# 茶饮改善高脂饮食诱导的肥胖小鼠的代谢紊乱

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**【摘要】 目的** 探究6种茶饮(绿茶、青茶、红茶、白茶、黑茶及黄茶)对高脂饮食(high-fat diet,HFD)诱导的肥胖小鼠代谢紊乱的改善作用及机制。**方法** 4周龄C57BL/6J雄性小鼠,随机分成8组,每组7只。建立高脂饲料诱导的肥胖小鼠模型,对照组小鼠保持标准饮食不变。6个实验组连续5周灌胃不同茶饮,检测小鼠的体质量、肝重比、空腹血糖及血脂,评估糖脂代谢功能。检测血清炎症因子IL-6和肿瘤坏死因子-α(tumor necrosis factor-alpha,TNF-α)及氧化应激标志物丙二醛(malondialdehyde,MDA)和超氧化物歧化酶(superoxide dismutase,SOD),结合肝脏组织病理学及糖脂代谢关键基因腺苷酸激活蛋白激酶(adenosine monophosphate-activated protein kinase,AMPK)和肉碱棕榈酰转移酶1(carnitine palmitoyltransferase 1,CTP-1)等表达分析探讨机制。**结果** 青茶显著抑制体质量增长,显示出良好的体质量控制能力;白茶显著降低空腹血糖水平,并显著降低口服葡萄糖耐量试验(oral glucose tolerance test,OGTT)和胰岛素耐受试验(insulin tolerance test,ITT)的曲线下面积,提示其协同改善糖代谢与胰岛素敏感性;黄茶表现出卓越的抗炎和抗氧化能力,显著降低肝脏中IL-6和MDA水平,同时提高SOD活性;绿茶通过上调AMPK/CTP-1表达激活脂质氧化通路。6种茶饮均显著减少肝脏脂滴的蓄积。**结论** 6种茶饮均通过降低肥胖小鼠肝脏脂肪含量缓解代谢异常,不同种类的茶通过糖代谢调控、脂质氧化、抗炎抗氧化等差异化机制发挥改善代谢紊乱的作用。

**【关键词】** 茶; 高脂饮食(HFD); 肥胖; 胰岛素抵抗; 炎症反应; 氧化应激; 代谢紊乱; 小鼠

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# Ameliorative effects of tea on metabolic disorders in obesity mice induced by high-fat diet

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**【Abstract】 Objective** To investigate the ameliorative effects and mechanisms of six types of tea (green tea, cyan tea, red tea, white tea, black tea and yellow tea) on metabolic disorders in obesity mice induced by high-fat diet (HFD). **Methods** Four-week-old male C57BL/6J mice were randomly divided into 8 groups with 7 mice per group. An HFD-induced obese mouse model was established, and the mice in control group maintained on standard diet followed by intragastric administration of different teas for 5 weeks. The body weight, liver weight ratio, fasting blood glucose, and lipid profile of the mice were measured to assess glucose and lipid metabolism. Serum inflammatory factors including IL-6, tumor necrosis factor-alpha (TNF-α) and oxidative stress markers [malondialdehyde (MDA) and superoxide dismutase (SOD) were measured. Additionally, liver histopathology and the expression of key glycolipid

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metabolism-related genes, adenosine monophosphate-activated protein kinase (AMPK) and carnitine palmitoyltransferase 1 (CPT-1), were analyzed to explore underlying mechanisms. **Results** Cyan tea significantly suppressed weight gain, demonstrating superior weight control. White tea markedly reduced fasting blood glucose levels and decreased the area under the curve of oral glucose tolerance test (OGTT) and insulin tolerance test (ITT), indicating synergistic improvements in glucose metabolism and insulin sensitivity. Yellow tea exhibited exceptional anti-inflammatory and antioxidant effects, reducing hepatic IL-6 and MDA while enhancing SOD activity. Green tea activated the lipid oxidation pathway by upregulating AMPK/CPT-1 expression. All kinds of tea significantly attenuated hepatic lipid droplet accumulation. **Conclusion** All six types of tea alleviated metabolic disorders by reducing hepatic fat content in obesity mice. However, different types of tea exert their unique effects on improving metabolic disorders through differential mechanisms such as glucose metabolism regulation, lipid oxidation, and anti-inflammatory and antioxidant actions.

**【Key words】** tea; high-fat diet (HFD); obesity; insulin resistance; inflammatory reaction; oxidative stress; metabolic disorders; mouse

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日常饮食中摄入过量脂肪会导致糖脂代谢紊乱,诱发肥胖、胰岛素抵抗、糖尿病等疾病[1-2]。异位脂肪会产生和分泌多种激素和炎症因子,对动脉、肝脏等组织器官造成损伤[3]。根据生产工艺和多酚氧化程度,茶可分成6类:绿茶、白茶、黄茶、青茶(主要是乌龙茶)、红茶和黑茶。茶叶中的多酚、黄酮、多糖等活性物质可通过调节脂质吸收、促进能量代谢及改善胰岛素敏感性、调节肠道菌群等多种途径,有效改善肥胖相关代谢紊乱[4-8]。动物实验结果显示,不同类型的茶干预均可有效降低肥胖小鼠的体质量和血脂,降低空腹血糖以及血清中的胰岛素含量,缓解胰岛素抵抗[9-12]。不同茶因其特征性次级代谢产物谱(如多酚氧化程度衍生的茶黄素/茶红素比例、黄酮苷类组成及微生物发酵产物等),作用于不同的代谢通路,从而发挥差异性的代谢调控作用[13-15]。关于茶对糖脂代谢的影响已有大量研究,但综合比较6种茶的研究相对较少。本研究旨在通过构建高脂诱导的肥胖C57BL/6J小鼠模型,系统评估不同种类的茶对体成分、糖耐量及肝功能的影响,检测高脂饲料诱导的肥胖小鼠体内的炎症及氧化应激水平,并通过分析肝脏关键基因的表达情况,探索茶的潜在作用机制,以期为研究茶叶预防和控制肥胖的功效及机制提供参考。

## 材料和方法

**实验动物和茶** 4周龄C57BL/6J雄性小鼠(SPF级)购自上海斯莱克有限公司。小鼠在标准实验室条件下饲养,温度23 ℃,12 h昼夜循环,可自由获取食物和水。动物实验均经复旦大学动物伦理委员会批准(批准号:202010012)。小鼠采用标准饮食(4.5%脂肪饲料,美国Purina公司)饲养1周后随机分组(每组7只):标准饮食(normal-fat diet, NFD)组、高脂饮食(high-fat diet, HFD)组、黄茶组、红茶组、白茶组、绿茶组、黑茶组、青茶组。NFD组继续用普通饲料饲养,其他小鼠用含60%脂肪的高脂饲料(D12492,美国ResearchDiets公司)饲养12周。高脂饮食12周起,小鼠每日灌胃茶饮,剂量为0.1 mL/10 g,NFD组小鼠灌胃dd$H_2O$,剂量为0.1 mL/10 g,共5周,饲料不变。每周记录小鼠体质量,灌胃第一天和处死当天测量空腹血糖。灌胃茶饮5周后,用七氟烷麻醉小鼠,脱颈处死,采集血样,4 ℃下静置3 h,1 500×$g$离心15 min,获得血清。

茶叶产地如表1所示。黑茶、白茶和黄茶煮沸,制成浓度为0.1 g/mL的茶汤,绿茶、青茶和红茶用沸水冲泡,浓度为0.1 g/mL。

**酶联免疫吸附实验** 根据试剂盒(英国Abcam公司)说明书测量血清中的总胆固醇(total cholesterol, TC)、甘油三酯(triglyceride, TG)、高密度脂蛋白(high density lipoprotein cholesterol, HDL)、低密度脂蛋白(low density lipoprotein cholesterol, LDL)、胰岛素(insulin, INS)浓度。根据ELISA试剂盒(英国Abcam公司)说明书测量肝脏

表1　茶叶来源

Tab 1　Characteristics of tea

| Category | Name | Origin |
|---|---|---|
| Yellow tea (HY) | Golden Buns | Jiangkou, Guizhou Province |
| Black tea (HB) | Golden Fungi | Qiyang, Hunan Province |
| White tea (HW) | Old Eyebrow | Fuding, Fujian Province |
| Green tea (HG) | Green Pearls | Meitan, Guizhou Province |
| Cyan tea (HT) | Titkuanyim | Anxi, Fujian Province |
| Red tea (HR) | Yunnan Red | Yongping, Yunnan Province |

中的丙二醛(malondialdehyde，MDA)、超氧化物歧化酶(superoxide dismutase，SOD)、IL-6、肿瘤坏死因子-α(tumor necrosis factor-alpha，TNF-α)浓度。

**口服葡萄糖耐量测试和胰岛素耐受性测试**　小鼠禁食12 h后进行口服葡萄糖耐量试验(oral glucose tolerance test，OGTT)，测试前0.5 h葡萄糖灌胃(2 g/kg)，使用血糖仪(ACCU-CHEK PerformaNano，瑞士Roche公司)测量尾静脉全血血糖，测量初始(0 min)血糖和体质量，在30、60、90、120 min时测量并记录血糖。小鼠禁食4 h后进行胰岛素耐受性测试(insulin tolerance test，ITT)。胰岛素(美国Sigma公司)现用现配，溶于生理盐水配置成150 mg/mL的胰岛素溶液，储存于4 ℃冰箱。按照0.75单位/kg的剂量皮下注射胰岛素溶液，使用血糖仪在0、30、60、90 min时测量血糖。

**肝脏组织病理学观察**　肝脏浸泡在4%多聚甲醛溶液中24 h后，梯度乙醇(75%～100%)脱水，石蜡包埋，切片，厚度为4 μm，HE染色或油红O染色，光学显微镜(Eclipse CI，日本Nikon公司)下观察并拍照。

**实时定量PCR**　用液氮冷冻并研磨分离出肝脏中的总RNA。使用Nanodrop 2000(美国Thermo Scientific公司)分析RNA浓度和纯度。逆转录条件：25 ℃，5 min；42 ℃，30 min；85 ℃，5 s。引物序列参见表2，以β-肌动蛋白作为管家基因。PCR循环扩增条件：95 ℃，15 s；60 ℃，60 s；40个循环。使用公式$2^{-(\Delta\Delta Ct)}$计算目标基因的相对表达量。

表2　实验所用的引物序列

Tab 2　Nucleotide sequences of primers

| Gene | Forward sequence (5'-3') | Reverse sequence (5'-3') |
|---|---|---|
| *AMPKα* | CTCAGGAAGGCTGTATGCGG | ACGGTTGAGATACTCCGGGAT |
| *ACC* | ATGCTATTTCTTTGTTTGGTCGT | CCCAGCACTCACATAACCAAC |
| *CPT-1* | CTTCAATACTTCCCGCATCCCT | AGCAGCCTCCCGTCATGGTA |
| *GAPDH* | TCATCTCTGCCCCCTCTGCT | CGACGCCTGCTTCACCACCT |
| *G-6-Paes* | GTAGAATCCAAGCGCGAAAC | TCTGTCCCGGATCTACCTTG |

**统计学分析**　使用GraphPad Prism 10.0和SPSS 27.0软件进行数据分析和绘图。使用单因素方差分析(ANOVA)、协方差分析(ANCONA)和非参数检验Kruskal-Wallis、事后Dunnett-*t*检验和Dunn's检验进行样本之间的多重比较，*t*检验用于比较两组之间的差异，$P<0.05$为差异有统计学意义。

## 结　　果

**茶饮抑制HFD小鼠的体质量增加**　茶对小鼠体质量的影响如表3所示。与NFD小鼠相比，高脂饮食小鼠初始体质量明显增加。以初始体质量为协变量引入协方差分析，判断接受灌胃5周后各组小鼠的体质量变化。与HFD组相比，黑茶、白茶、绿茶、红茶、青茶组最终体质量显著降低。通过分析最终体质量和初始体质量差值发现，与HFD组相比，黑茶、白茶、绿茶、红茶、青茶组小鼠体质量增加受到显著抑制。

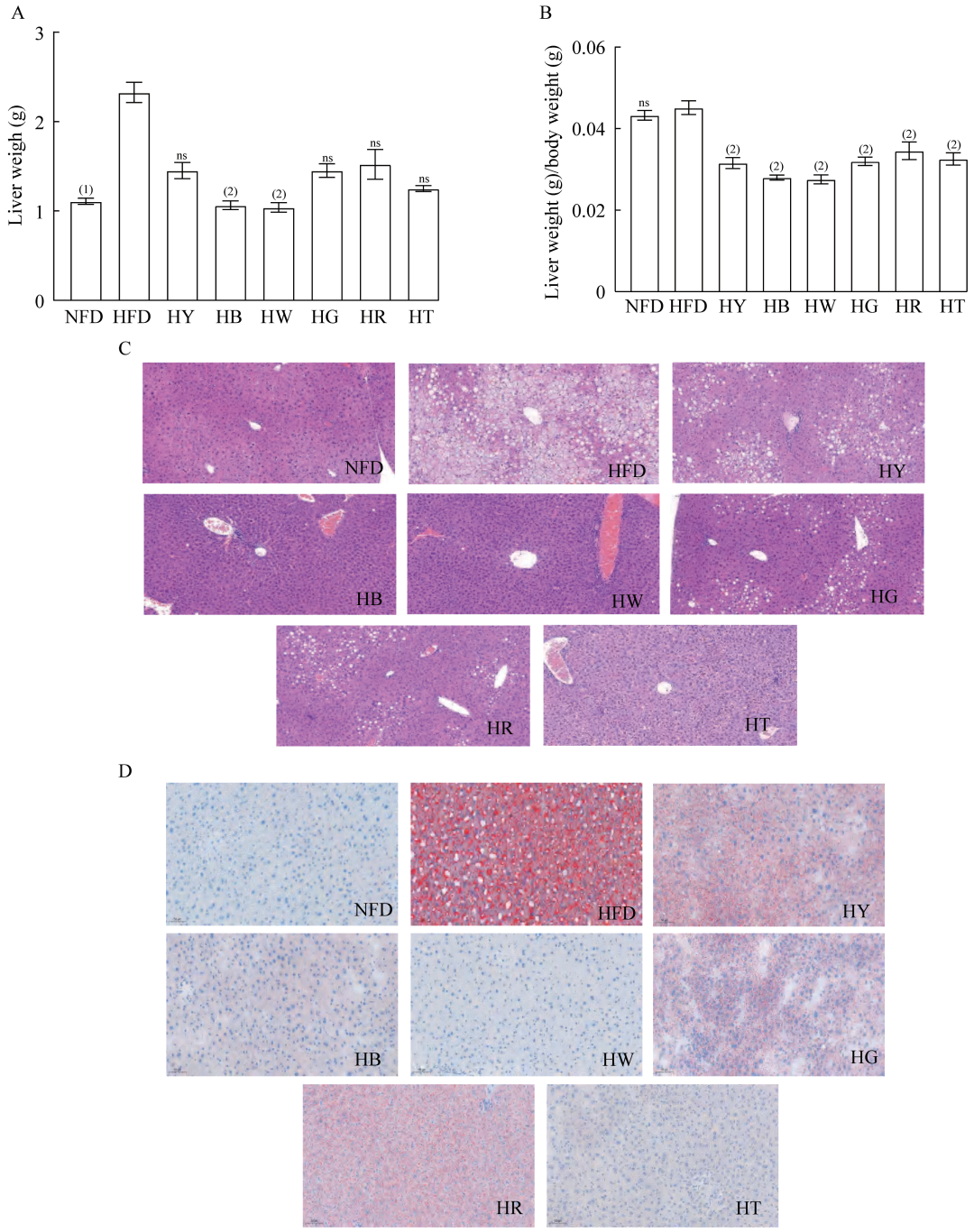
表3　6种茶对HFD小鼠体质量的影响

Tab 3　Effects of six types of tea on body weight of HFD-fed mice　($\bar{x}\pm s$)

| Group | Initial weight (g) | Final weight (g) | Weight gain (g) |
|---|---|---|---|
| NFD | $26.58\pm0.79$ | $27.01\pm0.88^{(4)}$ | $0.43\pm0.67$ |
| HFD | $47.58\pm4.33^{(1)}$ | $52.13\pm2.41^{(5)}$ | $4.56\pm2.60$ |
| HY | $47.16\pm3.48^{(1)}$ | $47.47\pm2.92$ | $0.30\pm1.74$ |
| HB | $38.82\pm2.66^{(1)}$ | $39.04\pm4.09^{(3)}$ | $0.21\pm2.43^{(3)}$ |
| HW | $45.93\pm3.43^{(1)}$ | $41.82\pm2.62^{(3)(5)}$ | $-4.11\pm3.77^{(3)}$ |
| HG | $47.36\pm3.28^{(1)}$ | $45.43\pm3.50^{(3)(6)}$ | $-1.92\pm0.63^{(3)}$ |
| HR | $44.60\pm7.77^{(1)}$ | $44.80\pm7.32^{(2)}$ | $0.20\pm1.86^{(2)}$ |
| HT | $45.22\pm4.65^{(1)}$ | $40.00\pm4.51^{(3)(5)}$ | $-5.23\pm3.72^{(3)}$ |

NFD: Normal-fat diet; HFD: High-fat diet; HY-HT: Refer to Tab 1; Initial weight: Body weight on the first day of gavage; Final weight: Body weight on the day before sacrifice. *vs.* NFD, $^{(1)}P<0.05$; *vs.* HFD, $^{(2)}P<0.05$, $^{(3)}P<0.001$, $^{(4)}P<0.000\ 1$; Initial weight *vs.* Final weight, $^{(5)}P<0.05$, $^{(6)}P<0.001$. Multiple group comparisons were performed by ANOVA and ANCOVA.

HFD引起的能量过剩所产生的脂肪主要在皮下累积,少部分会进入内脏,诱发肝脏脂肪变性。HFD组小鼠肝脏重量显著增加(图1A),其中黑茶组和白茶组的肝脏质量显著降低。所有实验组肝脏质量与体质量的比值均显著下降,但NFD组与HFD组之间差异无统计学意义(图1B)。




A: Liver weight analyzed by Kruskal-Wallis with Dunn's test; B: Liver index analyzed by ordinary one-way ANOVA with Dunnett-*t* test; C: HE staining of liver (×20); D: Oil red O staining of liver (×20). *vs.* HFD, $^{(1)}P<0.01$, $^{(2)}P<0.000\ 1$, ns: Not significant. Abbreviations refer to Tab 3.

**图1 6种茶对HFD小鼠肝脏重量以及肝脏脂肪变性的影响**

**Fig 1 Effects of six types of tea on liver weight and hepatic steatosis in HFD-fed mice**

与NFD组相比,HFD组肝脏切片表现出明显的病理变化,包括细胞排列紊乱、炎症细胞浸润和大量脂滴(图1C)。肝脏质量显著降低的黑茶组和白茶组在HE切片中同样表现良好,茶饮干预5周

后，HFD小鼠肝脏脂肪变性程度明显改善，脂滴数量减少，细胞边界清晰，细胞排列较为紧密。HE染色显示肝脏质量显著降低的黑茶组和白茶组表现良好，油红O染色显示小鼠肝脏组织的脂肪含量明显下降（图1D）。

**茶饮缓解高脂饮食引起的代谢紊乱**　高脂饮食与高血脂、高血糖相关。白茶组血浆TC、TG、HDL浓度显著降低（图2A～2D），显示出较好的降血脂作用。红茶组血浆TC和HDL浓度降低显著，而黄茶、黑茶、绿茶、青茶组仅血浆HDL浓度显著降低。各实验组血浆LDL浓度均无显著差异。

除红茶组，各实验组小鼠空腹血糖均显著降低（图2E）。高血糖常伴随高胰岛素血症，细胞对胰岛素的敏感性降低，形成胰岛素抵抗。通过测量小鼠血浆中胰岛素含量发现，除绿茶组，各实验组小鼠血浆胰岛素水平显著降低（图2F）。OGTT结果显示，HFD组AUC值显著高于NFD组，黄茶、白茶、绿茶、红茶组AUC值显著降低（图2G～2H）。ITT结果显示，HFD组与NFD组的AUC值无显著差异，但黑茶、白茶、红茶、青茶组的AUC值显著低于HFD组（图2I～2J）。这些结果表明6种茶均可改善由高脂饮食引起的代谢紊乱，其中白茶在提高胰岛素敏感性方面表现最明显。

**茶饮缓解高脂饮食引起的氧化应激和炎症反应**　高脂肪、高热量饮食已被证明会诱发全面的氧化应激和炎症反应。HFD组小鼠肝脏中IL-6和TNF-α含量相较NFD组显著增加，各类茶饮对肝脏IL-6和TNF-α蛋白表达的影响不同（图3A、3B）。黄茶可显著降低IL-6和TNF-α蛋白表达水平，具有最佳的抗炎作用；黑茶和白茶仅对TNF-α有显著影响。

检测肝脏中SOD的相对活性和脂质过氧化产物MDA的浓度来评估各实验组的氧化应激水平。结果显示，黄茶和红茶可显著降低肝脏中MDA浓度，并显著提高SOD的相对活性；黑茶、白茶、青茶仅对降低肝脏MDA浓度有显著作用（图3C、3D）。

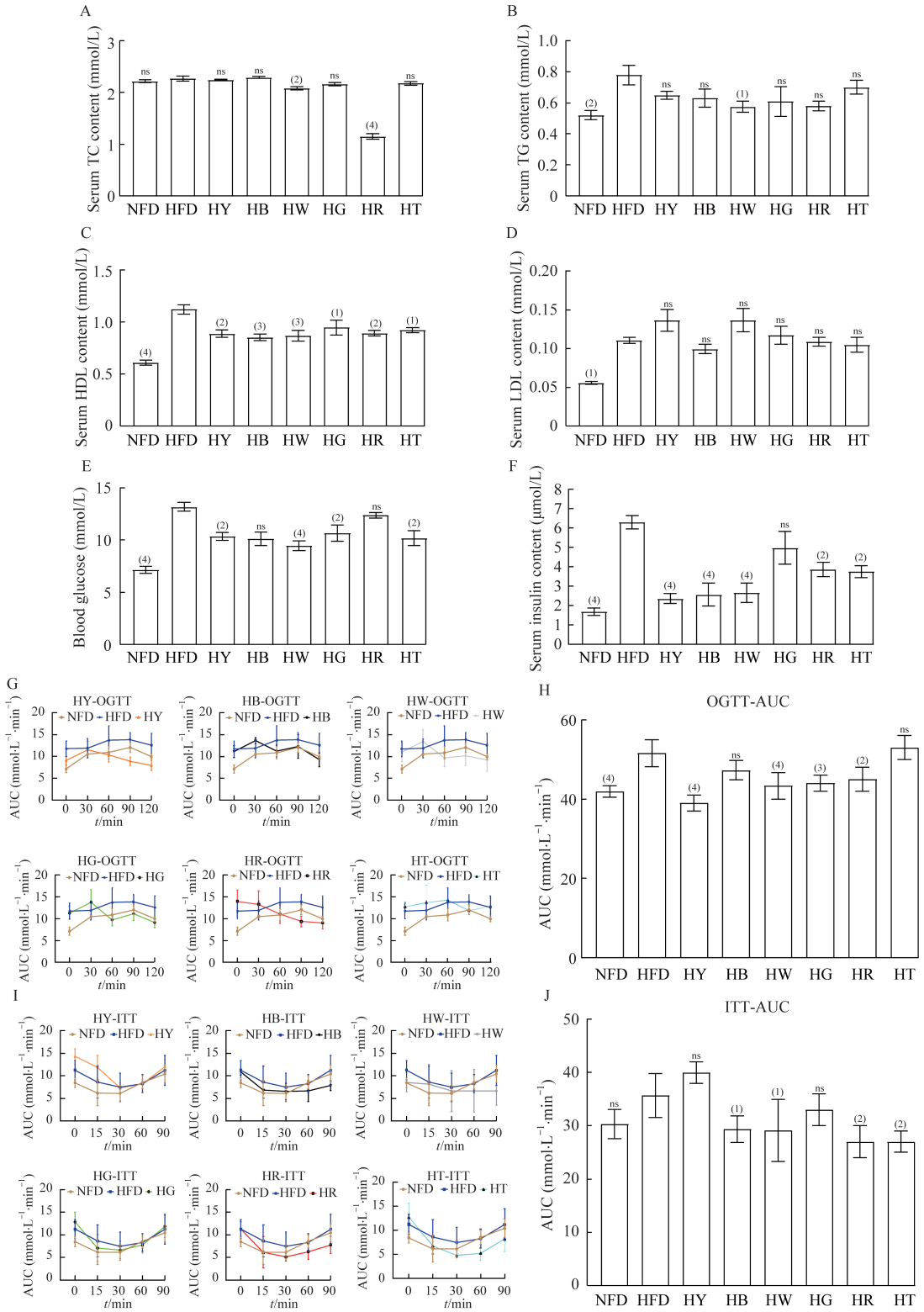
**茶饮影响糖脂代谢相关基因的表达**　AMPK是AMP依赖型蛋白激酶，激活AMPK可以刺激脂肪酸氧化，抑制脂肪酸合成及糖异生。磷酸化的AMPK可调节用于脂肪酸合成的乙酰辅酶A羧化酶（acetyl-CoA carboxylase，ACC）和用于脂酸β-氧化的肉碱棕榈酰转移酶-1（carnitine palmitoyl transferase，CPT-1）的表达或活性。绿茶能显著上调肝脏中AMPKα的mRNA表达水平（图4A）。G-6-Pase是糖异生的关键酶，可以促进肝脏中葡萄糖-6-磷酸水解成葡萄糖，从而提高血糖。黄茶、绿茶和红茶显著提高该酶的表达量（图4B）。甘油醛-3-磷酸脱氢酶（glyceraldehyde-3-phosphate dehydrogenase，GAPDH）是参与糖酵解的关键酶，红茶组GAPDH mRNA表达水平显著上升（图4C）。ACC是脂肪酸合成的限速酶，在所有实验组中ACC mRNA水平均未观察到显著变化（图4D）。CPT-1是脂肪酸氧化的限速酶，绿茶组CPT-1的表达显著升高（图4E）。

本研究比较了6种茶对高脂饮食诱导肥胖小鼠糖脂代谢的影响（图5）。所有茶饮均能改善HFD小鼠的肝脏脂肪变性和胰岛素抵抗。不同种类的茶因加工工艺和活性成分差异，通过差异化机制（如糖代谢调控、脂质氧化、抗炎抗氧化）发挥功效。青茶的减重效果最佳，白茶能显著改善胰岛素抵抗，黄茶具有最佳的抗氧化及抗炎症作用，绿茶通过调节AMPK/CTP-1信号通路来抑制肥胖。

## 讨　论

研究证明茶是一种减脂饮品[4,13,16]，可以改善非酒精性脂肪肝[17-19]。本研究中不同种类的茶均对HFD诱导的肥胖有抑制作用，茶饮降低了HFD小鼠的肝重比、血清中HDL的浓度，减少了肝脏脂滴数量和脂肪细胞的大小，缓解了肝脏脂肪变性。HDL水平与机体健康的关系比较复杂，其水平过低或过高都可能与增加的心血管风险相关[20]。本研究结果与文献报道的饮茶能够提升HDL水平不同[12,21]。这可能是由于高脂饮食导致TC和LDL水平上升，进而引起HDL水平的代偿性升高。饮茶可能帮助机体恢复正常的HDL水平，保持胆固醇代谢的平衡。

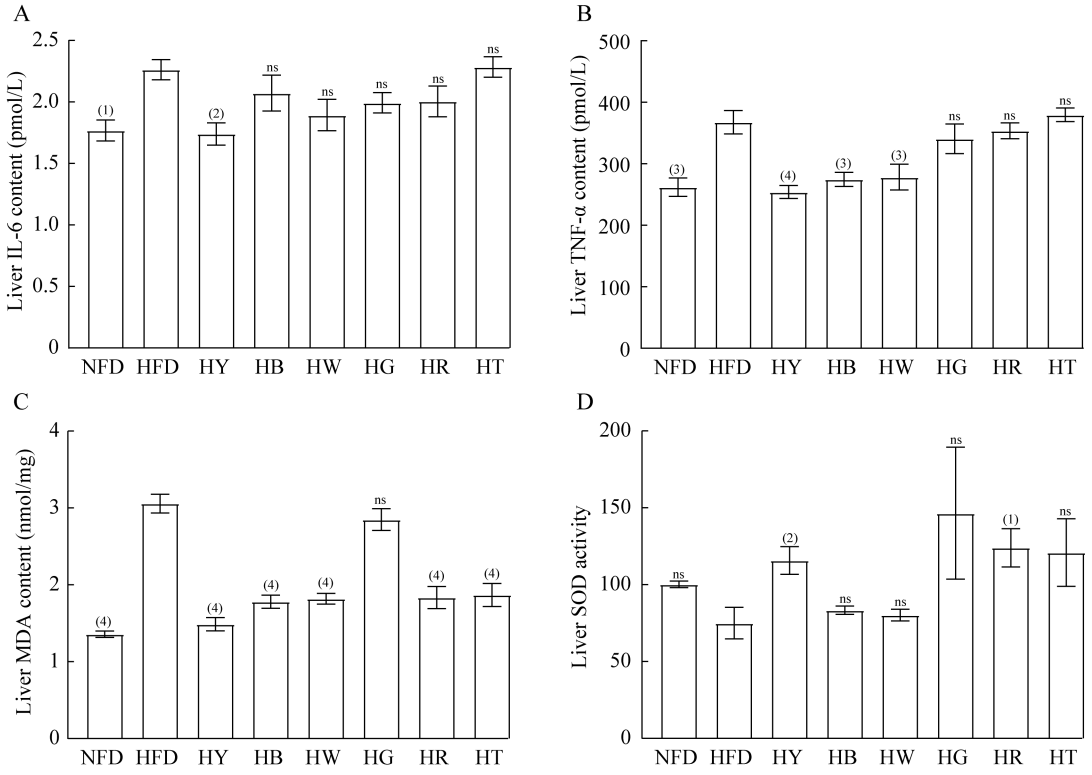
茶抑制脂肪积累的机制包括：（1）抑制食欲，减少摄食量；（2）减少肠道对脂质的吸收，从而减少热量摄入或增加粪便中的能量排泄；（3）下调肝脏、骨骼肌和脂肪组织中脂肪合成酶和相关转录因子的表达，上调脂肪氧化基因的表达水平[9,14,22-23]。研究显示，绿茶[11]、白茶[24]、青茶[25]、黄茶[14,26]均能提高AMPK的磷酸化水平，通过上调脂质氧化基因或下




A: Serum TC concentration; B: Serum TG concentration; C: Serum HDL concentration; D: Serum LDL concentration; E: Blood glucose; F: Serum insulin content; G: Changes in OGTT; H: AUC in OGTT; I: Changes in ITT; J: AUG in ITT. *vs.* HFD, $^{(1)}P<0.05$, $^{(2)}P<0.01$, $^{(3)}P<0.001$, $^{(4)}P<0.000\ 1$; ns: Not significant. A: Kruskal-Wallis with Dunn's test; B-J: Ordinary one-way ANOVA with Dunnett-*t* test. Abbreviations refer to Tab 3.

**图2 茶改善高脂饲料喂养小鼠的代谢紊乱相关指标**

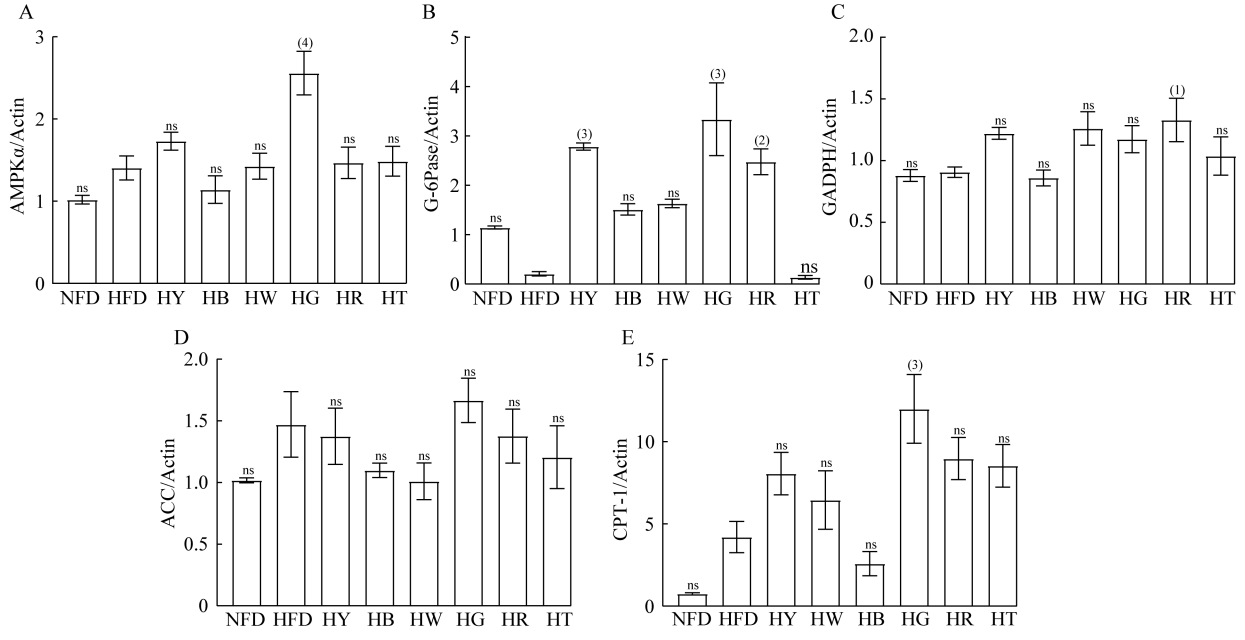
**Fig 2 Metabolic disorder-related symptoms improved by tea in HFD-fed mice**




A：IL-6；B：TNF-α；C：MDA；D：SOD. *vs.* HFD，$^{(1)}P<0.05$，$^{(2)}P<0.01$，$^{(3)}P<0.001$，$^{(4)}P<0.000\ 1$，ns：Not significant. A-C：Ordinary one-way ANOVA with Dunnett-*t* test；D：Kruskal-Wallis with Dunn's test. Abbreviations refer to Tab 3.

**图3　茶饮缓解高脂饲料喂养小鼠肝脏中的炎症及氧化应激反应**

**Fig 3　Liver injury and oxidative stress relieved by tea in HFD-fed mice**




A：AMPKα；B：G-6-Pase；C：GADPH；D：ACC；E：CPT-1. *vs.* HFD，$^{(1)}P<0.05$，$^{(2)}P<0.01$，$^{(3)}P<0.001$，$^{(4)}P<0.000\ 1$，ns：Not significant. A and C：Ordinary one-way ANOVA with Dunnett-*t* test；B，D and E：Kruskal-Wallis with Dunn's test. Abbreviations refer to Tab 3.

**图4　RT-qRCR测定茶饮对高脂饲料喂养小鼠肝脏中糖脂代谢相关基因表达的影响**

**Fig 4　Effects of tea on gene expression involved in glycolipid metabolism of liver measured by RT-qRCR in HFD-fed mice**

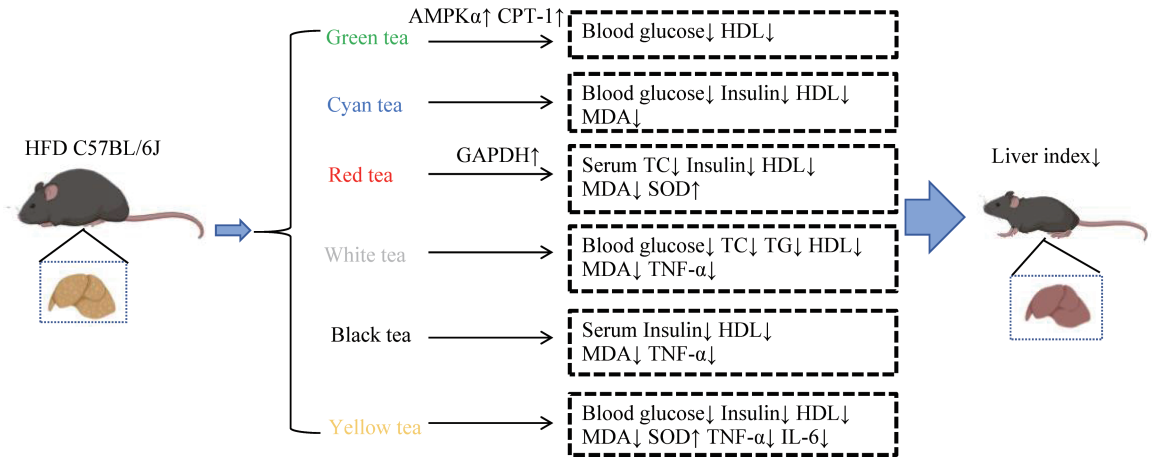



**图5 6种茶改善肥胖小鼠的代谢紊乱的作用和机制**

**Fig 5 Mechanism and effects of six types of tea in improving metabolic disorders in obesity mice**

调脂质合成基因的表达,实现降脂。活化的AMPK可以通过增强ACC的磷酸化来促进CPT-1的表达[25]。我们只在绿茶中发现AMPKα和CTP-1的mRNA表达增加,推测绿茶通过调节AMPK/CTP-1信号通路,促进脂肪酸的氧化来抑制肥胖。在其他品种的茶中并未发现与脂肪合成代谢相关基因的表达变化,这可能是由于茶的品种不同。此外,茶的剂量、提取物浓度甚至年份都可能影响研究结果。

本研究进一步证实茶是降糖饮品,可以改善高胰岛素血症,提高葡萄糖耐受量,缓解胰岛素抵抗[26-27]。空腹血糖升高主要由肝脏产生的内源性葡萄糖引起,是肝脏胰岛素抵抗的一种表现[27]。研究揭示,除红茶外,其他茶均能显著降低空腹血糖,增强肝脏胰岛素敏感性。但血糖和胰岛素水平无法全面体现肝脏的胰岛素敏感性,后续可以结合丙酮酸耐量实验(pyruvate tolerance test,PTT)、高胰岛素-正常血糖钳夹等实验进一步评估肝脏胰岛素敏感性[28]。OGTT和ITT则反映了骨骼肌的胰岛素敏感性[29-30]。实验结果表明,6种茶均能改善骨骼肌的胰岛素敏感性,但不同种类的茶在不同实验中的效果存在差异,由此提示其不同的作用机制。为了评估不同种类的茶对血糖的调控机制,我们测量了肝脏中糖异生及糖酵解关键酶的表达量。胰岛素通过上调GAPDH的表达和抑制G-6Pase的表达,有效调节血糖水平[31-32]。RT-qPCR结果显示,茶并不能降低G-6-Pase的表达量,可能是因为茶的降糖效果促进了肝脏的糖异生功能;只有红茶上调了肝脏中GAPDH的mRNA表达,提示其可能促进了糖酵解过程,但空腹血糖并未显著下降,这可能与肝脏中G-6-Pase的高度表达有关。茶降低空腹血糖的作用可能是通过其他酶或转录因子实现的,具体机制有待进一步研究。

肥胖与慢性氧化应激有关,氧化应激又会引发炎症反应[33]。通常认为绿茶由于多酚含量较高,而具有最好的抗氧化能力[34]。但本研究并未发现绿茶可以降低脂质过氧化物MDA的含量,而SOD的活性虽然较对照组升高,但差异无统计学意义,这可能与肝脏的胰岛素抵抗有关[35]。黄茶则表现出极佳的抗炎抗氧化功能,显著降低了肝脏中IL-6、TNF-α、MDA的含量,提高了SOD的活性。青茶能显著降低MDA的浓度;黑茶和白茶能显著降低肝脏TNF-α和MDA的含量;红茶能显著提高肝脏的抗氧化水平,但未见其对细胞因子含量有影响。

本研究所采用的茶饮浓度模拟了普通人群日常饮茶的习惯,并调整为小鼠可耐受的水平,因此浓度相对较低。研究聚焦于短期(5周)饮茶对HFD诱导肥胖小鼠代谢的影响,而长期饮茶的效果仍待进一步探讨。茶的活性成分如何影响代谢,以及其抗炎和抗氧化作用的机制,还需要进一步研究。

不同种类的茶成分各异,但都具有抗肥胖和抗高血糖的作用。未来将针对各类茶的显著功效进行探讨,纳入更多代谢相关基因和蛋白标记物,并结合成分分析,以期全面揭示茶对健康的益处。

**作者贡献声明** 王晨 数据收集,论文构思、撰写和修订。班翔 数据统计和分析,论文撰写和修订。刘佳星,桑思瑶,敖雪,苏明杰,胡彬蔚 研

究设计，数据收集，文献调研和整理。李辉　研究设计和指导，论文修订。

**利益冲突声明**　所有作者均声明不存在利益冲突。

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