植物多酚干预肥胖发生作用机制的研究进展

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摘 要: 肥胖及其并发症严重危害公众身体健康, 且肥胖发病率逐年上升, 给社会和个人带来巨大的经济和心理负担。因此, 肥胖的预防及治疗在学术界已成为亟待解决的重要问题。植物多酚是植物体内种类最多的一类次生代谢产物, 广泛存在于水果、蔬菜、中草药以及植物源食品中, 且多酚被报道具有抗氧化、抗炎、抗菌、抗病毒和抗癌等多种生物活性。此外, 大量研究表明植物多酚具有良好的预防或治疗肥胖及其并发症的效果。本文从调节机体脂质代谢, 影响脂肪细胞的增殖、分化与凋亡, 刺激机体产热、加速能量消耗, 抗炎和促进益生菌增长以调节肠道菌群等方面系统介绍了植物多酚的减肥降脂作用及其相关机制的研究进展, 以期为植物多酚的开发与推广应用提供有益参考。

关键词: 植物多酚; 肥胖; 降血脂; 肠道菌群

Research progress on the anti-obestiy effect and mechanism of plant polyphenols

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ABSTRACT: The incidence rates of obesity and its complications are increasing year by year, which seriously endangers human health and brings huge economic burden to the society and individuals. Therefore, the prevention and treatment of obesity has become one of the most important problems to be solved. As a class of natural secondary metabolites, plant polyphenols are widely presented in fruits, vegetables, herbal medicine and plant-derived foods, and possess various bioactivities, including antioxidant, anti-inflammatory, antibacterial, antiviral, anti-cancer and other biological activities. A growing number of studies have shown that plant polyphenols have positive effects in preventing or treating obesity and its complications. This paper introduced the recent research progress on the mechanism of reducing weight and lipid of plant polyphenols from the aspects of regulating lipid metabolism, influencing the proliferation, differentiation and apoptosis of adipocytes, stimulating thermogenesis, anti-inflammatory, and modulating the gut microbiota, in order to provide reference for the utilization of plant polyphenols.

KEY WORDS: plant polyphenols; obesity; hyperlipidemia-lowering; gut microbiota

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1 引 言

肥胖是指受遗传、生理、代谢、营养和社会环境等多种因素影响而发生的慢性代谢性疾病，主要表征为由于能量代谢平衡发生紊乱、能量摄入大于能量输出导致体内脂肪过多积累与分布异常[1]。世界卫生组织(World Health Organization, WHO)规定体质指数(body mass index, BMI)≥25 kg/m^2 为超重，BMI≥30 kg/m^2 为肥胖。我国规定超重为 BMI≥24 kg/m^2，BMI≥28 kg/m^2 为肥胖[2]。截至 2015 年，全世界已约有 22 亿人超重或肥胖，我国人口中成人的肥胖发生率为 11.9%，超重发生率为 30.1%。流行病学研究和临床实验表明，肥胖症易引起高血压、高血脂、胰岛素抵抗、糖尿病、冠心病、脂肪肝和心脑血管疾病等并发症[3−5]。随着肥胖及其并发症的发病率呈逐年上升趋势，肥胖的预防及治疗在学术界已成为亟待解决的主要问题之一。目前临床上主要采用药物、手术和饮食控制等方法治疗肥胖，但这些疗法大都存在降低机体免疫力、毒副作用大、易反弹等缺点，例如西布曲明和利莫那班因容易导致严重的心脏问题或精神问题已被停用[6−8]。

植物多酚又名植物单宁，是植物体内种类最多的一类次生代谢产物，其含量仅次于纤维素、半纤维素和木质素，其主要分布于植物界(主要存在于植物的根、茎、叶、花和果实中)，尤其是深色水果、蔬菜和谷物[9]。植物多酚的基本结构为苯酚，在此基础上以多羟基取代为基本特征。多酚有多种分类方法，基于多酚的化学结构，可将其分为黄酮类和非黄酮类多酚化合物。黄酮类化合物又可进一步分为黄酮类、异黄酮类、黄烷酮类、黄烷醇类、黄酮醇类和花青素，非黄酮类化合物包括酚酸、木脂素和芪类等[9]。关于多酚的具体分类及来源详见表 1。

近年来，不断有研究学者从多种植物中提取得到多酚物质，且研究发现多酚具有抗炎、抗氧化、抗衰老、降血糖、抗菌和抗病毒等药理活性[10]。大量文献表明膳食植物多酚可以从调节脂肪代谢、刺激机体产热以加速能量消耗、调节肠道微生物菌群等多个生理层面影响发挥减肥降脂作用。由于多酚具有良好的有效性和安全性，其在肥胖防治领域的研究已成为国内外的研究热点，作为天然产物的重要组成部分日益受到人们关注。因此本文就植物多酚对肥胖及并存症的防治功效及其调节机制作一综述，以期为植物多酚的活性研究和应用推广提供参考。

表 1 植物多酚的来源与分类

Table 1 Source and classification of plant polyphenols

分类	多酚	来源
黄酮类		
黄酮	白杨素、芹菜素、芦丁、木犀草素、黄芩素	木犀草、橄榄油、水果皮、红酒、开心果、柑橘、辣椒，等
黄烷酮	柚皮素、柚皮苷、黄杉素、橙皮苷、香蜂草苷、圣草酚	柑橘类水果、柚子、柠檬、橘子、杏仁，等
黄烷醇	(+)-儿茶素、(-)-表儿茶素、(+)-没食子酸、(-)-表没食子儿茶素没食子酸酯、原花青素	茶叶、红酒、巧克力、浆果、坚果，等
黄酮醇	鼠李素异鼠李素、树柳黄素、山奈酚、杨梅黄酮、槲皮素	橄榄油、浆果、柚子洋葱、葡萄酒、苹果、香料、柑橘类水果，等
异黄酮	大豆苷、染料木苷、大豆黄素	大豆、扁豆、鹰嘴豆、花生，等
花青素	矢车菊素、天竺葵素、飞燕草素、锦葵色素、芍药色素、牵牛花色素	覆盆子、草莓葡萄、葡萄酒、浆果、豆类，等
非黄酮类		
芪类	白藜芦醇、白皮杉醇	浆果、红酒、巧克力，等
木脂素	厚朴酚、连翘苷	亚麻籽、芝麻、小麦、西兰花，等
酚酸	羟基苯甲酸(鞣花酸)、羟基肉桂酸(咖啡酸)	浆果、咖啡、茶、核桃，等
姜黄素	姜黄素	香料，等

2　植物多酚干预肥胖作用及机制

2.1　调节机体脂质代谢

2.1.1　影响脂质的消化与吸收

胰脂肪酶是水解脂肪的关键酶, 而多酚是胰脂肪酶的潜在抑制剂。Rahim 等[11]研究表明植物多酚(包括黄酮类和非黄酮类)是胰脂肪酶的有效抑制剂。黑荆树多酚物质具有良好的抑制脂肪酶活性, 口服黑荆树多酚可有效抑制小鼠血清中甘油三酯浓度的升高[12]。Sugiyama 等[13]发现苹果多酚具有很强的体外抑制胰脂肪酶活性, 且在动物和人体内进行抑制脂质(甘油三酯)吸收实验的结果表明苹果多酚具有良好的抑制脂质吸收的效果。Wu 等[14]发现荔枝花多酚可以降低脂肪酶的活性, 控制大鼠体内脂肪组织的积累。Mcdougall 等[15]研究了浆果中多酚化合物的体外抑制胰脂肪酶活性并发现北极荆棘、越橘、草莓、黄莓和树莓中所提取的浆果多酚对胰脂肪酶具有很强的抑制作用。体外研究表明从茯砖茶、白茶、绿茶和乌龙茶提取物中提取的茶多酚在体外对胰脂肪酶具有不同程度的抑制作用[16−18]。动物研究实验发现绿茶多酚能显著减少雌性 Swiss 小鼠摄食量从而减轻体重[19], 发酵绿茶中所含有的儿茶素等多酚类物质能够通过抑制胰脂肪酶和增加能量消耗降低脂质含量[20]。此外, 作为茶多酚的主要组成成分, 黄烷醇(主要为儿茶素类化合物)在小肠中基本不被吸收而直接进入大肠, 在结肠与脂质及脂肪酶混合, 且能通过氢键或疏水相互作用与蛋白酶结合, 降低脂质消化率, 有效抑制脂质的吸收从而减少能量摄入[21,22]。表没食子儿茶素没食子酸酯(epigallocatechin gallate, EGCG)能够改变脂肪颗粒的理化性质, 降低胆汁酸的浓度, 抑制脂质的消化吸收, 减少脂质在肝脏中的积累[23,24]。Klaus 等[25]发现 EGCG 通过降低食物消化率减少脂肪的积聚, 且其减肥效果具有明显的剂量依赖性。

2.1.2　抑制糖类的消化吸收和转运

机体不仅能够通过对膳食脂肪的消化和吸收来增加体内脂肪含量, 还能够在肝脏和脂肪组织中将过多的葡萄糖转换成脂肪酸和甘油三酸脂, 促进脂肪合成。因此抑制 α-淀粉酶、葡萄糖苷酶和葡萄糖转运体, 减少机体对葡萄糖的吸收和转运是有效降低机体脂肪含量的有效途径[26]。张永军等[27]研究发现绿茶多酚能够显著地可逆非竞争性抑制胰 α-淀粉酶活性, 其 IC_{50} 为 0.82 mg/mL。刘杰超等[28]研究发现苹果多酚可竞争性抑制 α-淀粉酶活性, IC_{50} 为 1.48 g/L, 最大抑制率可达 88.01%。张文芹等[29]报道了苦丁茶多酚类物质对 α-淀粉酶和胰脂肪酶均具有明显的抑制作用。上述研究表明, 多酚类能够作为天然有效的 α-淀粉酶抑制剂抑制膳食淀粉的消化, 从而降低血糖水平, 减少脂质合成原料, 这在防治肥胖发生上具有重要的应用意义。

α-葡萄糖苷酶又称葡萄糖基转移酶, 在糖的催化反应中具有水解和转糖苷的双重作用。抑制 α-葡萄糖苷酶活性能够使单链淀粉水解为葡萄糖的反应减弱, 延缓葡萄糖的吸收, 能有效降低摄食后的高血糖, 阻止糖类向脂肪的转化[30]。从香蕉花、溪黄草、青钱柳和养心草中提取的多酚类物质均对 α-葡萄糖苷酶具有显著的抑制作用, 且抑制类型多是竞争性抑制[31−33]。Lordan 等[34]研究了海藻多酚对 α-葡萄糖苷酶的抑制作用, 发现墨角藻多酚对 α-葡萄糖苷酶的抑制作用效果最好, 且抑制率具有明显的剂量依赖性, 其 IC_{50} 为 0.49 μg/mL。Castroacosta 等[35]研究发现, 苹果多酚和黑加仑多酚可通过抑制肠道对葡萄糖的转运来降低高糖膳食餐后血糖。钠偶联葡萄糖转运体 1 (SGLT1)是葡萄糖的主要转运蛋白, 主要负责转运小肠肠腔及肾小管管腔的葡萄糖进入细胞, 且小肠上皮对葡萄糖的吸收功能与 SGLT1 呈正相关, SGLT1 还能够通过调节摄食影响葡萄糖的摄取, 进而影响血糖浓度[36,37]。EGCG 已被研究证明为 SGLT1 的竞争性抑制剂, 能够显著减少小肠细胞对葡萄糖的转运吸收[38]。Schulze 等[39]报道了多酚类物质能够有效降低葡萄糖摄入后的血糖峰值, 且这一作用可能是通过对肠道 SGLT1 的抑制从而抑制葡萄糖的转运来实现的。人体实验也已证明多酚类物质可以通过竞争性抑制机制来抑制 SGLT1, 减少肠道内葡萄糖的转运吸收[40]。

2.1.3　多酚调节脂质合成与分解

(1)抑制脂质合成

脂质合成酶如脂肪酸合酶(fatty acid synthase, FAS)、乙酰辅酶 A 羧化酶(acetyl coa carboxylase, ACC)、甘油-3-磷酸脱氢酶(glyceraldehyde-3-phosphate dehydrogenase, G3PDH)、苹果酸酶(malic enzyme, ME)、葡萄糖-6-磷酸脱氢酶(glucose 6-phosphate dehydrogenase, G6PDH)和硬脂酰辅酶 A 去饱和酶(stearoyl-CoAdesaturase, SCD-1)在脂肪合成过程中起到重要作用。抑制脂质合成酶的活性可以有效抑制脂质合成, 减少机体脂肪含量。细胞实验表明普洱茶多酚可以下调 PI3K/AKt 和 JNK 信号通路, 降低 FAS 的表达活性, 同时还可以通过 LKB1 途径激活 AMPK 信号通路, 抑制 FAS 的表达, 降低 ACC 的活性, 减少脂质的合成[41]; 葡萄皮多酚通过 PPAR-P 信号通路能够降低 3T3-L1 脂肪细胞中 FAS 表达, 减少脂质合成[42]; 石榴皮中的鞣花酸和安石榴苷同样报道可通过抑制 3T3-L1 脂肪细胞中 FAS 的表达, 减少脂质的合成[43]。儿茶素被报道可以抑制 FAS、ME、G6PDH 酶的活性, 从而抑制大鼠肝脏中脂肪的合成[44]。Murase 等[45]对大鼠喂饲儿茶素可显著增加中链脂酰-CoA 脱氢酶 mRNA 和肝脏脂酰-CoA 氧化酶的表达, 促进肝脏脂质代谢。Seo 等[46]报道发酵绿茶中提取的多酚类物质能够通过下调白色脂肪组织中 SREBP1c、FAS、ACC 和 SCD-1 的表达, 减少脂质的积累, 从而减轻小鼠肥胖及其并发

症。Li 等[47]在多酚干预大鼠实验中发现茯砖茶多酚能够抑制脂质生成基因甾醇调节元件结合蛋白 1c (SREBP-1c)的表达降低 FAS 的表达活性, 抑制肝脏和脂肪组织的脂肪生成, 从而减少肝脏脂质合成和积累。Perezjimenez[48]等发现白茶多酚能够通过抑制 FAS、ME 和 G6PDH 酶的活性来减少鱼类肝脏中的脂质合成。此外, 山楂多酚和石榴皮多酚被证明其可以通过下调 3-羟基 3-甲基戊二酸单酰辅酶 A(HMG-CoA)的表达减少胆固醇的合成, 从而起到降脂的作用[49,50]。

(2)促进脂质分解

脂肪代谢酶如甘油三酯脂肪酶(adipose triglyceride lipase, ATGL)、脂蛋白脂酶(lipoprteinlipase, LPL)、激素敏感性脂酶(hormone-sensitive triglyceride lipase, HSL)、脂肪酶(lipase, LPS)是脂分解酶, 肉碱酯酰转移酶(carnitine palmitoyl transterase-1, CPT-1)是脂肪酸氧化的关键限速酶, 它能够催化长链脂肪酸进入线粒体进行 β 氧化, 加速脂质的氧化分解。上调这些基因的表达, 提高脂肪代谢酶的活性能有效促进脂肪代谢, 减轻肥胖。研究表明, 白藜芦醇和柑橘类多酚可通过调节 HSL 和 CPT-1 的活性来提高脂肪酸在线粒体的 β-氧化水平, 加速脂类分解, 减少脂质积累[51]。目前茶多酚促进脂肪分解, 减轻体重的生物活性受到了较为广泛关注。刘天囡等[21]发现茯砖茶多酚能够抑制 LPS 活性, 从而减少膳食中脂肪的水解和吸收, 降低血清中脂质含量。Li 等[47]发现研究茯砖茶多酚能够上调 PPAR-α 的表达, 诱导肝脂肪酸氧化、调节脂质和脂蛋白代谢, 降低血脂水平, 减轻高脂膳食诱导的大鼠肥胖。Cao 等[52]报道了膳食普洱茶多酚能够增强白色脂肪组织中 LPL 和 HSL 的活性, 减少大鼠脂肪沉积, 降低血浆甘油三酯、总胆固醇和低密度脂蛋白水平, 减轻大鼠体重。此外, 绿茶多酚类被报道不仅可以提高前脂肪细胞 3T3-L1 内 HSL 的转录水平, 从而促进脂肪细胞内的脂质分解[53], 还能够通过显著地上调肉碱棕榈酰转移酶 a(CPT-1a)、酰基辅酶 A 氧化酶 1(Acyl-CoA Oxidase 1, ACOX1)、肝脏 PPARγ, ATGL 及 LPL 等基因, 在动物体内加速脂质分解[54]。

(3)促进脂质外排

脂质外排能够显著减少脂质在体内的累积, 预防肥胖的发生。Khateeb 等[55]发现石榴多酚如安石榴苷、没食子酸和鞣花酸可通过激活 PPAR-α 信号通路, 上调脂肪内酯酶 PON1 的表达, 增加 PON1 的合成与分泌水平, 抑制 LDL 和 HDL 的过氧化, 促进胆固醇外排, 而石榴中的鞣花酸在较低浓度(低于 5 μmol/L)时能够激活小鼠巨噬细胞中 PPAR-α 和三磷酸腺苷结合盒转运体 A1(ABCA1)信号通路, 较高浓度(5 μmol/L)可加速肝 X 受体-α(lLXRα)的表达和转录, 促进由氧化低密度脂蛋白(ox-LDL)诱导的泡沫细胞胆固醇的外排[56]。

2.2 植物多酚影响脂肪细胞的增殖、分化与凋亡

2.2.1 植物多酚抑制脂肪细胞的增殖与分化

多酚可参与细胞内多条信号通路如激活 AMPK、上调脂肪酸氧化、抑制原脂肪转录因子的表达以抑制脂肪细胞的增殖与分化。高远[57]报道从乌龙茶中提取的乌龙茶多酚、甲基化儿茶素和表没食子儿茶素没食子酸酯 3 种多酚对 3T3-L1 前脂肪细胞的增殖和分化均有明显的抑制作用。易娟等[58]发现绿茶多酚和红茶多酚可调节脂肪细胞分化相关基因, 实现脂肪细胞分化的逆转以达到减肥效果, 且绿茶多酚活性优于红茶多酚。另外, EGCG 被报道可显著降低脂肪细胞分化相关基因如 PPARγ、C/EBPα、胆固醇调节元件结合蛋白、脂肪酸结合蛋白、LPL、FAS 和 SCD-1 等的表达水平, 抑制前脂肪细胞增殖[59,60]。

2.2.2 诱导脂肪细胞的凋亡

有研究报道 EGCG 可以直接诱导成熟脂肪细胞的凋亡, 且该过程的实现可能是 EGCG 通过调节 DNA 复制和细胞有丝分裂相关酶的活性, 抑制胞周期依赖性蛋白激酶 Cdk2 的表达和诱导 DNA 片段化而实现的[61,62]。

2.3 刺激机体产热、加速能量消耗

机体能够通过提高去甲肾上腺素的水平来增强交感神经系统活性, 增加脂肪氧化和能量消耗而增强产热。去甲肾上腺素作为交感神经系统的活动的关键因子还可促进棕色脂肪的形成。因此, 交感神经系统是能量消耗和脂类分解的重要调控系统。多酚类物质的抗肥胖的功效与其能够刺激体内产热的作用密切相关[63,64]。Dulloo 等[63]报道绿茶多酚能够干预交感神经系统, 在 24 h 内提高去甲肾上腺素的分泌水平, 增加脂肪氧化和能量消耗, 延长通过交感神经激发的生热, 有效减轻体重, 减少内脏脂肪的积累。还提出绿茶多酚刺激棕色脂肪所诱导的产热主要来自儿茶素(尤其是 EGCG)与咖啡因的相互作用可以抑制儿茶酚-O-甲基转移酶(catecholamine-O-methyl transferase, COMT)的活性, 促进释放去甲肾上腺素, 增加能量释放和促进脂肪氧化, 从而达到减轻体重的效果[65]。Chen 等[66]也通过动物实验证明绿茶多酚能够上调大鼠脂肪褐色化基因的表达, 增加大鼠能量消耗, 减少脂质积累, 抵抗高能量膳食诱导的肥胖, 从而进一步从刺激棕色脂肪产热的角度阐明绿茶控制肥胖的作用。类似研究也发现普洱茶、乌龙茶、红茶等发酵茶中所提取的多酚类物质能够通过活化 AMPK, 从而上调 UCP1 和 UCP2 的表达, 促进白色脂肪组织褐化, 刺激机体产热, 增加能量消耗以减轻肥胖[67]。

2.4 抗炎作用

流行病学研究表明肥胖是一种慢性低度的炎症过程。研究已证实植物多酚具有良好的抗炎功效, 例如原花青素可以通过下调 TAK1-NF-κB 信号通路而抑制环氧合酶-2 和一氧化氮合酶表达而发挥其抗炎效果[68]; 白藜芦醇能够抑

制 TNF-α 前体信使 RNA(pre-mRNA)的剪接而发挥抗炎作用[69]。植物多酚通过抑制炎症而减轻肥胖及其并发症的作用及机制也被广泛报道。陈芝芸等[70]发现胡柚皮黄酮能激活 SIRT1 /PGC-1 信号通路, 增强非酒精性脂肪性肝炎模型小鼠的肝脏抗氧化能力, 减少脂肪酸代谢过程中活性氧的损伤, 减轻炎症, 预防 NASH 的发生。Tracey 等[71]发现茯砖茶多酚能够改善 Wistar 大鼠的小肠炎症和肠道屏障功能, 减轻胰岛素抵抗以及肝脏内质网应激反应。随后又有研究报道茯砖茶多酚能够显著减轻肝脏和脂肪组织的炎症水平, 缓解高脂饮食诱发脂肪肝症状[72]。类似研究还发现 EGCG 能降低高脂膳食诱导的肥胖小鼠中 MCP-1、HSCRP、IL-6 和 G-CSF 的表达水平, 改善小鼠肥胖症状[73]; 发酵绿茶多酚能够下调肥胖小鼠白色脂肪组织中白细胞介素 IL-1β、IL-6 和诱导型一氧化氮合酶的表达, 降低小鼠体重和脂肪含量, 同时显著改善了小鼠葡萄糖不耐受和脂肪肝等肥胖并发症[74]。

2.5　调节肠道菌群

人类肠道中栖息着超过 2000 种微生物, 其微生物种类包括细菌、真菌与病毒等, 数目约 1014 个, 几乎是人体细胞数目的 10 倍, 肠道菌群编码的基因数量是人类基因的 100 多倍, 因此也被称为“人体第二基因组”[75,76]。肠道菌群与宿主相互作用, 调控机体生理代谢, 为宿主提供其不具备的代谢酶和代谢通路[77,78]。大量文献[79-83]证明肠道菌群能够调节机体能量吸收, 调控脂肪生成, 引发慢性炎症等。植物多酚的消化与吸收及生物活性的发挥均需要肠道菌群的参与[84]。Qiao 等[85]发现白藜芦醇可以改善高脂饮食诱导的肥胖小鼠的肠道菌群失调, 并降低厚壁菌门/拟杆菌门的比例, 从而减轻小鼠体重。Anhe 等[86]报道蔓越橘多酚能显著增加阿克曼菌群数量, 减缓高脂膳食诱导的肥胖发生, 并有效缓解肥胖相关的胰岛素抵抗与肠道炎症。Chen 等[87]发现苦丁茶多酚能够减轻丹毒丝菌科菌群的丰度, 伏砖茶多酚则可降低厚壁菌门/拟杆菌门的比例, 增加双歧杆菌科菌群的丰度, 且 2 种茶多酚均显著减缓了肥胖的发生, 提高了高脂膳食小鼠的肠道菌群的多样性。绿茶多酚能够显著改变 db/db 小鼠肠道中厚壁菌门和拟杆菌门的丰度, 降低空腹血糖水平和肠系膜脂肪重量[88]。膳食原花青素能够显著增加小鼠肠道菌群的多样性, 降低毛螺菌丰度, 减小厚壁菌门/拟杆菌门的比例, 增加能量消耗, 减轻高脂饲料引起的肥胖和高血脂[89]。油菜多酚能够显著降低厚壁菌门/拟杆菌门的丰度比例, 增加乳杆菌、劳特氏菌和艾克曼菌的丰度, 改善肠道菌群失调和非酒精性脂肪肝与胰岛素抵抗[90]。

3　结论与展望

综上所述, 目前国内外学者对源于天然植物的大量多酚类化合物在预防肥胖及其并发症的研究领域中做了大量的科学研究, 并发现天然多酚能够通过调节机体脂质代谢, 影响脂肪细胞的增殖、分化与凋亡, 刺激机体产热、加速能量消耗, 抗炎和促进益生菌增长以调节肠道菌群等多种途径改善调控肥胖及其并发症。以上研究为开发天然的减肥降脂功能食品和药物提供了有益参考。但是基于目前植物多酚的研究进展, 多酚的研究与开发仍存在以下问题: (1)植物多酚的研究对象多为多种多酚类物质的混合物, 大多数研究并未获得单一多酚, 因此对于其中有效多酚成分的研究较少; (2)不同研究报道同一来源的多酚在减肥降脂方面具有多途径和多靶点, 但多数研究对特定途径和靶点缺少系统和深入的机制研究; (3)多酚的体内实验仍不够完善, 尤其是缺乏临床实验数据, 很难判断细胞与动物等基础实验与人体临床实验的实验结果是否一致, 因此真正植物多酚其应用于食品或药品的配料的研究较少。基于以上问题, 研究者应致力于对植物多酚的单一成分和相关代谢通路对多酚生物活性做深入系统的研究, 并加大人体临床实验的力度以探究多酚对人体的作用, 以期为植物多酚的产品开发和应用推广提供科学依据。

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