, 一直被广泛食用。蜂蜜的功能研究长期以来一直是热门课题, 每年都有大量学术论文发表。本文从蜂蜜抗氧化活性成分、影响其抗氧化活性成分相关因素、蜂蜜抗氧化作用机制、蜂蜜抗衰老功能四方面研究进展进行了总结, 并提出了几点展望, 以期对广大读者有参考价值。

关键词 蜂蜜; 抗氧化活性; 抗衰老; 进展

Advances in research on the antioxidant properties and anti-aging functions of honey

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Abstract Honey bees are both the most important pollinator and one of the most economically important insects. They also serve as an ideal model organism. Honey, a major product of beekeeping, is both an edible, and medicinal, food, and is widely consumed. Research on the functional properties of honey has long been a popular topic, with numerous articles published annually. This article reviews advances in four aspects of research: the antioxidant components of honey, the factors influencing these components, the mechanisms responsible for honey's antioxidant activity, and its anti-aging functions. In addition, several prospects for future research are proposed.

Key words honey; antioxidant activity; anti-aging; advance

蜜蜂是与人类生产活动最密切的经济昆虫之一, 同时也是一种理想模式生物(陈伟轩等, 2024)。在促进农业授粉绿色发展、维持生态系统中生物多样性、满足人们对优质蜂产品需要、以及助力乡村产业振兴等领域都发挥了重要作用(曾蜜等, 2022)。蜂蜜是工蜂采集植物的花蜜或分泌物, 经过充分酿造而贮藏在巢脾内的甜物质(曾志将, 2023), 也是养蜂生产中最主要的蜂产品之一, 还是养蜂生产者主要经济来源。我国不仅是世界养蜂第一大国, 也是世界蜂蜜年产量和出口第一大国(曾蜜等, 2023)。

蜂蜜功能研究一直是一个热门领域, 作为一种成分复杂的天然液体, 蜂蜜约含有 200 种化学物质(Escuredo *et al.*, 2013), 主要由单糖、二糖、低聚糖和多糖等糖类组成, 另外还含有酶类、矿物质、维生素、氨基酸及多酚等的生物活性物质(Luca *et al.*, 2024), 具有抗氧化、抗炎、抗菌、抗肿瘤和抗病毒等特性, 其中抗氧化性是最重要的特性之一(Martinello and Mutinelli, 2021)。作为天然的膳食抗氧化剂, 蜂蜜对糖尿病(Sharma *et al.*, 2020)、胃肠道、肝肾脏、心血管疾病和退行性衰老神经系统(Fadzil *et al.*,

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2023; Akanda *et al.*, 2024) 等疾病具有治疗或辅助治疗作用。本文综述了蜂蜜抗氧化活性成分、影响蜂蜜抗氧化活性成分相关因素、蜂蜜抗氧化机制以及蜂蜜抗衰老功能。

1 蜂蜜抗氧化活性成分

蜂蜜的抗氧化活性是酚类化合物和其他成分协同作用的结果。其中酚类化合物是最主要贡献者,它是植物产生的次级代谢物,通过蜜蜂的采集行为转移到蜂蜜中。蜂蜜含有的酚类主要是黄酮类和酚酸类化合物 (Michiu *et al.*, 2022; Wilczyńska and Żak, 2024)。蜂蜜中的其他成分如矿物质、氨基酸、美拉德反应产物、抗氧化酶和维生素等也会影响其抗氧化能力 (Gül and Pehlivan, 2018)。

1.1 黄酮类化合物

蜂蜜中发现的大多数酚类都是黄酮类化合物,主要以糖苷的形式存在 (Šarić *et al.*, 2020)。其结构特征是二苯丙烷骨架 (C6-C3-C6), 具有不同程度的中央吡喃环氧化 (Wilczyńska and Żak, 2024)。蜂蜜中广泛存在的黄酮类化合物主要有槲皮素、杨梅素、山奈酚、木犀草素、柚皮素、白杨素、芹菜素和高良姜素等 (Cianciosi *et al.*, 2018)。其中槲皮素是最常见的黄酮,几乎各种类型的蜂蜜中都存在,具有强抗氧化和自由基清除活性 (Fratta Pasini and Cominacini, 2023), 可以激活核因子 E2 相关因子 2 (Nuclear factor erythroid 2-related factor 2, Nrf2) 信号通路,降低氧化应激,增强细胞线粒体活性 (Li *et al.*, 2019)。通过上调谷胱甘肽过氧化物酶 (Glutathione peroxidase, GPx) 并减少铁在胰腺细胞中的积累来抑制脂质过氧化 (Li *et al.*, 2020)。

1.2 酚酸类化合物

蜂蜜的酚酸类化合物由一个酚环和至少一种有机羧酸官能团组成,根据结构可以分为羟基苯甲酸衍生物 (C6-C1) 如没食子酸、原儿茶酸、丁香酸和肉桂酸衍生物 (C6-C3) 如对香豆酸、

阿魏酸、咖啡酸、肉桂酸和绿原酸等 (Cianciosi *et al.*, 2018; Becerril-Sánchez *et al.*, 2021)。酚酸因其含有酚类羟基而具有良好抗氧化活性 (Chen *et al.*, 2020)。咖啡酸是蜂蜜中最普遍的酚酸,在调查的 159 种单花蜜中有 118 种蜂蜜含有 (Lawag *et al.*, 2022)。咖啡酸含量与蜂蜜氧自由基吸收能力 (Oxygen radical absorbance capacity, ORAC) 呈高度正相关 (Spilioti *et al.*, 2014), 可以抑制氧化应激,降低铁过载小鼠血浆中的脂质过氧化并增加维生素 E 水平 (Olas, 2020), 也能缓解淋巴细胞中紫外线辐射诱发的 DNA 损伤 (Khan *et al.*, 2016)。

1.3 其他成分

铜 (Cu^{2+}) 和铁 (Fe^{2+}) 等过渡性离子的存在可以显著提升蜂蜜抗氧化能力,比如黄酮类化合物与铜或锌形成配合体 (如槲皮素-铜, 山奈酚-铜等), 相较单独黄酮类物质,能够发挥双重抗氧化作用 (Simunkova *et al.*, 2021; Faúndez *et al.*, 2023)。脯氨酸是蜂蜜中最主要的游离氨基酸,其含量与自由基清除活性呈正相关 (Meda *et al.*, 2005)。美拉德反应通过形成糖-肽和/或糖-氨基酸相互作用,能够显著提升抗氧化活性,且产物具有预防脂质过氧化的效果 (Chaipoot *et al.*, 2025)。此外,蜂蜜中抗氧化酶如葡萄糖氧化酶、过氧化氢酶、超氧化物歧化酶等也可以作为氧化应激的保护剂 (Mir *et al.*, 2022)。蜂蜜中的水溶性维生素浓度与 1, 1-二苯基-2-三硝基苯肼 (2, 2-diphenyl-1-picrylhydrazyl, DPPH) 值显示正相关,能够作为自由基清除剂,有效减少氧化过程 (Chua *et al.*, 2013)。维生素 C 作为酶的辅助因子,逐级提供给电子转化成半脱氢抗坏血酸和脱氢抗坏血酸,在转化过程中达到清除自由基作用 (Akbari *et al.*, 2016)。

2 影响蜂蜜抗氧化活性成分相关因素

蜂蜜抗氧化活性是衡量其营养价值和功能特性重要指标之一,受多种因素共同影响 (Halagarda *et al.*, 2020)。蜜源植物和地理环境

决定了蜂蜜抗氧化成分初始组成, 蜂蜜颜色反映了这些成分浓度特征, 后续加工和储存影响着其活性物质及抗氧化活性变化(Islam *et al.*, 2021)。

2.1 蜜源植物与地理环境

蜂蜜成分主要源自蜜源植物, 而蜜源植物的种类对蜂蜜品质和抗氧化活性具有重要影响(Yuan *et al.*, 2024)。蔷薇科、苋科、豆科和菊科植物蜂蜜抗氧化剂含量高于其他植物科植物蜂蜜(Shakoori *et al.*, 2024)。麦卢卡蜂蜜中独特的甲基乙二醛(Methylglyoxal, MGO) 来自于麦卢卡树花蜜, 具有强抗氧化活性和抗菌性, 铁离子还原/抗氧化能力(Ferric reducing antioxidant power, FRAP) 值与独特麦卢卡因子(Unique manuka factor, UMF) 值呈强正相关(沙芳芳等, 2023)。百花蜜成分具有多样性, 塞尔维亚的草地和森林这两种百花蜜总酚酸含量和抗氧化能力是金合欢、酸橙和向日葵单花蜜的 2 倍(Atanacković- Krstonošić *et al.*, 2019)。蜜源植物开花丰度也会影响蜂蜜抗氧化活性, 一年中开花稀少的季节会导致蜂蜜酚类化合物含量降低(Stanković *et al.*, 2020)。

此外, 蜂蜜抗氧化活性与蜜源植物生长环境气候、海拔和土壤特性相关。在肯尼亚降水量高、炎热潮湿和半干旱地区生产的蜂蜜总酚酸含量存在显著差异(Becerril-Sánchez *et al.*, 2021)。土壤盐度会影响植物组织多酚含量, 在采矿和冶金工业地区, 土壤的盐度增加会导致蜂蜜抗氧化能力减弱(Nicewicz *et al.*, 2021)。在云南高海拔地区蜂蜜中的槲皮素和芦丁含量高于低海拔地区(1700 m 以下), 因为高海拔地区昼夜温差可以促进植物抗氧化次级代谢物积累(Wang *et al.*, 2024)。

2.2 蜂蜜颜色

蜂蜜颜色是重要的感官特征和质量参数之一, 常用测定方法是卜方特(Pfund) 比色法和 CIE Lab 色度分析。根据国标 Pfund 比色法, 蜂蜜颜色范围被分成从水白色(<8 mm) 到深色(>114 mm) 数种(Tuberoso *et al.*, 2014)。蜂蜜颜色深度与酚类含量、抗氧化活性具有强正相

关性(Alvarez-Suarez *et al.*, 2013)。深色枣花蜜的总酚含量是洋槐蜜的 3-4 倍, 其抗氧化能力远高于浅色洋槐蜜(问亚琴等, 2024)。蜂蜜颜色越深, 其 a^* (红调) 值越高, 抗氧化能力越强(Kivima *et al.*, 2021)。此外, 颜色强度是衡量蜂蜜中色素(如类胡萝卜素、矿物质、酚类和类黄酮等) 含量高低相关指标, 与抗氧化活性也具有很强相关性(Islam *et al.*, 2012)。

2.3 蜂蜜加工与储存条件

热处理是蜂蜜加工中常用技术, 热加工温度和时间直接影响着蜂蜜抗氧化活性成分(Scepankova *et al.*, 2021)。研究表明, 浅色、酚类含量低的蜂蜜在 90 °C 条件下加热 60 min, 抗氧化活性显著提升, 相反, 深色且富含酚类化合物的蜂蜜热处理后抗氧化活性降低(Kowalski, 2013), 原因是高温加热处理会促使蜂蜜美拉德反应, 其产物如类黑精的形成, 会增加抗氧化活性(Jahan *et al.*, 2015)。对于富含维生素 C 和抗氧化酶的蜂蜜, 热加工会导致它们的损失和变性, 从而降低抗氧化活性(Bhure *et al.*, 2025)。蜂蜜加工中超声、高压、微波处理等也会影响蜂蜜中抗氧化活性成分(Subramanian *et al.*, 2007), 比如超声处理可以增加蜂蜜中抗氧化化合物含量, 因为在超声波磁场下, 蜂蜜中花粉粒会被分解, 从而释放出酚类化合物(Pereira *et al.*, 2023); 在室温下高压处理可以使蜂蜜抗氧化活性提高 30%, 且颜色保持不变(Ramly *et al.*, 2021); 蜂蜜经过微波处理 6 min 后, 总酚含量和 2, 2-联氨-二(3-乙基-苯并噻唑-6-磺酸) 二胺盐(ABTS free radical scavenging capacity, ATBS⁺) 自由基清除活性均有所下降(Kowalski, 2013)。

存储温度、时间和光照也会使蜂蜜的抗氧化活性发生波动变化。龙眼蜜在 4、25 和 35 °C 储存 3-24 个月, 结果显示: 35 °C 高温下储存, 其总酚含量增加 10 余倍, 并增强了自由基清除能力(Chou *et al.*, 2020); 土耳其松树和百里香蜂蜜在常温下储存 12 个月, 总酚含量和总抗氧化状态均随着储存时间的延长显著降低(Yiğit *et al.*, 2024)。蜜露蜂蜜储存 24 个月之后, 白杨

素、山奈酚、柚皮素及咖啡酸等酚类化合物浓度下降约 50% (Seraglio *et al.*, 2021)。相比于光照条件,在黑暗条件下储存的蜂蜜总黄酮含量下降速度更慢 (Manickavasagam *et al.*, 2024)。

3 蜂蜜抗氧化作用机制

蜂蜜可以展现多方面抗氧化作用,除了直接清除自由基、通过螯合过渡金属来中和活性氧 (Reactive oxygen species, ROS) 以发挥抗氧化作用之外 (Rice-Evans *et al.*, 1996; Morar *et al.*, 2025), 还可以增强氧化应激的防御机制 (Cianciosi *et al.*, 2018), 通过调节炎症信号、免疫细胞以及肠道菌群组成与功能来实现抗氧化效果 (Ranneh *et al.*, 2021; Cárdenas-Escudero *et al.*, 2023)。

3.1 直接机制

自由基是正常细胞代谢的产物,因其含有奇数个电子使其化学性质活跃,易与其他化合物发生反应。常见的自由基有超氧阴离子 ($O_2^{\cdot-}$)、羟基自由基 ($\cdot OH$) 等 (Phaniendra *et al.*, 2015)。当自由基产生过多,或抗氧化防御减弱导致不平衡时,会引发氧化应激,进而造成细胞损伤和疾病产生 (Battino *et al.*, 2021)。蜂蜜中酚类可以通过提供氢离子或电子直接清除自由基。麦卢卡蜂蜜中的丁香酸甲酯 (酚类) 对 $O_2^{\cdot-}$ 显示出特异性清除活性 (Inoue *et al.*, 2005); 荞麦蜂蜜的主要酚酸是对羟基苯甲酸和对香豆酸,其 $\cdot OH$ 清除活性为 23.2%-44.1%, 在体外条件下,荞麦蜂蜜对 $\cdot OH$ 介导的 DNA 损伤和 DNA 链断裂具有保护作用 (Zhou *et al.*, 2012)。蜂蜜的自由基清除活性与总酚酸含量呈相关性,枣花蜜总酚酸为 467.31 mg PCA/kg, 其 DPPH 自由基清除能力的半数抑制浓度 (Half maximal inhibitory concentration, IC₅₀) 为 46.36 mg/mL, FRAP 为 2.53 mmol Fe^{2+} /kg (张敏等, 2023)。欧洲松木蜂蜜 ORAC 为 11.60 μmol TE/g, 与其总酚酸含量相关性为 0.94 (Gorjanović *et al.*, 2013)。

蜂蜜中的酶类可以调控过氧化氢 (Hydrogen peroxide, H_2O_2) 浓度实现氧化平衡。葡萄糖氧

化酶能将蜂蜜中的葡萄糖分解成葡萄糖酸和 H_2O_2 , 低浓度的 H_2O_2 (1-10 $\mu mol/L$) 可以激活细胞抗氧化防御 (Sies, 2017)。过氧化氢酶 (Catalase, CAT) 把 H_2O_2 降解成水和氧气 ($Oxygen, O_2$), 防止 H_2O_2 产生毒性的 $\cdot OH$ (Osés *et al.*, 2024)。超氧化物歧化酶 (Superoxide dismutase, SOD) 将 $O_2^{\cdot-}$ 转化为 H_2O_2 和 O_2 , 减少自由基对细胞的损伤 (Erejuwa *et al.*, 2011)。蜂蜜中丝氨酸蛋白酶的主要产物小肽,也可以起抗氧化作用 (Alaerjani *et al.*, 2022)。

蜂蜜中的 Fe^{2+} 、 Cu^{2+} 等过渡金属离子会与 H_2O_2 发生芬顿反应,导致强氧化性的 $\cdot OH$ 产生,从而引发复杂的氧化链式反应,包括脂质、糖类、蛋白质、DNA 的过氧化等,导致细胞氧化损伤 (Timoshnikov *et al.*, 2022)。酚类化合物含有芳香环和羟基,可以与过渡金属离子在 3 个位点螯合,从源头上抑制活性氧的产生 (Faúndez *et al.*, 2023)。槲皮素可以通过其 3', 4'-邻苯二酚羟基与 Cu^{2+} 形成稳定的铜-槲皮素螯合物 (配位比 2 : 1), 阻断芬顿反应产生有害 $\cdot OH$ (Birjees Bukhari *et al.*, 2009)。槲皮素也可以与 Fe^{2+} 螯合来缓解 DNA 的损伤 (Perron and Brumaghim, 2009), 咖啡酸的羟基和羧基与 Fe^{2+} 螯合可以抑制脂质过氧化 (Kiokias and Oreopoulou, 2022)。

3.2 间接机制

3.2.1 激活内源性抗氧化系统 除了直接清除自由基之外,蜂蜜也可以通过激活细胞自身的抗氧化防御系统来抵抗氧化应激。内源性抗氧化剂包括酶促抗氧化剂,如 SOD、CAT 和 GPx 等,以及非酶促抗氧化剂谷胱甘肽 (Glutathione, GSH), 小分子如尿酸、胆红素、辅酶 Q 等 (Erejuwa *et al.*, 2012a; Aguilar *et al.*, 2016)。Nrf2/抗氧化反应元件 (Antioxidant response element, ARE) 信号通路的调控是内源性抗氧化系统的核心 (Akpoveso *et al.*, 2023)。Nrf2 是调控细胞氧化还原平衡和防止氧化损伤的关键因子,正常情况下,与 Kelch 样 ECH 相关蛋白 1 (Kelch-like ECH-associated protein 1, Keap1) 结合成复合物,稳定在细胞质内,通过泛素化降

解维持低水平表达。氧化应激会破坏 Keap1 中的半胱氨酸残基, 解除 Nrf2 泛素化降解, 使其转位到细胞核中, 与 ARE 的启动子区域结合并激活下游细胞抗氧化酶的转录 (Jung and Kwak, 2010; Pasupuleti *et al.*, 2020)。麦卢卡蜂蜜的多酚提取物可以诱导腺苷酸活化蛋白激酶 (AMP-activated protein kinase, AMPK) 磷酸化来激活 Nrf2 结合 ARE 反应, 从而增加下游 SOD 和 CAT 基因的表达, 保护人类真皮成纤维细胞免受氧化损伤 (Alvarez-Suarez *et al.*, 2016)。血红素加氧酶-1 (Heme oxygenase-1, HO-1) 通过降解血红素发挥抗氧化作用, 胡枝子蜂蜜通过上调 Nrf2/HO-1 通路并促进下游基因和抗氧化酶醌氧化还原酶 1 和 HO-1 的表达来抑制铁死亡 (氧化应激和铁代谢失调有关的细胞死亡形式) (Ren *et al.*, 2024)。蜂蜜处理也可以显著提高庆大霉素诱导的肝肾功能障碍大鼠肝脏和肾脏的 CAT、GPx、GSH 活性, 降低丙二醛 (Malondialdehyde, MDA) 水平, 缓解氧化应激和肝肾损伤 (Laaroussi *et al.*, 2021)。

3.2.2 抗炎与免疫 持续氧化应激会引起炎症反应, 蜂蜜通过抑制促炎信号通路和促炎酶活性, 调节免疫细胞功能来构建氧化防御系统 (朱文振等, 2024)。在脂多糖 (Lipopolysaccharide, LPS) 诱导的巨噬细胞炎症模型中, 用红花蜂蜜处理可以抑制核因子 κ B 抑制蛋白 α 磷酸化来抑制核因子 κ B (Nuclear factor-kappaB, NF- κ B) 信号通路激活, 并降低相关炎症因子诱导型一氧化氮合酶、白介素-1 β (Interleukin-1 β , IL-1 β)、肿瘤坏死因子- α (Tumour necrosis factor alpha, TNF- α) 和单核细胞趋化蛋白-1 表达水平, 从而保护受氧化应激细胞 (Sun *et al.*, 2020)。羊刺蜂蜜多糖提取物可以提高血清中抗炎因子白细胞介素-10 和干扰素- γ 含量, 降低 MDA 和 ROS 水平, 下调 Toll 样受体 4/丝裂原活化蛋白激酶 (Toll-like receptor 4/Mitogen-activated protein kinase, TLR4/MAPK) 信号通路来消除肝脏氧化应激并减轻炎症 (Song *et al.*, 2024)。蜂蜜中的抗氧化剂可以诱导 T 和 B 淋巴细胞的活化和增殖, 刺激吞噬作用, 触发免疫调节反应

(Al-Hatamleh *et al.*, 2020), 马来西亚蜂蜜的多酚提取物可以减少受 LPS 刺激的巨噬细胞中一氧化氮 (Nitric oxide, NO) 的产生, 抑制炎症反应, 并与蜂蜜浓度呈剂量性依赖 (Majtan, 2014)。蜂蜜可以抑制人体中性粒细胞的超氧化物产生促进慢性伤口愈合 (Leong *et al.*, 2012)。也能诱导骨髓细胞产生促炎因子, 在癌症的情况下, 通过其抗增殖和促凋亡性来减少肿瘤细胞生长, 促进抗肿瘤免疫反应 (Masad *et al.*, 2021)。

3.2.3 肠道菌群调控 蜂蜜是一种潜在的益生元, 能通过调节肠道菌群组成与功能来间接增强氧化应激防御 (Mohan *et al.*, 2017; Deledda *et al.*, 2021)。蜂蜜中的低聚糖可以有效抑制有害菌如沙门氏菌、大肠杆菌和艰难梭菌增殖, 同时促进有益菌乳酸杆菌和双歧杆菌生长 (Schell *et al.*, 2022)。山乌柏蜂蜜多酚提取物能提高酒精诱导的肝损伤小鼠拟杆菌门相对丰度, 并降低厚壁菌门相对丰度, 从而逆转酒精诱导的菌群失衡, 减轻氧化损伤 (Luo *et al.*, 2021)。溃疡性结肠炎是一种免疫疾病, 百花蜜多酚可以通过调控溃疡性结肠炎大鼠的结肠基因表达及肠道菌群组成来改善肠道炎症和抗氧化应激能力 (Zhao *et al.*, 2019)。此外, 蜂蜜能促进老年人肠道菌群中有有益菌的生长, 并增加有益菌产生的有益代谢物如短链脂肪酸和丁酸的含量来发挥其免疫活性, 促进结肠内稳定和健康 (Wu *et al.*, 2022)。蜂蜜可以通过提高紧密连接蛋白表达, 降低细胞旁通透性, 缓解肠上皮细胞损伤, 改善 LPS 诱导的肠屏障损伤 (Nathani *et al.*, 2023), 也能恢复杯状细胞数量和粘蛋白面积, 改善肠道不规则形态和抑制 MAPK 磷酸化修复肠黏膜损伤 (Cai *et al.*, 2021), 保护肠道屏障完整性, 减少氧化应激因子进入循环系统。

4 蜂蜜抗衰老功能

衰老是一个复杂过程, 会伴随着机体内各系统功能逐渐衰退, 并引发一系列与衰老相关疾病 (Booth and Brunet, 2016)。氧化应激、慢性炎症及代谢失衡等是衰老核心驱动因素 (Liguori *et al.*, 2018; Baechle *et al.*, 2023)。蜂蜜通过其

多酚类化合物、抗氧化酶类及其他活性成分发挥抗氧化性, 进而对衰老相关疾病产生积极作用 (Jubri *et al.*, 2013)。

4.1 代谢综合征改善与心血管健康

代谢综合征是机体发生碳水化合物、脂肪、蛋白质等物质代谢紊乱, 进而导致肥胖、糖尿病、血脂异常等疾病 (陈思南等, 2023)。衰老是代谢紊乱的主要危险因素, 随着年龄的增加, 血液里的炎症指数升高, 会加速代谢紊乱发病机制的发展过程 (DiLoreto and Murphy, 2015)。蜂蜜富含的酚类和糖类可以改善代谢综合征 (Gaddanakeri *et al.*, 2024)。25 名 II 型糖尿病患者以每公斤体重每天 1.0 g 的剂量口服未加工的蜂蜜 56 d 后, 患者空腹血糖显著降低 18.9% (Ramli *et al.*, 2018)。蜂蜜降血糖机制之一是蜂蜜中酚类化合物可以有效抑制 α -淀粉酶和 α -葡萄糖苷酶活性 (Sharma *et al.*, 2020)。蜂蜜可以调节胰岛素信号通路的关键部分磷脂酰肌醇-3 激酶/蛋白激酶 B 信号 (Phosphatidylinositol 3-kinase/Protein kinase B, P13k/Akt) 通路, 在高血糖条件下, 用不同浓度蜂蜜酚类提取物处理氧化应激诱导的胰腺仓鼠细胞 24 h 后, 磷酸化 Akt 表达显著增加, 并对 β 细胞功能障碍和胰岛素抵抗具有改善作用 (Safi *et al.*, 2016; Ahmed *et al.*, 2018)。血脂异常是肥胖主要并发症之一, 其特征是高密度脂蛋白胆固醇降低, 极低密度脂蛋白胆固醇、甘油三酯 (Triglycerides, TG) 和低密度脂蛋白胆固醇升高 (Hashim *et al.*, 2021)。血脂异常患者连续 30 d 服用 70 g 天然蜂蜜后, 其 TG 水平显著降低 19%, 总胆固醇显著降低 3.3% (Yaghoobi *et al.*, 2008)。蜂蜜中白杨素 (黄酮) 可以有效减轻与年龄相关的血脂异常 (Farkhondeh *et al.*, 2019), 蜂蜜抗高脂血症作用机制是通过其酚类化合物抑制羟甲基戊二酸单酰辅酶 A 还原酶 (3-hydroxy-3-methylglutaryl coenzyme A reductase, HMG-CoA) 活性 (Erejuwa *et al.*, 2018)。

心血管疾病是衰老相关慢性疾病核心表现, 与慢性炎症和氧化应激相关 (Idrus *et al.*, 2020)。大量研究表明, 蜂蜜凭借其黄酮类化合物、抗氧

化酶等活性成分, 展现出从动脉粥样硬化抑制到血压调控再到心脏保护的多维心血管保护潜力 (Nguyen *et al.*, 2019; Olas, 2020)。蜂蜜处理可以抑制凝血酶诱导的吞噬细胞的低密度脂蛋白氧化和细胞信号传导, 减轻内皮损伤, 从而缓解动脉粥样硬化斑块部位的炎症反应 (Ahmad *et al.*, 2009)。槲皮素可以通过改变肠道微生物群和减少致动脉粥样硬化脂质代谢物来抑制动脉粥样硬化病变 (Nie *et al.*, 2019)。蜂蜜可以降低自发性高血压大鼠的收缩压和 MDA 水平, 上调 Nrf2 的表达减轻大鼠肾脏对氧化损伤的易感性 (Erejuwa *et al.*, 2012b)。氧化应激会导致血管壁炎症、血管舒张剂 NO 生物利用度降低、以及血管细胞增殖增加等, 这些会导致内皮功能障碍, 从而形成高血压 (Schulz *et al.*, 2011)。蜂蜜中的芦丁可以增强内皮型一氧化氮合酶基因表达及其活性, 促进 NO 的产生, 从而调节心血管风险, 槲皮素可以增加肱动脉直径, 减少 56% 的内皮增殖, 并提高 5 倍环磷酸鸟苷水平, 从而改善内皮功能障碍 (Afroz *et al.*, 2016; Hashim *et al.*, 2021)。蜂蜜具有保护心脏的潜力, 肌钙蛋白 I (Cardiac troponin I, cTnI) 是心肌细胞损伤的高度敏感和特异性标志物, 通常不在血清中出现, 只有在心肌坏死后才会释放到血清中 (Antman *et al.*, 2000)。用土朗蜂蜜 (每千克体重 3 g/d) 预处理 45 d, 可预防异丙肾上腺素诱导的大鼠心肌梗死, 显著降低其血清中 cTnI 水平和心脏标志酶活性 (Khalil *et al.*, 2015)。蜂蜜也可以改善心率失常, 通过蜂蜜灌注缺血大鼠可以降低性心动过速、室性异位搏动以及全心室颤动的发生率 (Najafi *et al.*, 2011)。

4.2 皮肤抗衰老和慢性伤口愈合

皮肤是人体最大的器官, 其衰老过程由多种内源性和外源性因素共同决定。内源性因素包括遗传年龄和细胞代谢过程改变; 外源性因素主要受环境影响, 如紫外线辐射、氧化应激、胶原代谢失衡等 (Khalid *et al.*, 2025)。蜂蜜因其活性成分和抗氧化性有助于延缓皮肤衰老 (Barbosa and Kalaaji, 2014)。酪氨酸酶和弹性蛋白酶与皮肤老化密切相关, 葡萄牙的海桐蜂蜜对这两种酶

有显著抑制能力,抑制率分别为 9.37%和 45.88% (Santos *et al.*, 2023)。I 型胶原蛋白 (Collagen type I, Col I) 是人皮肤中最丰富的结构蛋白,含量随皮肤的衰老而逐渐减少,基质金属蛋白酶-1 (Matrix metalloproteinase-1, MMP-1) 能够降解 Col I, 在衰老过程中表达量上调 (Gelse *et al.*, 2003; Varani *et al.*, 2006), 无刺蜂蜂蜜富含酚类化合物,其抗氧化活性能抑制衰老的人皮肤成纤维细胞的 MMP-1 基因表达,并刺激 Col I 合成,从而延缓皮肤衰老 (Malik *et al.*, 2020)。此外,蜂蜜中的高良姜素可以增强去乙酰化酶 1 介导肿瘤蛋白 53 (Tumor protein 53, p53) 去乙酰化,抑制核易位,从而减轻紫外线诱导的皮肤衰老 (Wen *et al.*, 2024)。外用 15%百花蜜添加量的护手霜 4 周后,皮肤皱纹面积显著降低 21.4%,平均皱纹深度减少 11.7% (Suwiński and Nowak, 2024)。

修复已有的损伤也是抗衰老的一部分,慢性伤口 (如糖尿病足溃疡和压疮) 是皮肤修复领域的技术难题 (Falanga *et al.*, 2022)。蜂蜜凭借其抗菌、抗炎、抗氧化和促进创面再生的特性,在治疗慢性伤口愈合方面具有安全性和有效性 (Kurek-Górecka *et al.*, 2020)。伤口愈合分为炎症、增殖和重塑三个阶段,蜂蜜在每个阶段发挥着不同作用:炎症期增加过氧化物形成,促进炎症因子 TNF- α 、白细胞介素-6 (Interleukin-6, IL-6)、IL-1 β 和前列腺素 E₂ 的释放,诱导成纤维细胞合成胶原 (Oryan *et al.*, 2016);增殖期促进肉芽形成、皮化和伤口收缩;重塑期促进伤口重塑,减少疤痕形成和挛缩 (Yilmaz and Aygin, 2020)。蜂蜜基敷料能改善不同阶段的压疮。与传统敷料相比,蜂蜜治疗组愈合时间缩短 1.7 d,压疮愈合量表评分显著提高 (Lee *et al.*, 2011)。

4.3 认知功能改善与神经保护

衰老会引发认知功能的缓慢退化,尤其是学习和记忆 (Azman and Zakaria, 2019)。蜂蜜中的膳食多酚可以通过多种方式来改善学习和记忆 (Othman *et al.*, 2015)。蜂蜜能增强大脑的胆

碱能系统和血液循环,65 岁及以上的轻度认知障碍受试者每天服用一汤匙蜂蜜可以预防认知能力下降和痴呆 (Al-Himyari, 2009)。认知能力在绝经期会逐渐降低,绝经后妇女 (45-60 岁) 补充大蜜蜂蜂蜜 16 周后,语言学习能力显著提高,血浆中 GPx 和 CAT 活性增强 (Shafin *et al.*, 2014)。补充蜂蜜可以改善暴露于噪声压力的老年大鼠的长期和短期记忆,减少氧化应激并增加脑源性神经营养因子 (Brain-derived neurotrophic factor, BDNF) 浓度 (Azman *et al.*, 2016)。给 2 个月的大鼠饮食中连续补充蜂蜜一年,并评估它们的大脑状态,大鼠在衰老的各个阶段的焦虑都显著减少并改善空间记忆 (Mijanur Rahman *et al.*, 2014)。突触可塑性对大脑的信息处理至关重要,是学习和记忆过程基础,无刺蜂蜂蜜补充剂可以使瑞士白化小鼠体内参与突触可塑性功能 BDNF 和肌醇 1,4,5-三磷酸受体 1 型基因 (Inositol1,4,5-triphosphate receptor type 1, Itp1r1) 显著上调,从而改善小鼠的空间工作和参考记忆 (Martin *et al.*, 2000; Mustafa *et al.*, 2019)。

神经元结构或功能逐渐丧失最终导致神经元死亡这一过程叫神经退行性变,年龄的增长是重要引发因素。慢性神经退行性变会导致神经退行性疾病,如阿尔茨海默病、帕金森病等 (Repici *et al.*, 2007; Yankner *et al.*, 2008)。蜂蜜神经保护作用取决于其酚类含量 (Putteeraj *et al.*, 2018)。阿尔茨海默病的病理特征是细胞外 β 淀粉样蛋白 (Beta-amyloid, A β) 斑块沉积和细胞内神经原纤维缠结 (Neurofibrillary tangles, NFT) (Shaikh *et al.*, 2023)。A β 诱导的阿尔茨海默病大鼠食用无刺蜂蜂蜜 28 d 可以减少微管相关蛋白过度磷酸化和 NFT 形成,同时减少纤维状和寡聚 A β 向斑块中聚集 (Shaikh *et al.*, 2024)。蜂蜜是胆碱酯酶抑制剂的丰富来源,可以治疗阿尔茨海默病,荞麦蜜乙酰胆碱酯酶抑制率 39.51%,百花蜜丁酰胆碱酯酶抑制率 39.76% (Baranowska-Wójcik *et al.*, 2020)。帕金森病影响 2%-3%老年人口的生活,其病理特征是脑部黑质中的多巴胺神经元变性导致运动障碍 (Bassani *et al.*, 2015)。蜂蜜与左旋多巴联合使

用可以通过保护尼氏体减少多巴胺能神经元损失,来改善被诱导的白化成年小鼠帕金森病后的步态异常 (Ayobami and Arafah, 2022)。蜂蜜中鞣花酸能通过抑制单胺氧化酶活性,减少氧化损伤治疗帕金森病 (Iftikhar *et al.*, 2022)。蜂蜜也可以显著改善 LPS 诱导的神经炎症大鼠的神经功能障碍,降低 TNF- α 、IL-6,恢复海马、前额叶皮层和纹状体的神经元组织 (Adeniyi *et al.*, 2023)。

5 展望

综上所述,虽然蜂蜜抗氧化活性和抗衰老功能方面研究取得显著进展,但以下几个方面仍需深入研究:(1)由于蜂蜜品种繁多,不同蜂蜜活性成分鉴定和精准解析,以及多组分协同的分子机制需要进一步阐明;(2)通过深入研究特种蜂蜜的活性成分和抗氧化活性,来提高蜂业生产的新质生产力,给特种蜂蜜生产、加工和销售科技赋能,增加其宣传亮点和卖点,提升特种蜂蜜的品质、品牌和效益,从而实现养蜂生产者和蜂蜜消费者双赢;(3)如何科学评价服用蜂蜜剂量达到最佳抗氧化活性效果和抗衰老功能,仍需进一步研究;(4)在蜂蜜生产、加工和销售过程中,如何维持蜂蜜抗氧化活性,也值得关注;(5)建立蜂蜜抗氧化活性成分的指纹图谱库,开发功能型蜂蜜衍生品,实现个性化蜂蜜抗衰老干预。

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