茶褐素（Theabrownin）的功效及体内代谢途径文献梳理（增补版）

基于原 C:\Users\dm09177\Desktop\茶褐素文献（63 份 PDF）+ 新增 3 篇文献，共 66 份 PDF；去重后 62 篇文献条目并完成分类整理

核心结论速览

茶褐素（TB）是黑茶/普洱茶发酵产生的大分子多酚-多糖复合色素，分子量 0.7–100+ kDa。 现有研究证实其具有**降血糖、降血脂、抗肥胖、降尿酸/护肾、改善认知、抗炎/抗氧化、抗肿瘤、改善烧伤后急性肾损伤**等多种功效。 因茶褐素在上消化道基本不被吸收，其"体内代谢"实质是**肠道微生物介导的代谢命运**：结肠菌群将其发酵为短链脂肪酸等代谢物，并通过重塑胆汁酸肠肝循环、抑制肠道 FXR-FGF15、激活肝替代胆汁酸合成通路，最终增加粪便胆汁酸排泄，实现降胆固醇等全身效应。 涉及 FXR/TGR5、PPAR-γ/NF-κB、AMPK-PGC1α、PI3K-AKT、Nrf2/ARE 等信号通路，以及"菌-肠-肝-脑"轴。

1. 茶褐素概述：来源、形成与结构

茶褐素（theabrownin, TB）是茶叶中儿茶素类多酚经氧化、聚合、与多糖/蛋白质偶联后形成的一类水溶性褐色高分子复合物， 主要富集于黑茶（普洱茶、茯砖茶、六堡茶、藏茶、青砖茶）和红茶中（Liu et al., 2025, *Food Research International*）。

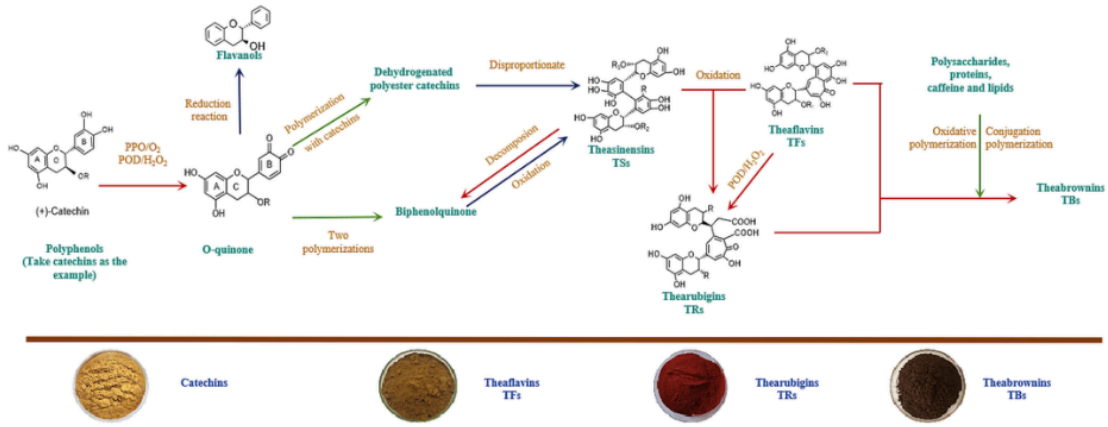


Fig. 3. The mechanism of theabrownin formation.

Table 1
TB content, production processes and flavor characteristics of five major Chinese dark teas.

Types of dark tea	TB content	Origin	Main production processes	Flavor characteristics	References
Pu-erh tea	11.00 ± 4.00	Yunnan province	Fixing → Rolling → Drying → Moistening → Piling (60–80 cm, 25–30 d) → Dying	Reddish-brown leaf, bright and deep red tea infusion, aged aroma, superior stale and mellow taste	(Gong et al., 2012)
Liubao tea	2.87 ± 0.12	Guangxi Zhuang Autonomous Region	Fixing → Rolling → Piling→ *Re*-rolling → Drying	Lustrous dark brown-black leaf with yellow spots, deep red infusion, unique betel nut aroma	(Feng et al., 2023; Shi et al., 2021)
Ya'an Tibetan tea	4.51 ± 2.62	Sichuan province	Fixing → Rolling → Drying → Piling (70 cm, 25–60 d) → Refinement → Blending → Compression	Reddish tea infusion, strong, aged and mellow taste, mild aroma	(Wang, Li, Sun, & Li, 2024; Xu et al., 2020)
Fuzhuan Brick tea	6.48 ± 0.67	Shaanxi and Hunan provinces	Crude green tea/dark tea → Steaming → Piling (200–300 cm, 10–20 h) → Steaming and Compression → 'Flowering' (~ 12 d) → Drying	Orange/orange red infusion, with golden flowers appearance, unique fungus fragrance	(Chen et al., 2022; Lv et al., 2017)
Qingzhuan Brick tea	3.05 ± 2.91	Hubei province	Crude tea → Piling (55 °C , 25–30 d) → Reprocessing → Blending → Steaming and Compression → Drying	Organe red infusion, pure and camphoraceous aroma	(Cheng et al., 2020; Xu et al., 2019)

Table 2
Pyrolysis products of TB analyzed by Pyrolysis-Gas Chromatography-Mass Spectrometry (Py-GC–MS).

Pyrolysate	TB1 (%)	TB2 (%)	TB3 (%)	Average content (%)
Phenols	24.19	23.08	29.91	25.73 ± 3.67
Alkaloid	25.23	25.67	21.91	24.27 ± 2.06
Nitrogen compounds	11.79	11.25	13.39	12.14 ± 1.11
Hydrocarbon	10.96	10.37	11.37	10.90 ± 0.50
Ketones	10.34	6.58	8.22	8.38 ± 1.89
Furan	5.40	4.06	2.33	3.93 ± 1.54
Alcohol	3.30	4.16	3.34	3.60 ± 0.49
Fatty acid	2.80	4.03	1.42	2.75 ± 1.31
Esters	2.17	0.99	2.35	1.84 ± 0.74
Amines	1.61	2.81	0.86	1.76 ± 0.98
Unidentified	2.21	7.00	4.90	4.70 ± 2.40

3.1. Systematic Analysis Extraction Method

Systematic analysis extraction method leverages the distinct solubility profiles of theaflavins, thearubigins, and TB in different organic solvents to extract and separate these compounds. Briefly, the procedure is as follows: (1) extraction and filtration: brew ground tea samples with boiling water, extract them in a boiling water bath, and filter the solution while it is still hot. (2) theaflavins and SI-type thearubigins extraction: extract the tea infusion with ethyl acetate to isolate theaflavins and some SI-type thearubigins. These compounds can then be removed from the ethyl acetate layer using a sodium bicarbonate solution. (3) SII-type thearubigins and TB separation: after the ethyl acetate extraction, the SII-type thearubigins and TB remain in the aqueous layer. Given that SII-type thearubigins are more soluble in n-butanol while TB is not, the aqueous layer is extracted with n-butanol. This process separates the SII-type thearubigins into the n-butanol organic phase, leaving the TB in the water layer (Zhang, 2021). (4) absorbance measurement: dilute each fraction to a specific concentration and measure the absorbance at 380 nm. The purity is then calculated using the extraction colorimetric method established by Roberts and Huang (Huang, 1997).

This method is primarily suited for the total quantification of theaflavins, thearubigins, and TB. While the extraction process is straightforward, it is most effective for samples with low thearubigin and TB content. However, when applied to dark tea, especially instant dark tea, this method can produce inaccuracies, occasionally leading to measured content values exceeding 100 % (Wang et al., 2017). To address these limitations, Yuan et al. (2022) refined the approach by employing weak alkali oxidation of tea polyphenols, establishing 270 nm as the characteristic UV absorption wavelength for TB, which, when combined with the use of TB reference materials, significantly enhanced the method's accuracy and applicability (Yuan et al., 2022). Building on this foundational work, Zou et al. (2016) optimized the extraction process for TB from Sichuan dark tea by conducting a quadratic regression orthogonal rotation combination design experiment, determining optimal conditions at a water temperature of 65.69–77.88 °C, an organic solvent

图 1：茶褐素形成化学路径（儿茶素→邻醌→茶黄素/茶红素→茶褐素），以及五种主要黑茶中茶褐素含量与加工工艺。 来源：Liu C 等，*Food Research International*, 2025，第 4 页（Figure 3 & Table 1）。

1.1 形成过程

- 儿茶素（catechins）在多酚氧化酶作用下氧化生成邻醌（o-quinone），再经缩合生成茶黄素（theaflavins, TFs）和茶红素（thearubigins, TRs），进一步与多糖、蛋白质、脂类等生物大分子氧化偶联形成茶褐素（Liu et al., 2025, p.4）。
- 微生物发酵（*Aspergillus*、*Eurotium cristatum*、乳酸菌等）产生的漆酶、过氧化物酶、多酚氧化酶可显著促进茶褐素积累。普洱茶渥堆过程中茶褐素可从发酵初期的约 9.29 mg/g 升至 13.24 mg/g（红曲霉混合发酵）或更高（Chen et al., 2025, *Foods* 14:1894）。

1.2 结构特征

- 茶褐素为无定形、非晶态聚合物，UV 特征吸收约 204–280 nm，FT-IR 显示富含 –OH、–COOH、C–O–C 糖苷键（Liu H et al., 2026, *J Food Sci* 91:e71314；Zhang 凤, 2024）。
- 分子量分布宽：不同制备工艺可得 Mw 773 Da（碱氧化）至 >100 kDa（大孔树脂+超滤分级）；>300 kDa 组分可占 36.83%（杨柳等，2025，*食品科学*）。
- 巯基降解研究揭示其骨架仍保留黄酮醇、表儿茶素没食子酸酯等儿茶素单元（Gong P et al., 2022, *Food Sci Hum Wellness*）。

2. 茶褐素的主要功效

2.1 调节糖脂代谢：降血糖、降血脂、抗肥胖

（1）降血糖与胰岛素敏感性

- **Li Y 等，2026，*Advanced Science*（Sex-Specific Regulation…）**：以纯化普洱茶茶褐素为对象，在女性正常及糖耐量受损（IGT）人群中，4 周干预显著降低餐后血糖；机制为茶褐素在小肠抑制 α-葡萄糖苷酶，且雌激素通过上调 MUC2 黏蛋白增强茶褐素对酶的抑制作用（非竞争性可逆抑制，荧光淬灭证实非共价结合）。

  > "Pu-erh tea or TB intervention effectively lowered postprandial blood glucose levels in females… TB's inhibition of α-glucosidase in vivo was significantly enhanced by mucin 2 (MUC2)."（摘要，p.1）

  关键图见 图 11。

- **侯艳，2023，云南农业大学博士论文**：db/db 代谢综合征小鼠灌胃茶褐素 1.215 g/kg·bw 12 周，改善胰岛素与血糖异常，调节肝脏昼夜节律基因（Clock/Npas2/Cry/Per）与 SCFA 转运受体（Slc16a/Olfr78）。

> "普洱茶和茶褐素可通过'菌-肠-肝-脑'轴（肠道菌群-粪便短链脂肪酸代谢-血清和脑组织脂质代谢-肝脏炎症和衰老因子及昼夜节律基因）改善代谢综合征。"（摘要，p.9）

- **张凤，2024，云南农业大学硕士论文**： 三种茶褐素（酶氧化、碱氧化、普洱熟茶来源）560/1120 mg/kg 灌胃高脂 C57BL/6J 小鼠 12 周，均显著抑制 TG、TC、LDL-C 升高，并改善 OGTT/ITT； 其中 TBs-f（560 mg/kg）和 TBs-a（1120 mg/kg）降脂最显著。

> "三种茶褐素干预后，小鼠血清 TG 水平均低于 HFD 组（P<0.0001）……TBs-f（560 mg/kg bw）和 TBs-a（1120 mg/kg bw）可以显著抑制 TC 水平的升高。"（3.4.2.3，p.61–62）

（2）降脂与抗肥胖

- **Huang F 等，2019，*Nature Communications* 10:4971（贾伟团队）**： 高脂饮食 C57BL/6J 小鼠灌胃**纯化茶褐素 225 mg/kg/天（纯度>95%）×8 周**，血清 TC、TG 及肝脏 TC、TG 显著降低， 且降胆固醇效果**强于等量普洱茶**；13 名健康男性饮用即溶普洱茶（茶褐素含量约 50%）50 mg/kg/天 ×4 周后血清 TC、TG 亦显著下降。 该研究被引 670+ 次，是茶褐素降胆固醇的奠基性工作，完整机制链条见 3.4 节。

> "Theabrownin increases the levels of ileal conjugated bile acids (BAs) which, in turn, inhibit the intestinal FXR-FGF15 signaling pathway, resulting in increased hepatic production and fecal excretion of BAs, reduced hepatic cholesterol, and decreased lipogenesis."（摘要，p.1）

- **Jensen GS 等，2016，*Clinical Interventions in Aging* 11:367**： 北美非亚裔高胆固醇血症受试者（n=59）随机双盲试验，每日服用**普洱茶提取物 3 g**，干预 20 周。 PTE 组平均减重约 1 kg（P<0.01），DEXA 显示总脂肪及 gynoid（臀/腹）区脂肪减少；TC 自第 4 周起显著下降，TG 约 −20% 并在第 8 周回到正常范围（<150 mg/dL）。 这是首个报道非亚裔人群普洱茶提取物减脂+血脂改善的 RCT。

> "Consumption of PTE was associated with statistically significant weight loss when compared to placebo (P<0.05). Fat loss was seen for arms, legs, and the gynoid region (hip/belly), as well as for total fat mass."（摘要，p.367）

> "The average reduction in body weight in the PTE group was small (1 kg), but highly significant (P<0.01)."（Results，p.370）

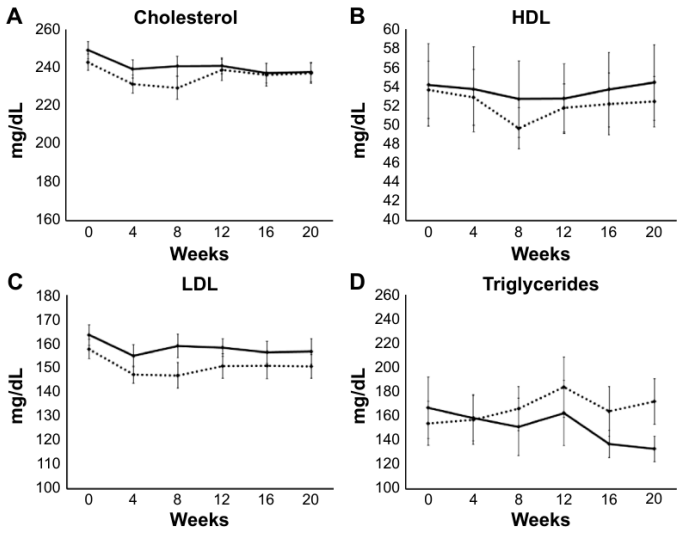


Figure 4 Lipid data are shown as group averages ± standard error of mean for the Puer tea extract (PTE) group (solid lines) and the placebo group (dashed lines).
Notes: There were no significant differences between the PTE and placebo results for cholesterol (**A**), HDL (**B**), or LDL (**C**). Within the PTE group, a significant reduction in cholesterol levels was seen already at week 4, and remained statistically significantly lower than baseline throughout the study. Triglyceride levels showed a reduction in the PTE group that was not statistically significant, but was clinically important (**D**): the group average in the PTE group went down into the normal range (below 150 mg/dL) after 8 weeks, and remained low throughout the remainder of the study. After 20 weeks, there was a 20% reduction in the average triglyceride levels within the PTE group ($P<0.08$).
Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein.

the group averages for each time point were similar for both product and placebo across the 20-week study.

Fasting blood glucose

Subjects were examined for fasting blood glucose levels at each visit (Figure 5). There was no significant change in fasting glucose over the course of the 20-week study. However, from week 8 and onward, the fasting blood glucose levels for the PTE group remained below the levels seen in the placebo group. This was seen for both sexes.

Discussion

Preserving good metabolic and cardiovascular health is important, and is related to many dietary and lifestyle factors,

Table 2 Systolic and diastolic blood pressure during the study

	Placebo		Puer tea extract		*P*-value	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Females						
Baseline	129±4.7	82±2.3	120±2.8	75±1.9	0.110	0.018
4 weeks	130±5.2	80±2.0	119±2.8	75±1.9	0.080	0.055
8 weeks	123±3.8	80±1.6	118±3.0	76±1.8	0.291	0.086
12 weeks	128±3.3	82±2.3	123±3.2	76±1.9	0.245	0.027
16 weeks	129±5.0	82±2.7	122±5.1	74±2.2	0.330	0.028
20 weeks	125±4.2	81±2.1	121±3.7	76±1.9	0.404	0.072
Males						
Baseline	134±4.9	84±2.0	128±4.2	82±1.5	0.334	0.308
4 weeks	137±6.0	85±2.1	131±6.9	81±2.7	0.472	0.304
8 weeks	143±8.1	83±3.9	135±7.9	84±3.0	0.506	0.842
12 weeks	138±9.0	86±2.8	132±5.9	82±2.0	0.598	0.280
16 weeks	132±7.2	85±2.3	134±8.8	83±2.1	0.880	0.544
20 weeks	134±6.2	87±3.2	125±3.3	81±1.2	0.212	0.121

Notes: Data presented as mean ± standard deviation unless otherwise stated.

图 12：普洱茶提取物（PTE）组与安慰剂组在 20 周干预期间的胆固醇、HDL、LDL、甘油三酯变化。 PTE 组 TC 自第 4 周起显著低于基线，TG 在第 8 周降至正常范围（<150 mg/dL）并维持。 来源：Jensen GS 等，*Clinical Interventions in Aging*, 2016，第 7 页（Fig. 4）。

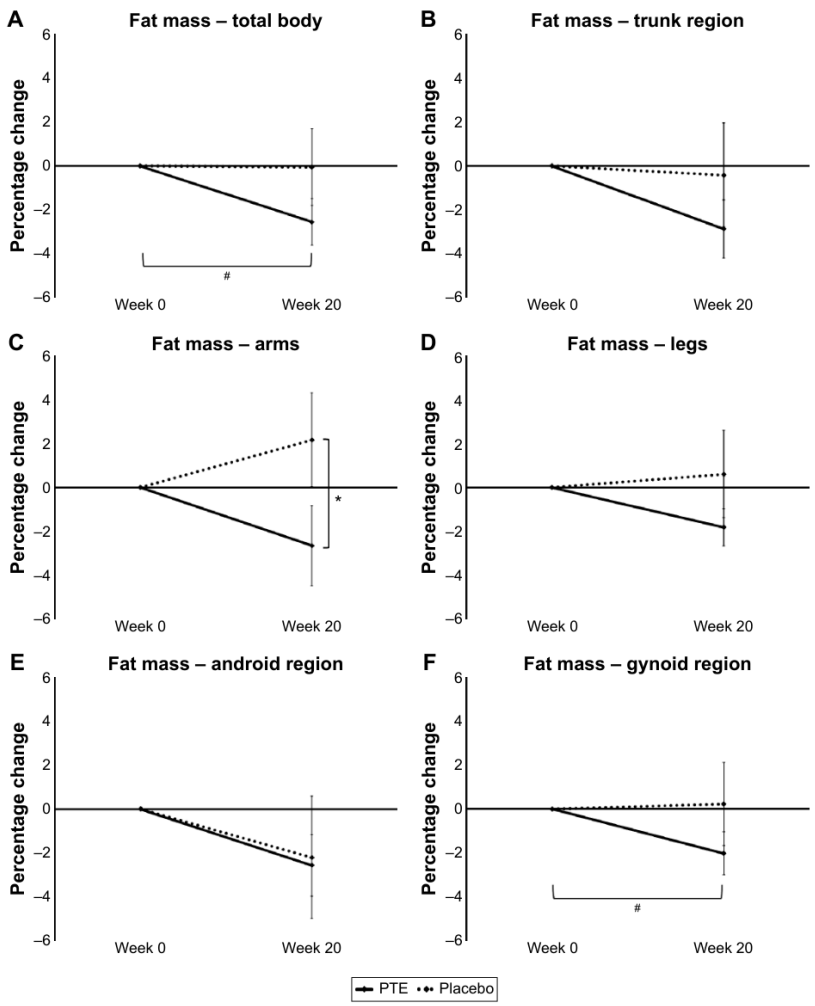


Figure 3 Changes in total body fat and regional fat composition.
Notes: Shown as the group averages of the individual percentage changes ± standard error of mean for the Puer tea extract (PTE) group (solid lines) and the placebo group (dashed lines). There were no significant differences between the two groups at week 20; however, the between-group difference in fat loss in the arms showed a statistical trend. Within the group consuming PTE, a significant loss of total body fat was seen. The total fat loss was associated with regional fat loss in the trunk, arms, thighs, and gynoid (belly/hip) regions. The fat loss in the gynoid region within the PTE group was statistically significant. *$P<0.1$, #$P<0.05$.

Blood pressure

There was no change in blood pressure in either group, despite the content of stimulants in PTE. There were no statistically significant differences between average systolic or diastolic blood pressure at baseline or during the study in either the female or male subpopulation (Table 2).

C-reactive protein

Blood samples were tested for CRP levels at each visit. At baseline, the average CRP level in the PTE group was 0.25±0.04 mg/dL, and in the placebo group was 0.22±0.04 mg/dL. Over the course of the 20-week study, average high-sensitivity CRP levels remained well within the normal range (below 3.0 mg/dL), and

图 13：PTE 组总脂肪及躯干、手臂、大腿、gynoid 区脂肪的百分比变化；gynoid 区减脂组内显著。 来源：Jensen GS 等，*Clinical Interventions in Aging*, 2016，第 6 页（Fig. 3）。

- **Yang TY 等，2014，*Phytotherapy Research* 28:1096**： 70 例代谢综合征患者随机双盲平行组 RCT，普洱茶提取物 1.5 g/天，干预 3 个月。 全人群体重 −1.3 kg 仅呈趋势（p=0.077）；**男性亚组**（n=40）体重与 BMI 显著下降（p=0.004，体重 96.15→94.10 kg）。 血脂、血糖、HbA1c 未见显著改善；HDL 反而显著下降。

> "Intake of Puerh tea for 3 months was associated with a slight reduction in body weight and BMI, especially in male patients, but did not improve lipid and glucose homeostasis in the patients with metabolic syndrome."（结论，p.1101）

- **Zhou F 等，2025，*Food Research International***： 高脂饮食肥胖 SD 大鼠中，黑茶（茶褐素 10.97%）较白茶（3.78%）更显著降低 LPS、提升肠屏障蛋白 Claudin-1/Occludin 和抗氧化酶 SOD/CAT； Pearson 相关显示茶褐素含量与 LPS、IL-10、SOD、CAT、Claudin-1 等呈正相关。

> "TB content was positively correlated with LPS, IL-10, SOD, CAT, PAS, Claudin-1, Occludin..."（结果-相关分析）

- **Feng Zhang 等，2025，*Food Chemistry* 463:141317**： 三种工艺茶褐素结构表征+体内双重证据：560 mg/kg 茶褐素调节血糖血脂，1120 mg/kg 增加降脂相关肠道菌群。 纠正：该文第一作者为 **Feng Zhang**（Ya Wang 为第二作者），此前旧笔记误记为 Wang Y，引用时应以 PDF 首页为准。

人体证据分层说明：Huang 2019（纯化茶褐素，>95%）、Ding 2025（高尿酸，TB 胶囊）、Li 2026（血糖，纯化 TB）为茶褐素直接证据； Yang 2014 与 Jensen 2016 的干预物为普洱茶提取物且未注明茶褐素含量，应归为“普洱茶整体”人体 RCT 证据链，综述中需区分表述。

3.4.3.5 HFD 诱导的肥胖小鼠肠道微生物与肥胖相关参数的相关性分析

为了进一步揭示肠道微生物与高脂饮食诱导的肥胖相关参数之间的相关性，本研究采用 spearman 相关性分析，建立了关键菌门、菌属与肥胖相关参数的相关性热图（图 3-46）。变形菌门（*Proteobacteria*）、放线菌门（*Actinobacteria*）、阿克曼氏菌属（*Akkermansia*）均与体重增长呈正相关，控制这几种菌的增加，可能有助于控制体重。F/B 与体重、TG、TC、LDL-C、OGTT-AUC、ITT-AUC 呈正相关，与 HDL-C 呈负相关，进一步证明了用高 F/B 值作为判定肥胖的标准之一是有说服力的。拟杆菌门（*Bacteroidetes*）、颤螺菌属（*Oscillospira*）、狄氏副拟杆菌属（*Parabacteroides*）和拟杆菌属（*Bacteroides*）与体重呈负相关，其中，颤螺菌属（*Oscillospira*）与肥胖参数表现出良好的相关性，它与体重、TG、TC、LDL-C、OGTT-AUC、ITT-AUC 均呈负相关，进一步印证了颤螺菌属在抑制体重增长方面发挥了重要作用。

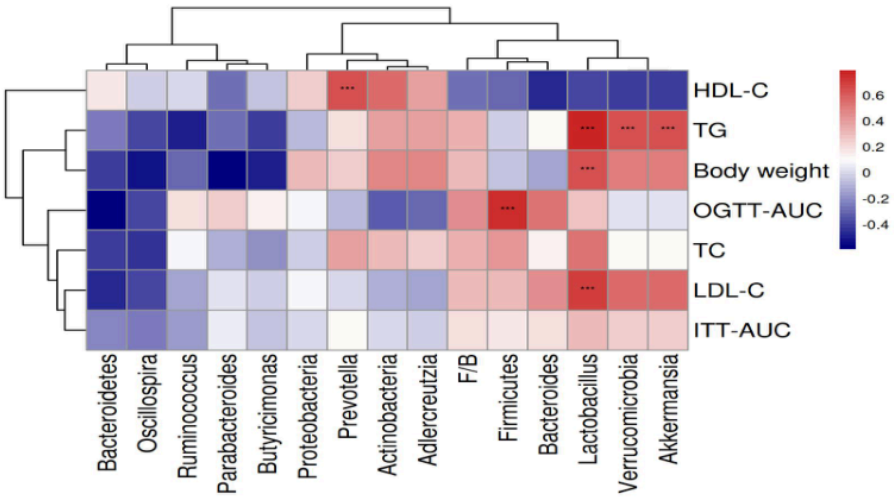


图 3-46 肠道微生物与肥胖相关参数的相关性分析

Fig. 3-46 Correlation analysis of gut microbiota with obesity-related parameters

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图 2：茶褐素干预后高脂小鼠肠道菌群（Bacteroidetes、Oscillospira、Parabacteroides 等）与肥胖指标的相关性热图。 来源：张凤，*茶褐素的制备、结构表征及其对高脂小鼠糖脂代谢的调节作用*，2024，第 73 页（图 3-46）。

2.2 降尿酸与肾脏保护

- Ding X 等，2025，*International Journal of Biological Macromolecules* 293:139373：在氧嗪酸钾+果糖诱导的高尿酸血症（HUA）小鼠中，TB（280 mg/kg，28 天）降低血清尿酸、肌酐和 BUN，改善肾小管扩张与间质损伤； 临床自对照试验（34 名男性 HUA 受试者，12 周）显示血尿酸较基线下降约 7%（p=0.033）。

  > "TB could reduce serum uric acid and improve renal pathological damage in HUA mice by inhibiting xanthine oxidase (XOD) activity in the liver and increasing the expression of uric acid transporter proteins in the kidney."（摘要，p.1）

  机制：抑制肝脏 XOD → 减少尿酸合成；上调肾脏 OAT1/OAT3（Slc22a6/8）→ 促进尿酸排泄；并通过 PI3K-AKT 抑制 NLRP3 炎症小体、NF-κB 及肾纤维化（Fig. 5，p.8）。

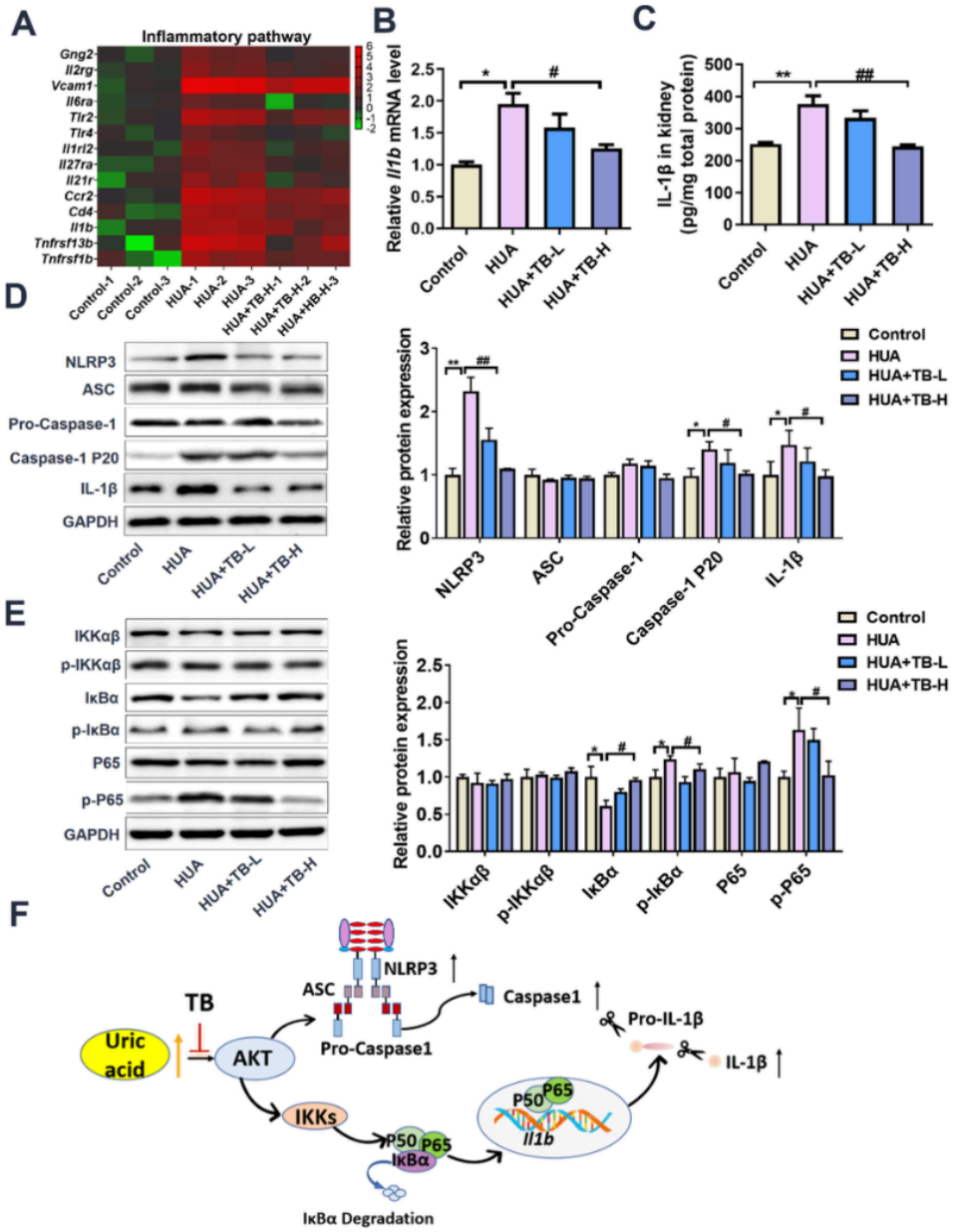


Fig. 5. TB ameliorated renal inflammation in HUA mice. (A) RNA-sequence analysis of inflammatory factors downstream of PI3K-AKT signaling pathway. (B) The relative mRNA expression of *Il1b*. (C) Expression levels of IL-1β determined by ELISA. (D) The protein expression of the NLRP3 signaling pathway was analyzed using western blotting. (E) The protein expression of the NF-κB signaling pathway was assayed using western blotting. (F) Schematic diagram of TB ameliorated renal inflammation. Statistical significance was analyzed by using the student's *t*-test. All data are presented as the mean ± *SEM* (n = 3). Compared with control, $^{*}p < 0.05$, $^{**}p < 0.05$. Compared with HUA, $^{\#}p < 0.05$, $^{\#\#}p < 0.05$.

significantly increased in the HUA and AP treatment compared with the control. Therefore, while AP effectively reduces the level of serum uric acid in humans, its potential risks to overall health should not be overlooked [13].

A previous study has demonstrated that tea extracts and bioactive compounds, such as tea polyphenols, play a beneficial role in the management of HUA [41]. Nevertheless, the bioavailability of tea polyphenols is limited. For example, the gastric bioavailability of green tea

图 3：红茶茶褐素通过 PI3K-AKT/NF-κB/NLRP3 通路改善高尿酸血症小鼠肾炎症的机制示意图。 来源：Ding X 等，*Int J Biol Macromol*, 2025，第 8 页（Fig. 5）。

2.3 神经保护与认知改善（肠-脑轴）

- **Lei M 等，2026，*Foods* 15:1587**： 自然衰老 C57BL/6J 小鼠灌胃速溶黑茶（茶褐素为关键活性成分）后，Y-maze 自发交替率从 51.91% 升至 71.59%，水迷宫逃避潜伏期从 44.84 s 缩短至 26.59 s； 抗生素清除菌群可消除认知获益，粪菌移植可部分恢复。

  > "TB treatment was associated with gut microbiota remodeling and increased serum acetate and 3-hydroxybutyrate; both metabolites partially recapitulated these benefits."（摘要，p.1）

  提示机制：TB→菌群→乙酸/3-羟基丁酸→海马功能改善。

- **Peng L 等，2026，*npj Science of Food***： D-半乳糖衰老小鼠中，熟普洱茶（茶褐素 1035.49 mg/g）与原料普洱茶（191.60 mg/g）神经保护效果相当， 提示熟普洱的神经保护高度依赖发酵衍生的茶褐素、茶黄素、茶红素等。

time ($p < 0.05$; Figure 4H,I). Collectively, these findings suggest that the gut microbiota contributes to TB-associated improvements in spatial learning and memory, and that the presence of TB, together with a reconstituted gut microbiota, is associated with greater behavioral improvement in aged mice.

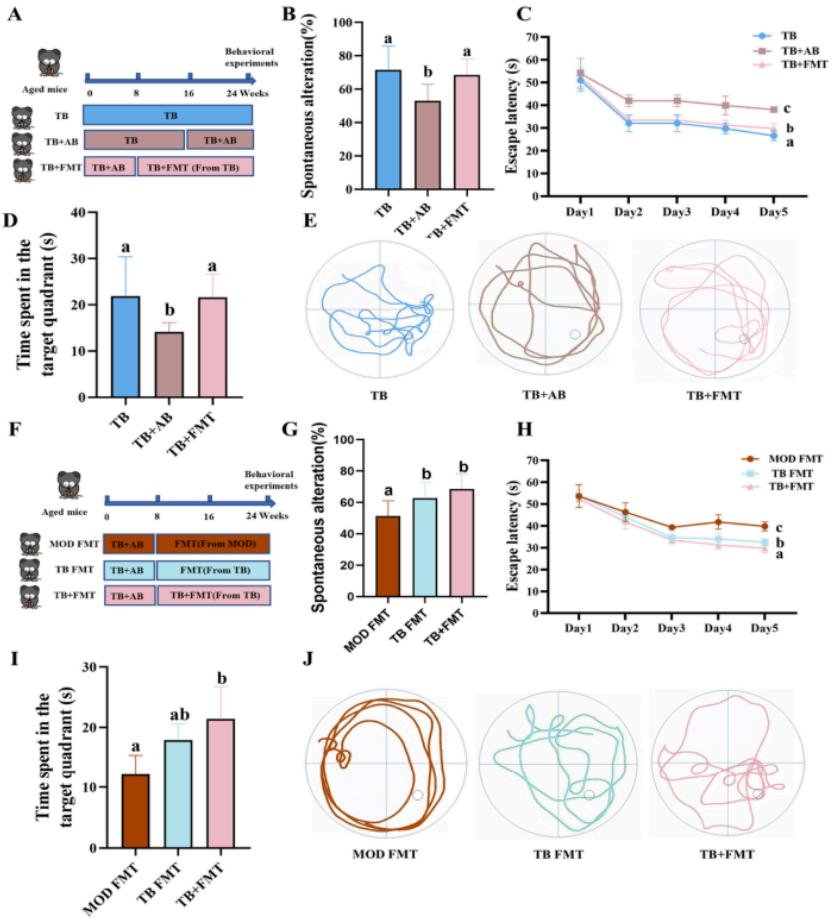


Figure 4. The cognitive-improving effects of theabrownin depend on the gut microbiota. (**A–E**) Antibiotic-induced microbiota depletion experiment: (**A**) experimental design; (**B**) Y-maze spontaneous alternation rate; (**C**) escape latency in the MWM test; (**D**) time spent in the target quadrant; (**E**) representative swimming trajectories. (**F–J**) Fecal microbiota transplantation experiment: (**F**) experimental design; (**G**) Y-maze spontaneous alternation rate; (**H**) escape latency in the MWM test; (**I**) time spent in the target quadrant; (**J**) representative swimming trajectories. Data are presented as mean ± SEM ($n = 8$ per group). Different letters indicate significant differences ($p < 0.05$).

3.5. Theabrownin Remodels Gut Microbiota Composition

16S rDNA sequencing analysis showed that TB intervention was associated with higher gut microbiota diversity indices. Specifically, the TB group had higher Chao1, ACE, and Shannon indices and a lower Simpson index than the MOD group ($p < 0.05$; Figure 5A–D). PCoA revealed distinct clustering of the TB and MOD groups (Figure 5E). Taxonomic profiling further indicated that TB treatment induced marked compositional shifts at both the phylum and genus levels. At the phylum level, the relative abundances of

https://doi.org/10.3390/foods15091587

图 4：茶褐素对自然衰老小鼠的认知改善依赖肠道菌群（抗生素清除与粪菌移植验证）。 来源：Lei M 等，*Foods*, 2026，第 10 页（Fig. 4）。

2.4 改善烧伤诱导的急性肾损伤

- **Gao Y 等，2025，*Phytomedicine* 140:156609**： 烧伤小鼠口服 TB 40/400 mg/kg 24 h，显著降低 60 h 死亡率、改善肾损伤；代谢组学发现 TB 升高肾内胍基乙酸（GAA）和富马酸（FA）， 抑制肾小管上皮细胞凋亡与铁死亡（恢复 GPX4）。

  > "TBs alleviate burn-induced renal apoptosis and ferroptosis by increasing the levels of GAA and FA in the kidneys."（摘要，p.1）

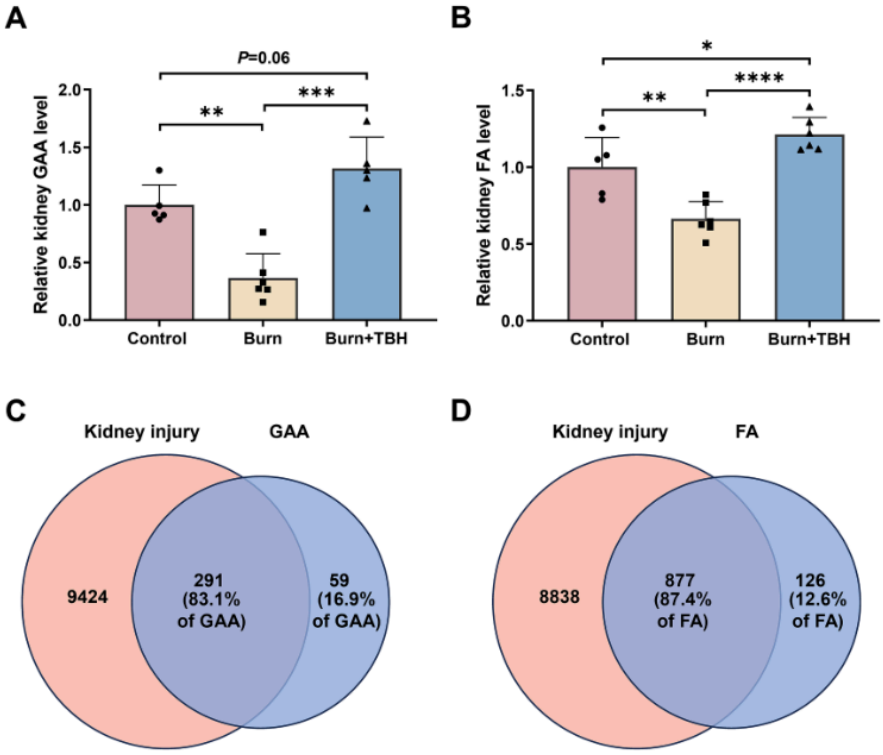


Fig. 9. TB treatment promoted the accumulation of GAA and FA in the kidneys. (A-B) The levels of the small-molecule metabolites GAA (A) and FA (B) in kidney samples of mice from the Control, Burn, and Burn+TBH groups. **(C-D)** Correlation analysis between the expression of genes regulated by GAA (C) and FA (D) and kidney injury using the GeneCards database. The data were presented as the mean ± SEM (n = 5–6). Statistical significance was indicated as $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$, and $^{****}p < 0.0001$.

FA resulted in a marked increase in GPX4 expression, indicating that both compounds may inhibit ferroptosis in renal tubular epithelial cells (Fig. 12A-B, E-F and I-J). Furthermore, intracellular GSH levels were noticeably reduced in response to H_2O_2, CDDP and erastin, whereas GAA and FA treatments significantly restored GSH levels (Fig. 12C, G and K). Conversely, MDA levels were significantly increased in HK2 cells exposed to H_2O_2, CDDP and erastin, but the GAA and FA treatments significantly reduced MDA accumulation in these cells (Fig. 12D, H and L). Taken together, these results indicated that GAA and FA could inhibit ferroptosis in renal tubular epithelial cells and alleviate cell damage.

Discussion

AKI is a common clinical syndrome that frequently occurs as a complication of severe burns, trauma, infections, postoperative stress, and nephrotoxic drug use. It is an important contributor to the high mortality rates observed in burn patients (Chen et al., 2023; Enescu et al., 2022; Lavrentieva et al., 2022; Li et al., 2023c). The pathophysiological mechanisms by which burns lead to AKI are still not fully understood. However, accumulating evidence underscores the crucial involvement of inflammation, oxidative stress, immune dysfunction, and apoptosis in AKI development (Lau et al., 2022; Li et al., 2023a; Lin et al., 2021; Liu et al., 2019; Thomas et al., 2022; Zhang et al., 2023; Zhao et al., 2021). Exploring more effective and safer therapeutic approaches is crucial for the prevention and treatment of burn-induced kidney injury and provides valuable insights into kidney damage caused by other etiologies.

Through an analysis of population databases, we found that individuals who drink tea exhibit significantly less kidney injury than other individuals. TBs are important products generated during the fermentation of tea leaves. Our previous studies revealed that TBs effectively alleviate the gut injury and hyperuricemia caused by pollutant exposure (Liu et al., 2024). Our research revealed that dorsal burns in mice result in severe renal injury, whereas TB treatment can improve burn-induced renal damage in mice by increasing the levels of GAA and FA in renal tubular epithelial cells (Fig. 13).

The development and progression of AKI are linked to pathways involving oxidative stress, inflammation, apoptosis, and ferroptosis (Chen et al., 2021). Our multiomics analysis revealed that TB treatment alleviated burn-induced kidney injury by modulating the signaling pathways related to inflammation, apoptosis, and ferroptosis.

Burn patients commonly experience early fluid loss, resulting in a decreased blood volume, increased release of inflammatory mediators, and protein denaturation (Khair et al., 2023; Zhao et al., 2023). Severe burn injuries often lead to significant disturbances in metabolism, inflammation, and coagulation (El-Kased et al., 2017; Zhao et al., 2022). Burn-induced liver injury is characterized by acute inflammation, including increased infiltration of inflammatory cells and elevated levels of proinflammatory factors in the liver tissue (Kulp et al., 2012; Wu et al., 2020). Elevated levels of circulating inflammatory cytokines, such as IL-6 and TNF-α, are associated with an increased risk of mortality in AKI patients (Ambruso et al., 2021; Yoo et al., 2020). Our study revealed that in the kidneys of burn-injured mice, the expression of the inflammatory cytokines IL-1β, IL-6, and TNF-α and macrophage infiltration

图 5：茶褐素促进烧伤小鼠肾脏中胍基乙酸（GAA）和富马酸（FA）积累，减轻肾损伤。 来源：Gao Y 等，*Phytomedicine*, 2025，第 12 页（Fig. 9）。

2.5 肠道健康、抗炎与抗氧化

- **Wei K 等，2026，*Food Research International* 229:118473**： 供体小鼠经茶褐素（300 mg/kg，30 天）调制菌群后，粪菌移植给 DSS 结肠炎小鼠，显著降低疾病活动指数（DAI）、恢复结肠长度、改善病理评分， 并恢复肠屏障蛋白 occludin/Muc2；机制涉及菌群→SCFAs/次级胆汁酸→Nrf2/ARE 与 TLR4/MyD88/NF-κB p65 通路。

  > "Fecal microbiota modulated by TPP, TPS, or TB from donors into colitis mice… significant reductions in DAI were observed, alongside attenuated intestinal histopathological damage."（摘要，p.1）

- **Fan X 等，2025，*Food Chemistry* 496:146847**： 氧化鱼油诱导肠道氧化应激大鼠模型中，热处理使茶多酚含量降低 10.3%、ROS 阳性细胞增加 4.6 倍，但茶褐素经热处理后仍保持抗氧化活性， BCL2/BAX 比值增加 25.6%，并可产生 Teadenol A 等低分子量活性产物。

- **Lin Y 等，2025，*Food Research International* 213:116555**： 金花白茶（JWT）提取物显著缓解 DSS 诱导的 IBD，作者将功效归因于发酵产生的儿茶素氧化产物如茶褐素、黄酮、没食子酸等，涉及 NF-κB 与 MAPK 通路。

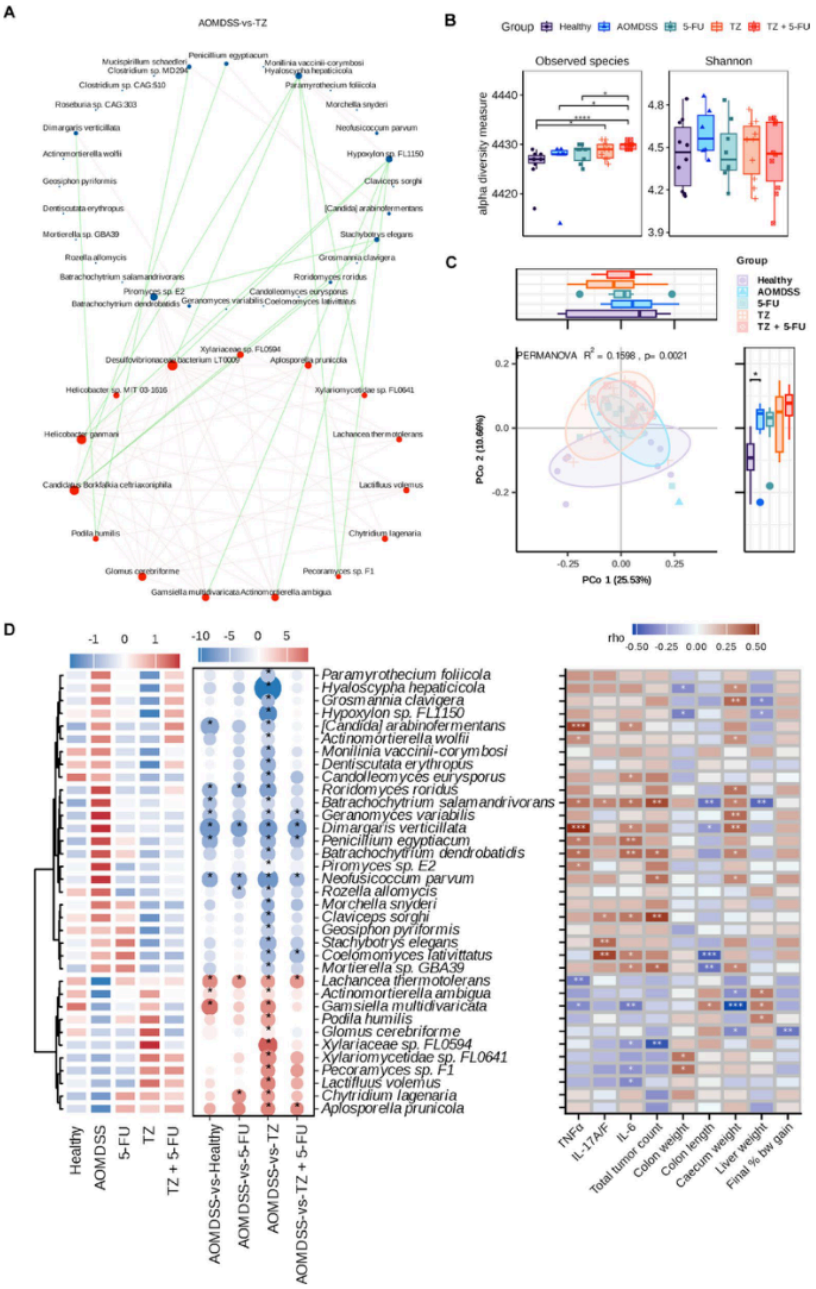
2.6 抗肿瘤

- **Jin W 等，2018，*Journal of Cellular and Molecular Medicine***： 茶褐素浓度/时间依赖抑制人骨肉瘤 U2OS 细胞，24 h IC_{50} = 51.98 μg/mL；斑马鱼异种移植模型中抑瘤率 24.6%–27.3%。 机制：诱导 DNA 双链断裂（p-H2AX）→ 激活 p53 信号通路 → 凋亡；p53 敲低后作用消失。

  > "TB-triggered DNA damage and induced apoptosis of U2OS cells by regulation of Mki67, PARP, caspase 3 and H2AX…activation of p53 signalling pathway."（摘要，p.1）

- **Leung HKM 等，2025，*Scientific Reports* 15:26386**： 茶褐素+玉米赤霉烯酮（TZ）在 AOM/DSS 结肠炎相关结直肠癌（CAC）小鼠中降低肿瘤数、抑制 PI3K/AKT/cyclin D1， 并显著升高粪便乙酸和丙酸；乙酸与肿瘤计数负相关（r=−0.63）。

  > "TZ suppressed the phosphorylation of PI3K and Akt, as well as their downstream transcription factor cyclin D1… Acetate and propionate levels were negatively correlated with tumor count."（结果，p.5）



Conclusion

The study demonstrated that the combination of theabrownin and zearalenone (TZ) significantly attenuated AOM/DSS-induced colorectal tumorigenesis. TZ demonstrated greater efficacy in alleviating CRC tumorigenesis than 5-FU, while the combination of TZ and 5-Fu did not yield additional or synergic benefits. TZ treatment improved survival rate, tumor count, colon length, colonic crypt depth, collagen fibrosis and colonic inflammation in AOM/DSS mice through the suppression of proliferative PI3K/AKT pathways and lowering pro-inflammatory cytokines. Moreover, TZ improved the gut microbiota and mycobiota, enhanced anti-CRC metabolic profile and increased fecal acetate and propionate concentration. The enrichment of the bacterial

图 6：TZ（茶褐素+玉米赤霉烯酮）改善 AOM/DSS 小鼠结直肠癌的肠道菌群、真菌群与代谢物关联图。 来源：Leung HKM 等，*Sci Rep*, 2025，第 10 页（Fig. 5）。

2.7 血管舒张

- **纪旭等，中国营养学会第十四届全国营养科学大会摘要集，p.320**： 普洱茶（0.1–10 mg/mL）浓度依赖性舒张 KCl 预收缩的离体大鼠胸主动脉，为内皮非依赖性；氯仿沉淀物（茶色素为主）是主要活性成分。

> "茶色素可能是其舒张血管的主要成分……为茶褐素在血管营养学中的应用提供科学的理论依据。"（结论，p.1）

2.8 胚胎/发育保护

- **Zhang C 等，2025，*Food Research International* 202:115555**： 藏茶茶褐素 100 μg/mL 显著改善乙醇诱导的斑马鱼胚胎畸形，降低 ROS、提升 SOD/CAT、降低 MDA，调节磷脂与支链氨基酸代谢。

2.9 安全性

- **龚加顺等，2007，*茶叶科学* 27(3):201-210**： 普洱茶特征成分提取物（含茶褐素 58.3%）经口 LD_{50} > 10 g/kg，属"实际无毒级"；Ames、微核、染色体畸变试验均未见致突变。

> "普洱茶特征成分提取物经口 LD50>10g/kg，属实际无毒级物质……未见有致突变作用。"（摘要，p.1）

- **Kim S 等，2022，*Toxics* 10(6):286**： 黑茶提取物（含茶褐素近 80%）急性毒性 LD_{50} = 21.68 g/kg；28 天亚急性试验中 40、400 mg/kg/day 无异常，4000 mg/kg/day 出现轻度肾毒性（涉及 NF-κB 通路）。
- **Wu SC 等，2011，*J Ethnopharmacol***： 普洱茶提取物 LD_{50} > 10,000 mg/kg，91 天亚慢性 NOAEL = 5000 mg/kg/day；高剂量下 TG/TC 下降、肾周脂肪减少。

3. 茶褐素的体内代谢途径

茶褐素因分子量大、亲水性强，上消化道吸收极为有限，其"体内代谢"更准确地说是"肠道微生物介导的代谢命运"及其对宿主代谢通路的系统性影响。

3.1 上消化道：基本不吸收、以大分子形式到达结肠

- **Liu H 等，2026，*J Food Sci* 91:e71314**： 体外模拟口腔/胃/小肠消化后，HPGPC 保留时间仅轻微偏移；小肠期末多酚残留率 TBSP5 60.6%、TBSE 50.2%，多糖残留率 69.9%/60.4%。

> "Undergoing only limited hydrolysis before reaching the colon…suggesting that TBs could reach the colon in macromolecular form."（3.4 节，pp.8–9）

部分成分下降可能与茶褐素结合胆汁酸形成沉淀有关。

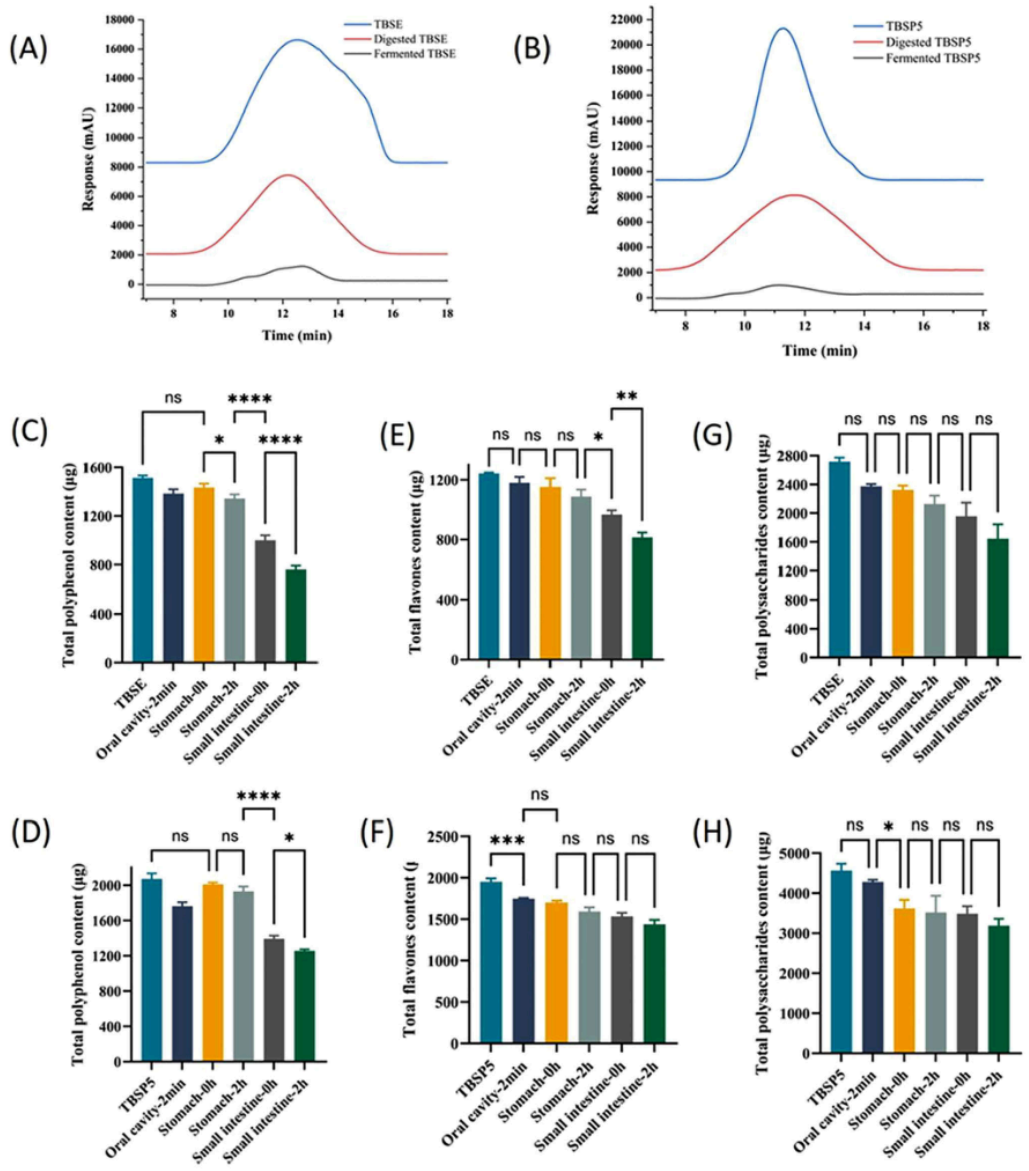


FIGURE 4 | Changes in TBSE during in vitro digestion: total polyphenols content (A), total flavonoids content (B), polysaccharides content (C), and relative molecular weight changes in TBSE after in vitro digestion and fermentation (D). Changes in TBSP5 during in vitro digestion: total polyphenols content (E), total flavonoids content (F), polysaccharides content (G), and relative molecular weight changes in TBSP5 after in vitro digestion and fermentation (H). Data are expressed as the mean ± standard deviation of three replicates. $*p < 0.05$, $**p < 0.01$.

groups exhibited a significant reduction in the relative abundance of *Firmicutes*, thereby decreasing the F/B ratio. Previous studies have suggested that TBs may alleviate hyperglycemia by lowering the F/B ratio of the gut microbiota (Yue et al. 2022).

The 10 most abundant genera were identified and are shown in Figure 6A. Compared with the BLK group, both the TBSE and TBSP5 treatments significantly reduced the relative abundances of *Parasutterella* and *Collinsella*, while markedly increasing the relative abundance of *Bifidobacterium*, a genus widely recognized

图 7：茶褐素在口腔、胃、小肠模拟消化过程中分子量及多酚/黄酮/多糖含量的变化。 来源：Liu H 等，*J Food Sci*, 2026，第 12 页（Fig. 4）。

3.2 结肠：被肠道菌群发酵利用

- **Liu H 等，2026，*J Food Sci***： 体外粪便厌氧发酵 24 h 后，茶褐素中茶多糖被细菌消化利用（HPGPC 峰面积下降）；TBSP5 组乙酸 62.43 mM、丙酸 7.47 mM，显著高于葡萄糖组； 菌群结构显著改变：降低厚壁菌门、降低 F/B 比，下调 *Parasutterella* 与 *Collinsella*，上调双歧杆菌（*B. longum*、*B. pseudocatenulatum*）。

  > "B. longum and B. pseudocatenulatum were strongly positively correlated with acetate, propionate, and isobutyrate (R = 0.936 for B. longum–acetate)."（讨论，p.14）

- **张凤，2024**： 高脂小鼠经茶褐素干预后，H-TBs-a 组 F/B 值最接近正常组；颤螺菌属（*Oscillospira*，产丁酸）丰度显著升高， 脱硫弧菌科（影响次级胆汁酸生成）丰度被调节。

  > "除 M-TBs-a 组，其余茶褐素干预组均能逆转高脂饮食小鼠的 F/B 比率……颤螺菌属与体重、TG、TC、LDL-C、OGTT-AUC、ITT-AUC 均呈负相关。"（3.4.3.2，p.55–57）

3.3 代谢物：短链脂肪酸与胆汁酸

- **短链脂肪酸（SCFAs）**：茶褐素促进乙酸、丙酸产生；侯艳论文中普洱茶和茶褐素干预后，MS 小鼠粪便总 SCFA 异常升高被显著回调。

3	异丁酸	0.96	96	0.97	97
4	丁酸	1.05	105	1.06	106
6	异戊酸	1.01	101	1.02	102
7	戊酸	0.99	99	1.01	101
7	己酸	0.99	99	1.02	102

在本研究中，实验样本前后各一个 QC（浓度为 1 mg/L），用于监测和评价系统的稳定性。图 6-1 和表 6-2 显示，各 QC 样本总离子流图的出峰面积和保留时间基本一致，QC 回收率为（80%~120%），说明 GC-MS 系统在样品分析中具有较高的重现性和稳定性。

6.2.2 普洱茶和茶褐素对 MS 小鼠粪便短链脂肪酸含量的影响

利用 GC-MS 对普洱茶和茶褐素干预 12 W 的健康对照小鼠和 MS 小鼠粪便中的 SCFAs 进行定量分析，结果见图 6-2。

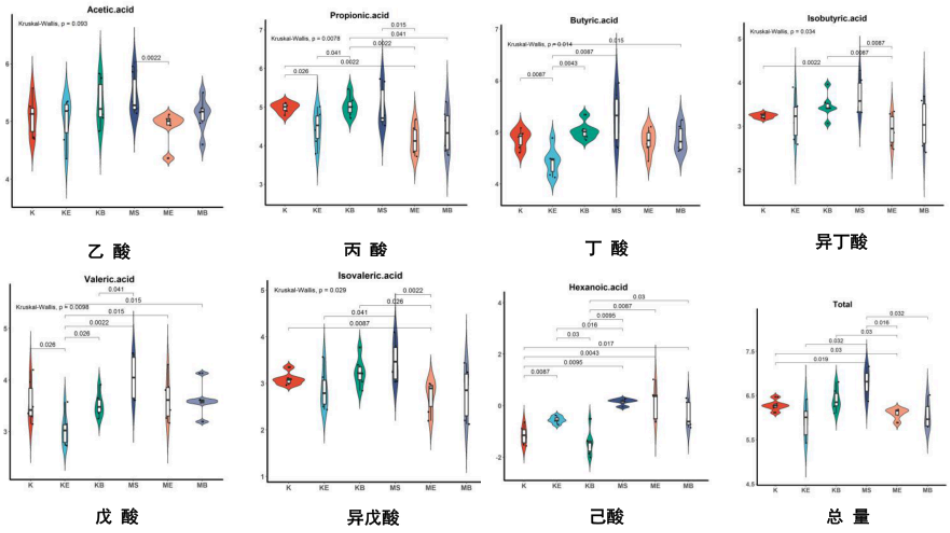


图 6-2 普洱茶和茶褐素对 MS 小鼠粪便短链脂肪酸含量的影响
Fig. 6-2 Effects of pu-erh tea and theabrownin on fecal short-chain fatty acid content in MS mice

图 6-2 显示，MS 小鼠粪便总短链脂肪酸含量上升，MS 组含量显著高于 K 组（$P<0.05$），普洱茶和茶褐素干预 MS 小鼠后显著降低（$P<0.05$），MS-PE 效果最为显著，并显著低于 K 组（$P<0.05$）；所有短酸在 MS 小鼠体内的含量均升高，其中 isobutyric acid（异丁酸）、

中国知网 https://www.cnki.net

图 8：普洱茶和茶褐素对代谢综合征小鼠粪便乙酸、丙酸、丁酸等短链脂肪酸含量的影响。 来源：侯艳，2023，第 170 页（图 6-2）。

- **胆汁酸**：茶褐素可在小肠结合胆汁酸（甘氨胆酸钠 51.58%、牛磺胆酸钠 39.90%，Liu X 等，2025，*Foods* 14:2855）， 并调节胆汁酸肠肝循环；次级胆汁酸（DCA、LCA、6-

KLCA）和初级胆汁酸（CA、α-MCA）比例被重塑（Wei K 等，2026）。 综述进一步指出胆汁酸-FXR 信号通路是茶褐素降胆固醇/抗肥胖的核心代谢轴（Liu C 等，2025）。

3.4 宿主代谢通路

根据 Liu C 等（2025）综述及多项原始研究，茶褐素通过以下主要宿主通路发挥作用：

- **胆汁酸/FXR 轴**：菌群重塑→胆汁酸代谢重编程→FXR 信号→降胆固醇/抗脂肪生成（Huang 2019 *Nat Commun*；Liu C 2025）。
- **SCFA/PPAR-γ/NF-κB 轴**：SCFAs 激活 PPAR-γ、抑制 NF-κB，介导抗炎与免疫调节（结肠炎模型）。
- **AhR/IL-22 轴**：调节色氨酸代谢→激活芳香烃受体 AhR→促进 IL-22→强化肠道屏障与先天免疫。
- **AMPK-PGC1α 轴**：改善线粒体能量代谢（NASH 模型）。
- **PI3K-AKT/NF-κB/NLRP3 轴**：在降尿酸肾损伤、结直肠癌、结肠炎中被反复验证。
- **Nrf2/ARE 抗氧化通路**：抑制 ROS、上调抗氧化酶。

3.4.1 胆汁酸-FXR-FGF15 机制（Huang 2019 详细机制链）

Huang F 等，2019，*Nature Communications* 首次完整揭示了茶褐素降胆固醇的分子机制链：

1. **抑制 BSH 产酶菌**：茶褐素降低小鼠回肠和人粪便中 *Lactobacillus*、*Bacillus*、*Streptococcus*、*Lactococcus*、*Enterococcus*、*Leuconostoc* 等产生胆汁盐水解酶（BSH）的菌属丰度，并降低 BSH 酶活（Fig. 3）。
2. **结合型胆汁酸回肠蓄积**：BSH 活性下降→回肠结合型胆汁酸（TCDCA、TUDCA、GCDCA、GUDCA）水解减少而蓄积。
3. **抑制肠道 FXR-FGF15/FGF19 信号**：结合型胆汁酸作为 FXR 拮抗剂，抑制回肠 FXR 核转位与 FGF15/FGF19 表达；血清 FGF15（小鼠）/FGF19（人）下降。
4. **解除肝脏胆汁酸合成抑制**：FGF15-FGFR4 信号减弱→肝脏 CYP7B1、CYP27A1（替代通路）及 CYP7A1 表达升高；同时 CDCA 增多→激活肝 FXR-SHP→选择性抑制 CYP8B1（经典通路），进一步放大替代通路，产生更多 CDCA 而非 CA。
5. **结局**：肝脏胆汁酸从头合成增加 + 粪便胆汁酸排泄增加 → 肝脏胆固醇消耗增加 → 血清/肝脏胆固醇下降。

该机制经过粪菌移植（FMT）、FXR 激动剂/拮抗剂、重组 FGF19 及 AAV-shRNA 敲低 CYP7B1/CYP7A1 等多重因果验证，其中 **CYP7B1（替代通路）被证实为关键效应分子。**

Fig. 7 FXR-FGF15 and FXR-SHP regulated BA synthesis in alternative pathway. **a** Dominant BAs in liver and ileum of HFD fed mice gavaged with 225 mg/Kg/day theabrownin and 50 mg/Kg/day TCA, TCDCA, TUDCA respectively for 8 weeks. $n = 8$ individuals/group. **b** mRNA expression of ileal FXR, FGF15 and hepatic FXR, SHP and BA synthetic genes in HFD fed mice gavaged with 225 mg/Kg/day theabrownin and 50 mg/Kg/day TCA, TCDCA, TUDCA respectively for 8 weeks. $n = 8$ individuals/group. **c** Immunofluorescence staining of ileal FXR, FGF15 and hepatic FXR, SHP of HFD fed mice gavaged with 225 mg/Kg/day theabrownin and 50 mg/Kg/day TCA, TCDCA, TUDCA respectively for 8 weeks (scale bar, 50 μm). **d** Fifty micromole per liter of TCDCA and TUDCA inhibited protein expression of nuclear FXR in the human FHs 74 Int and L02 cell lines, and 50 μM of CDCA and TCA activated the expression of nuclear FXR in human FHs 74 Int and L02 cell lines and promoted expression of CYP27A1 and CYP7B1 in alternative pathway of bile acids synthesis. $n = 3$ individuals/group. **e** Immunofluorescence staining of FXR, FGF19 in FHs 74 Int cells supplied with 50 μM CDCA, CDCA coupled with 50 μM of TCDCA, CDCA coupled with 50 μM of TUDCA, and 50 μM of TCA for 24 h (scale bar, 100 μm). **f** Immunofluorescence staining of FXR, SHP in L02 cell lines supplied with 50 μM of CDCA, CDCA coupled with TCDCA, CDCA coupled with TUDCA and TCA for 24 h (scale bar, 50 μm). **g** The mRNA expression of ileal FXR, FGF15 and hepatic BA synthetic genes in HFD fed mice gavaged with 225 mg/Kg/day theabrownin, theabrownin coupled with 100 mg/Kg/day fexaramine for 8 weeks. $n = 8$ individuals/group. **h** The mRNA expression of ileal FXR, FGF15 and hepatic BA synthetic genes in HFD fed mice gavaged with 225 mg/Kg/day theabrownin, theabrownin coupled with 50 μg/Kg/day recombinant FGF19 protein by intraperitoneal injection for 8 weeks. $n = 8$ individuals/group. The mRNA expression was normalized to GAPDH. Protein expression was normalized by β-Actin or Lamin B1. Data were expressed as mean ± SEM. Differences between data were assessed by the Mann–Whitney U test, $^{*}p < 0.05$, $^{\#}p < 0.005$

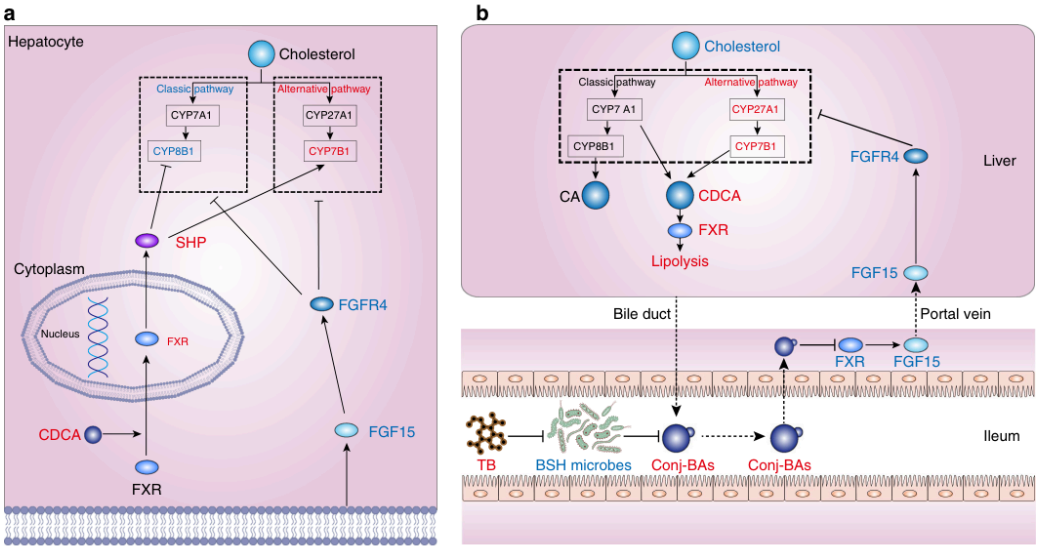


Fig. 8 Proposed mechanism for theabrownin reduce cholesterol level. **a** Theabrownin promoted alternative bile acids synthetic pathway by simultaneous inhibition of intestinal FXR-FGF15 signaling with TCDCA or TUDCA and activation of hepatic FXR-SHP signaling with CDCA.Intestinal FXR-FGF15 signaling inhibits hepatic bile acid synthesis genes CYP7A1, CYP8B1, CYP27A1 and CYP7B1 with no selectivity, whereas hepatic FXR-SHP signaling selectively inhibits CYP8B1 in the classic pathway. Theabrownin in Pu-erh tea induced TCDCA and TUDCA in distal ileum to inhibit FGF15 production and thus, activated bile acid synthesis enzymes. Additionally, increased CDCA production in the liver promoted nuclear FXR expression as well as SHP to inhibit CYP8B1 expression. Ultimately, the combined regulation of decreased intestinal FXR-FGF15 and increased hepatic FXR-SHP on bile acid synthesis enzymes resulted in increased expression of CYP7B1 in alternative pathway and decreased expression of CYP8B1 in classic pathway leading to increased production of CDCA rather than CA. **b** Proposed mechanism for theabrownin induced attenuation of hepatic cholesterol levels via modulation of gut microbiome-mediated bile acid metabolism. Primary BAs, mainly CA and CDCA, were produced in the liver from cholesterol and conjugated to glycine or taurine to form conjugated BAs and then secreted to the intestinal. BSH enzymes are produced in intestinal microbes and function to hydrolyze conjugated BAs into unconjugated BAs. BSH microbes were suppressed by theabrownin in Pu-erh tea resulting in the accumulation of conjugated BAs in distal ileum. Conjugated BAs inhibited intestinal FXR-FGF15 signaling which subsequently alleviated the suppression of BA synthesis gene expression by this signaling pathway, resulting in increased BA production in alternative synthetic pathway and fecal BA excretion and ultimately, decreased cholesterol levels

βMCA[34]. As reported, the BSH activity was mainly attributed to Lactobacilli and Bifidobacteria[35–37]. Our experimental analyses of the gut microbiota showed that there were decreased relative abundances of BSH-producing *Lactobacillus, Bacillus, Streptococcus and Lactococcus* genera in humans and mice with Pu-erh/theabrownin treatment as well as decreased BSH activity in the ileal lumen of mice. Additionally, when theabrownin was administered to HFD fed mice, intestinal BSH activity and serum and liver cholesterol levels were significantly reduced. These results revealed that Pu-erh tea derived theabrownin directly inhibited the activity of intestinal BSH enzymes, suppressed BA deconjugation in the small intestine, and impacted hepatic and serum cholesterol levels.

BAs are also endogenous signaling molecules that bind to the BA receptors, FXR and TGR5, to regulate BA homeostasis in the enterohepatic circulation, modulate cholesterol and triglyceride metabolism, and maintain glucose and energy homeostasis[38,39]. Potent endogenous ligands of FXR include mainly unconjugated

图 14：Huang 2019 提出的茶褐素降胆固醇机制总图。 a 图：肠道 FXR-FGF15 与肝 FXR-SHP 对经典通路（CYP8B1）和替代通路（CYP7B1/CYP27A1）的选择性调控； b 图：TB→抑制 BSH 菌→回肠结合型 BA 蓄积→肠道 FXR-FGF15↓→肝替代 BA 合成↑+粪便 BA 排泄↑→胆固醇↓的完整流程。 来源：Huang F 等，*Nature Communications*, 2019，第 12 页（Fig. 8）。

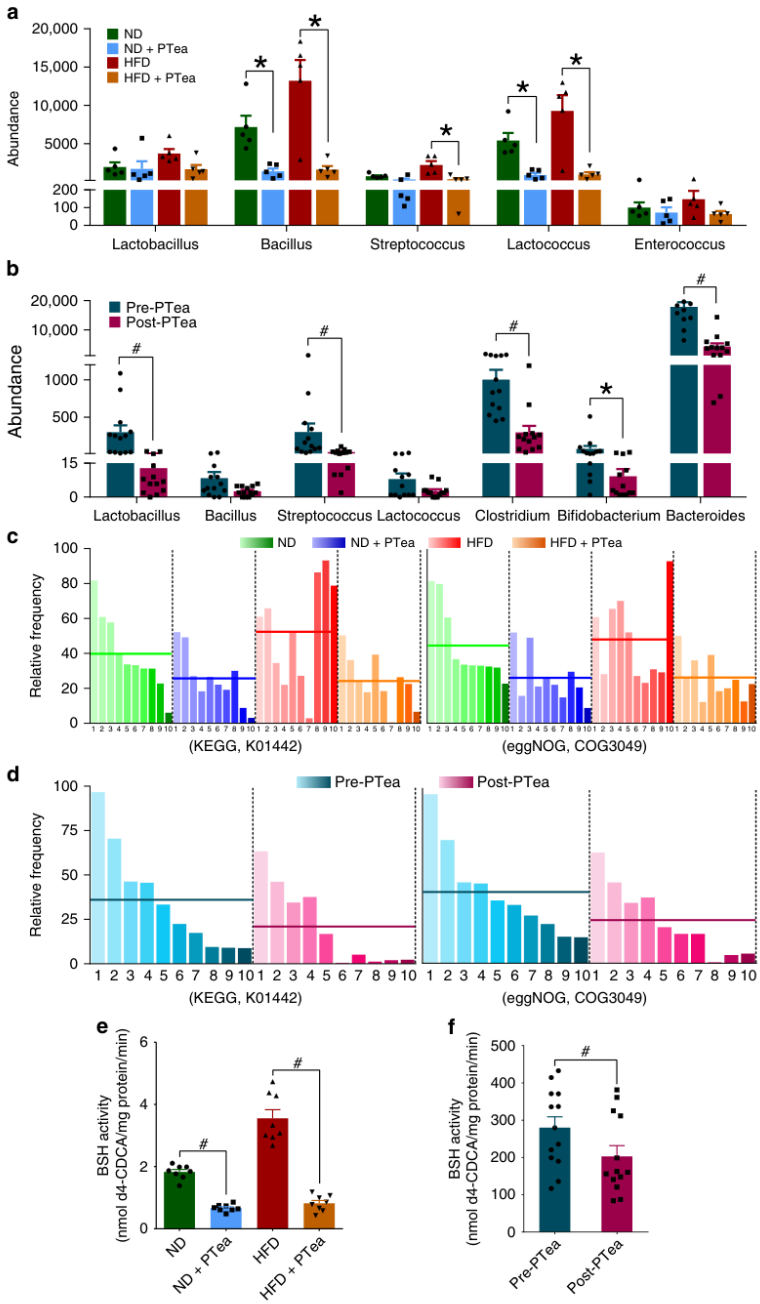


Fig. 3 Pu-erh tea reduced the abundance of BSH microbes and BSH activity. **a** Abundance of BSH enriched microbes in ileum of mice undergoing Pu-erh tea consumption. $n = 5$ individuals/group. **b** Abundance of BSH enriched microbes in feces of humans consuming Pu-erh tea. $n = 13$ individuals/group. **c** The microbial BSH enzyme activity in the ileal contents of mice treated with Pu-erh tea. **d** The microbial BSH activity in human feces from subjects on Pu-erh tea consumption. The functional profiles were identified by metagenomic analysis utilizing both KEGG and eggNOG databases. BSH related proteins ranked in the top ten are shown and the mean abundances are illustrated as the horizontal line in each group. **e** Ileal BSH activity in mice on Pu-erh tea consumption. $n = 8$ individuals/group. **f** Fecal BSH activity inhuman subjects on Pu-erh tea consumption. $n = 13$ individuals/group. Data were expressed as mean ± SEM. Differences in the data for mice and human subjects were assessed by the Mann–Whitney U test and Wilcoxon rank-sum test, respectively, $^{*}p < 0.05$, $^{\#}p < 0.005$

图 15：普洱茶/茶褐素降低小鼠回肠和人粪便中 BSH 产酶菌丰度及 BSH 酶活性。 来源：Huang F 等，*Nature Communications*, 2019，第 5 页（Fig. 3）。

Table 4
Effects of TB (or related dark teas) in alleviating inflammation and regulating immunity in in vivo and in vitro studies.

Disease	Subjects	Experimental models	Source and Dosage	Mechanism of action	References
Colitis	C57BL/6 mice	3.5 % dextran sulfate sodium (DSS) induced ulcerative colitis (UC)	Ripened Pu-erh tea: 3 % , 6 %, and 9 %	Inhibited the release of pro-inflammatory cytokines (TNF-α , IL-1β and IL-6), reversed the dysbiosis state of gut microbiota and mediated the SCFAs production and PPAR-γ/NF-κB pathway in intestinal epithelial cells	(Huang et al., 2021)
	C57BL/6 mice	5 % DSS induced UC	Ripened Pu-erh tea: 1215 mg/kg·bw/day	Inhibited the release of pro-inflammatory cytokines, restored intestinal barrier function (increased occludin, claudin-1 and MUC2), promoted the balance of Treg/Th17, restoring intestinal microbial homeostasis	(Zhao et al., 2024)
	Female C57BL/6 mice	3.5 % DSS induced UC	TB from Pu-erh tea: 300 mg/kg·bw/day	Regulated the activation of Nrf2/ARE and the TLR4/MyD88/NF-κBP65 pathway, recovery of SCFAs levels; reduced the abundance of harmful microbiota, restore colon shortening, enhance antioxidant capacity and reduced Bcl-2/Cytc/ Cleaved-caspase 3 regulated apoptosis	(Wei et al., 2023)
	Female C57BL/6 mice	3.5 % DSS induced UC	TB from Ripened Pu-erh tea: 3, 4, or 7 mg/mL/ day	Ameliorated severe crypt damage, downregulated the protein of inflammatory cytokines (IL-1β, IL-6 and TNF-α), suppressed macrophage aggregation into colon by reducing expression levels of CD 68 + and CD 11b, and modulated PPAR-γ and NF-κB signaling pathways	(Cai et al., 2023)
	Male C57BL/6 mice	1.5 % DSS induced chronic UC	TB from Fuzhuan brick tea: 400 mg/kg bw/day	Elevated microbial tryptophan metabolites (IAld and IAA) levels by selectively promoting *Lactobacillus* growth; activated AhR and enhanced IL-22 production to repair the intestinal barrier; alleviated UC by targeting the gut microbiota involved in the AhR pathway.	(Yang et al.)
Atherosclerosis	ApoE−/− mice	High-fat diet (HFD) induced aortic atherosclerosis	TB from Pu-erh tea: 675 mg/kg·bw/day	Reduced blood lipid levels, decreased the number of macrophages, lymphocytes within AS plaques and lowered the expression of inflammatory cytokines	(Zhong et al., 2020)
	Sprague-Dawley (SD) rats	High-sugar model induced obesity	TB from Pu-erh tea: 1124 mg/kg·bw/day	Increased the diversity of cecal microorganisms and promoted	(Yue et al., 2019)
	SD rats	HFD induced aortic atherosclerosis	*Monascus purpureus*-fermented Pu-erh tea: 125, 250, 500 mg/ kg·bw/day	Reduced TG, TC and LDL-C, lowered inflammatory factor expression	(Deng et al., 2021)
Nonalcoholic steatohepatitis	Male C57BL/6 J mice	Methionine/choline deficient induced Nonalcoholic fatty liver disease	TB: 200 mg/kg·bw/day	Improved steatosis and liver damage, alleviated liver oxidative stress and fibrosis, increased hepatic level of FGF21 and reduced the phosphorylation of p38	(Zhen et al., 2023)
	Male C57BL/6 J mice	HFD induced Nonalcoholic steatohepatitis	TB from Pu-erh tea: 2300 mg/kg·bw/day	Improved fatty acid oxidation, lipolysis, and oxidative stress, and increased the abundance of serotonin-related gut microbiota, such as *Akkermansia*, *Bacteroides* and *Parabacteroides*	(Li, Huang, et al., 2023)
	Male C57BL/6 J mice	HFD induced Nonalcoholic steatohepatitis	Pu-erh: 450 mg/kg/d TB from Pu-erh tea: 225 mg/kg·bw/day	Reduced the levels of Cer (d18:1/16:0) in the intestine and liver, reduced hepatic TNF-α and IL-6 expression levels, downregulated transcriptional expression of Cer synthase and inhibited the intestinal Ferchs-Serge synthetase pathway	(Wang, Li, Huang, et al., 2024)
	Male C57BL/6 J mice	HFD induced metabolic dysfunction-associated liver disease (MASLD)	TB from Qingzhuan brick tea: 180, 360 mg/ kg·bw/day	Increased the relative abundance of *Bacteroides*, *Blautia* and *Lachnoclostridium* and their main metabolites acetate and propionate, up-regulated the expression of ATGL, PPARα, FFAR2 and FFAR3, and inhibited the expression of LXRα, SREBP-1c, FAS and HMGCR genes.	(Chen, Xie, et al., 2024)
Innate immunity	RAW264.7 cell	Lipopolysaccharide induced inflammation	TB from Ripened Pu-erh tea: 62.5, 125, 250, 500, 1000, 1500 μg/mL	Modulated LR/MAPK/NF-κB/PI3K–AKT signal pathway and modulated the immune response and inflammation	(Zhao et al., 2023)
Inflammatory inhibition in other metabolic disease interventions	Male C57BL/6 J mice	HFD induced obesity disorders	TB from raw Pu-erh and ripened Pu-erh tea:400 mg/kg·bw/day	Improved insulin sensitivity and glucose homeostasis, regulated the lipid level and reduced the chronic inflammation	(Deng et al., 2023)
	Male C57BL/6 J mice	HFD induced obesity disorders	TB from Wuniuzao dark tea: 150, 300 mg/ kg·bw/day	Regulated the *Srebp* lipid metabolism pathway and bile acid metabolism process, and improved the gut microbiota composition	(Xu et al., 2023)
	Male Syrian golden hamster	High-lipid diet induced hyperlipidemia	TB from Pingwu Fuzhuan brick tea: 400, 800 mg/kg·bw/day	Improved gut microbiota dysbiosis and alterated microbiota-derived metabolites and regulated FoxO and PPAR pathways	(Xiao et al., 2024)
	Db/db and db/ m mice	High-lipid diet induced metabolic syndromed	TB from Pu-erh tea: 1215 mg/kg·bw/day	Improved the physiological functions of liver, jejunum and adipose tissues, regulated the	(Hou et al., 2022)

(*continued on next page*)

图 9：Liu C 等综述中 Table 4（节选）汇总了茶褐素在结肠炎、动脉粥样硬化、NASH、先天免疫等疾病模型中的剂量与作用机制。 来源：Liu C 等，*Food Research International*, 2025，第 7 页（Table 4）。

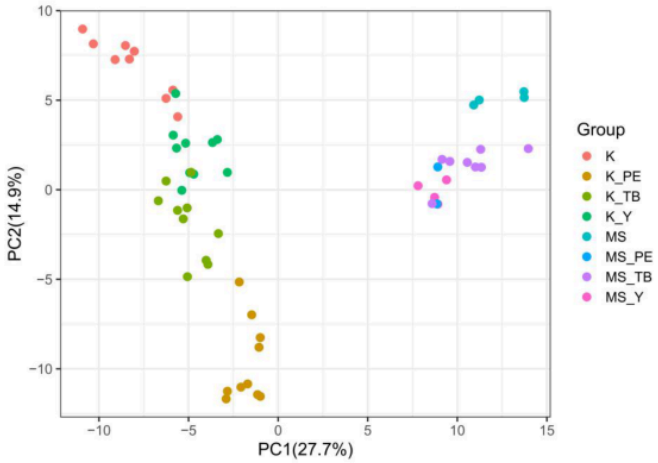
3.5 “菌-肠-肝-脑”轴：茶褐素全身代谢调节的核心框架

侯艳（2023）博士论文首次系统提出该多组学轴：

> "肠道微生物（Cutibacterium_acnes, Clostridiales_bacterium 42_27, Firmicutes_bacterium ASF500 和 Blautia_coccoides）可能与肝脏昼夜节律基因（Clock、Npas2、Cry1、Cry2、Per2、Per3 和 Nfil3）、SCFAs 转运基因（Slc16a1、Slc16a3、Slc16a7 和 Slc22a7），以及 SCFAs 受体基因（Olfr78）相互作用。"（第 7 章 7.4 小结，p.197）

具体链条可概括为：

1. 茶褐素进入结肠，选择性富集产丁酸菌（ASF500、梭菌 42_27、布氏球菌）并抑制条件致病菌（泡囊短波单胞菌等）。
2. 菌群变化调节粪便 SCFA 谱，经 SCFA 转运体/受体（Slc16a 家族、Olfr78）向肝脏和中枢传递信号。
3. 肝脏节律基因（Clock/Npas2/Cry/Per）与脂质代谢通路被调控，炎症与氧化应激下降。
4. 血清和脑组织甘油磷脂、亚油酸、嘌呤代谢显著改变，从而改善代谢综合征。



B．不同组学筛选出的具有相关性显著差异物的 PCA 图

图 7-3 基于 Rdcv PLS 的转录组和代谢组微生物关联分析筛选重要标志物

Fig.7-3 Screening of important biomarkers using transcriptome and metabolome microbial association analysis based on Rdcv PLS

在皮尔逊相关系数（Pearson correlation coefficients）>0.6 的条件下，我们筛选出了不同比较中的重要信息。rdcv PLS 分析筛选到 71 种血清代谢物、32 种脑组织代谢物，81 种肝脏基因和 20 种肠道微生物（图 7-3A，表 7-1）。此外，这些组合数据的 PCA 可以区分 MS 组和 MS 干预组，以及 K 组和 K 干预组（图 7-3B）。结合 rdcv PLS 分析我们发现 6 种重要物种 *Clostridiales_bacterium 42_27*,*Firmicutes_bacterium_ASF500*,*Blautia_coccoides*, *Brevundimonas_vesicularis*, *Cutibacterium_acnes*, *Desulfovibrio_sp_UNSW3caefatS* 与肝脏昼夜节律基因（*Npas2*, *Nr1d1*, *Cry2*, *Per3*, *Arntl*）相关，重要的是普洱茶和茶褐素干预后氨基酸、嘌呤衍生物和脂质代谢（N,N-dimethylglycine（N，N-二甲基甘氨酸）, deoxyguanosine（脱氧鸟苷）, deoxycholic acid (+)（脱氧胆酸（+））, beta-guanidinopropionic acid（β-鸟苷丙酸）, 12-hydroxydodecanoic acid（12-羟基十二烷酸）, 2-deoxyycastasterone（2-脱氧胆固醇）与血清和脑组织代谢相关。

中国知网 https://www.cnki.net

图 10：基于 Rdcv PLS 的转录组-代谢组-微生物关联分析筛选的重要标志物（6 种关键菌与 81 个肝脏基因、71 个血清代谢物、32 个脑组织代谢物）。 来源：侯艳，2023，第 179 页（图 7-3）。

3.6 排泄

关于茶褐素原形及代谢产物的排泄，研究证据可分为以下几个层面：

- **粪便胆汁酸排泄增加（直接证据）**： Huang 2019 中，普洱茶/茶褐素干预小鼠**粪便总胆汁酸流失显著增加**，胆汁酸池增大；人体试验亦观察到粪便胆汁酸排泄上升（Supp.Fig. 7b）。 这是目前茶褐素功效研究中与"排泄"最直接相关的证据，但排泄物是胆汁酸而非茶褐素原形本身。

  > "...increased elimination of BAs via the feces."（Discussion，p.12，Fig. 8b）

- **茶褐素原形经粪便排出（间接/推断证据）**： 体外模拟消化显示茶褐素在上消化道结构稳定、生物可及性低（Liu H 2026），说明大部分茶褐素大分子可到达结肠； 结肠菌群发酵后部分骨架可能以原形或高分子片段随粪便排出。但目前尚无量化的物料平衡或同位素示踪数据直接测定茶褐素原形排泄比例。
- **小分子代谢物经尿液/胆汁排泄（类比与机制推断）**： 茶褐素在结肠被菌群水解后可释放酚酸、没食子酸、焦棓酚类小分子，这些代谢物可经肠肝循环进入胆汁，或吸收入血后经尿液排泄。 详见配套报告《茶褐素排泄途径补充文献检索》。

研究缺口提示：目前仍缺乏针对茶褐素原形及特征代谢物经尿液、胆汁、粪便的定量药代动力学/物料平衡研究，尤其是同位素示踪或高灵敏度代谢组学证据。 这是"体内代谢途径"中尚需补强的关键环节。关于该缺口的补充检索，可参阅同日生成的《茶褐素排泄途径补充文献检索.html》。

4. 关键图片一览

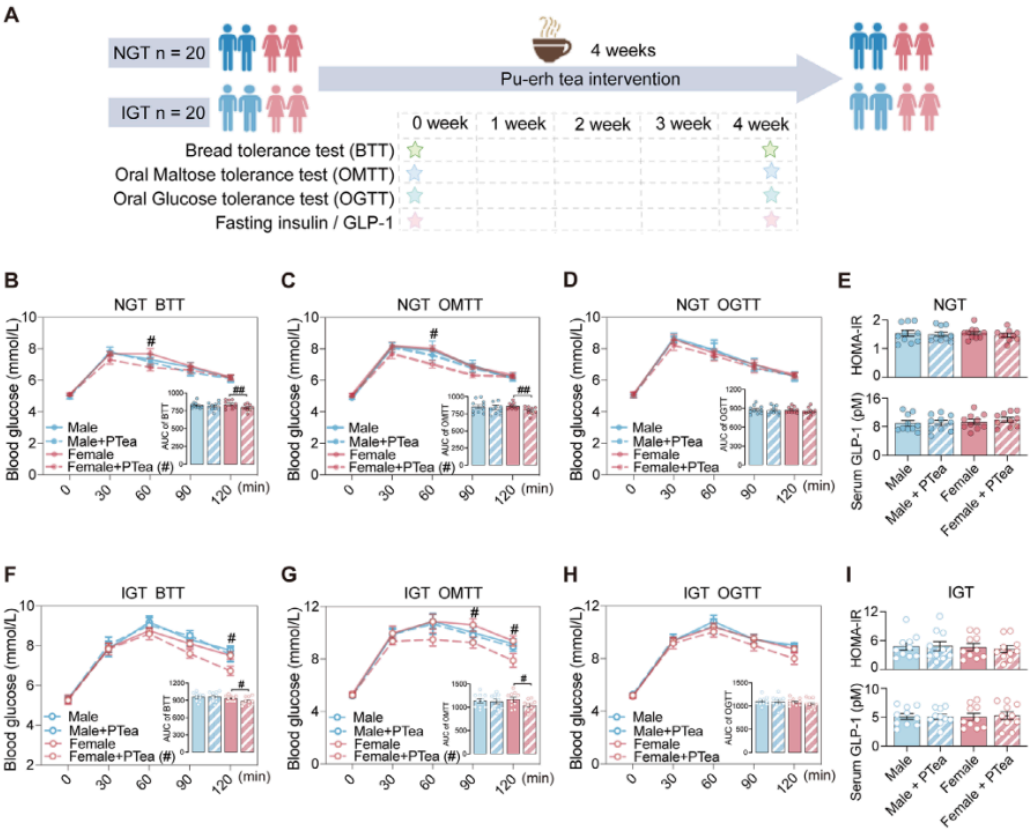


FIGURE 1 | Pu-erh tea regulated glucose homeostasis with sex differences in the NGT and IGT individuals. (A) Diagram of human study 1: After enrolment and adaptation period, the NGT (n = 20, 10 individuals of each sex) and IGT (n = 20, 10 individuals of each sex) individuals consumed Pu-erh tea for 4 weeks. The assessments are displayed in the diagram. (B–E) (B) Blood glucose levels and AUC of BTT, (C) blood glucose levels and AUC of OMTT, (D) blood glucose levels and AUC of OGTT, and (E) HOMA-IR and serum active GLP-1 levels in the NGT individuals. (F–I) (F) Blood glucose levels and AUC of BTT, (G) blood glucose levels and AUC of OMTT, (H) blood glucose levels and AUC of OGTT, and (I) HOMA-IR and serum active GLP-1 levels in the IGT individuals. Data are presented as the mean ± SEM. $^{\#}p < 0.05$ and $^{\#\#}p < 0.01$ compared with the female groups with Pu-er tea based on the Wilcoxon matched-pairs signed rank test.

In the HFD+STZ diabetic mouse model, mice were treated with the same doses of instant Pu-erh tea infusion used in the HFD model for 4 consecutive weeks (Figure 2F). The result also showed no significant changes in body weight, dietary, and water intake compared to Pu-erh tea treatment with control groups (Figure S3A–C). Similar to the HFD model, Pu-erh tea improved glucose levels in the OMTT (Figure S3D), particularly in female mice (Figure 2G). However, there were no significant changes in the OGTT (Figure 2H; Figure S3E), ITT (Figure 2I; Figure S3F), fasting serum insulin (Figure S3G,H), HOMA-IR (Figure 2J; Figure S3I), and serum GLP-1 levels (Figure 2J; Figure S3J) in this model.

To verify that Pu-erh tea improves PBG levels by inhibiting α-glucosidase in the small intestine, we measured the mucosal enzymatic activity across different physiological segments of the small intestine in both mouse models. Our results showed that α-glucosidase inhibition was significantly greater in female mice treated with Pu-erh tea compared to male mice in the duodenum (Figure S3K,L), jejunum (Figure 2K,L), and ileum (Figure S3M,N).

To pinpoint the specific compounds in instant Pu-erh tea responsible for its blood glucose-lowering effects and inhibition of α-glucosidase activity, we conducted a quantitative analysis of tea polyphenols and tea pigments in the small intestinal contents of mice treated with HFD and Pu-erh tea using quadrupole time-of-flight mass spectrometry (UPLC/QTOFMS). Among the various components present in the tea, TB demonstrated the most significant and negative correlation with blood glucose levels at 15 and 120 min during the OMTT, as well as with the α-glucosidase activity in the small intestine (Figure 2M).

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图 11：普洱茶茶褐素在女性正常及 IGT 人群中降低餐后血糖，并设计 BTT/OMTT/OGTT 评估方案。 来源：Li Y 等，*Advanced Science*, 2026，第 3 页（Fig. 1）。

5. 核心文献速查表

文献	模型/剂量	主要功效	关键机制	关键位置
Huang F 等，2019，*Nat Commun*	小鼠 225 mg/kg 纯化 TB×8 w + 13 名男性 50 mg/kg 即溶普洱×4 w	降胆固醇、降血脂、减重	BSH 菌↓→结合型 BA↑→肠道 FXR-FGF15↓→CYP7B1/CYP27A1↑→粪便 BA 排泄↑	摘要 p.1；Fig. 8 p.12；Fig. 3 p.5
Ding X 等，2025，*Int J Biol Macromol*	HUA 小鼠 + 临床男性 12 周	降尿酸、护肾	XOD↓、OAT1/3↑、PI3K-AKT/NF-κB/NLRP3↓	摘要 p.1；Fig. 5 p.8
Li Y 等，2026，*Adv Sci*	女性人群 + 高血糖小鼠	降餐后血糖	α-葡萄糖苷酶抑制、雌激素-MUC2	摘要 p.1；Fig. 1 p.3
Jensen GS 等，2016，*CIA*	RCT n=59 高胆固醇北美人群，普洱茶提取物 3 g/d×20 w	减脂、改善血脂	TC↓、TG−20%、DEXA 总脂肪/gynoid 区减脂	摘要 p.367；Fig. 3 p.6；Fig. 4 p.7
Yang TY 等，2014，*Phytother Res*	RCT n=70 代谢综合征，普洱茶提取物 1.5 g/d×3 月	男性亚组减重	男性体重/BMI 显著下降（−2.05 kg）	摘要 p.1096；Results p.1098；Conclusion p.1101
Lei M 等，2026，*Foods*	自然衰老小鼠	认知改善	肠-脑轴、乙酸/3-羟基丁酸↑	摘要 p.1；Fig. 4 p.10
Gao Y 等，2025，*Phytomedicine*	烧伤小鼠 + HK2 细胞	改善 AKI、降低死亡率	GAA/FA↑、抑制凋亡与铁死亡	摘要 p.1；Fig. 9 p.12
Jin W 等，2018，*J Cell Mol Med*	U2OS + 斑马鱼移植瘤	抗骨肉瘤	DNA 损伤、p53 通路激活	摘要 p.1

文献	模型/剂量	主要功效	关键机制	关键位置
Leung HKM 等，2025，*Sci Rep*	AOM/DSS 结直肠癌小鼠	抑制 CAC	PI3K/AKT/cyclin D1↓、丙酸↑	结果 p.5；Fig. 5 p.10
Wei K 等，2026，*Food Res Int*	DSS 结肠炎 FMT	抗结肠炎	菌群→SCFAs/胆汁酸→Nrf2/TLR4-NF-κB	摘要 p.1；讨论 p.10–11
Liu H 等，2026，*J Food Sci*	体外消化+粪便发酵	类益生元	上消化道不吸收、结肠产 SCFA、富集双歧杆菌	Fig. 4 p.12；Table 3 p.13
侯艳，2023，博士论文	db/db 小鼠 1.215 g/kg×12w	改善代谢综合征	菌-肠-肝-脑轴、昼夜节律、SCFA 受体	摘要 p.9；图 6-2 p.170；图 7-3 p.179
张凤，2024，硕士论文	高脂 C57BL/6J 小鼠	降糖降脂	F/B 比↓、颤螺菌属↑、胆汁酸线索	3.4.2.3 p.61–62；图 3-46 p.73
龚加顺等，2007，*茶叶科学*	昆明种小鼠	降胆固醇、抗疲劳、安全	LD_{50}>10 g/kg 实际无毒	摘要 p.1；表 1 p.3
纪旭等，全国营养科学大会摘要	离体大鼠胸主动脉	舒张血管	内皮非依赖性、Ca^{2+}/平滑肌	p.320
Kim S 等，2022，*Toxics*	SD 大鼠	安全性	LD_{50}=21.68 g/kg；4000 mg/kg 肾毒性	摘要 p.1–2
Wu SC 等，2011，*J Ethnopharmacol*	SD 大鼠 91 天	安全性/降脂	LD_{50}>10 g/kg、NOAEL 5000 mg/kg	摘要 p.1–2
Liu C 等，2025，*Food Res Int*	综述	抗炎免疫调节	胆汁酸/FXR、SCFA/PPAR-γ/NF-κB、AhR/IL-22、AMPK-PGC1α	Table 4 p.7–8；Fig. 3 p.4

6. 按研究类型分类

本次梳理共覆盖 **66 份 PDF**（原文件夹 63 份 + 新增 3 篇），去重后得到 **62 篇文献条目**。 其中 57 篇为与茶褐素功效、代谢、结构或安全性相关的有效文献，5 篇为无关/无效文件（下载失败或主题不符）。 分类结果如下：

研究类型	篇数	说明
人体试验	5	含 RCT、开放标签人体观察；部分研究同时含动物实验验证
动物实验	15	小鼠、大鼠、斑马鱼、线虫等体内模型；部分含细胞验证
细胞实验	2	纯体外细胞系研究
综述	3	叙述性综述或药理学综合评述
其他-体外/化学表征	28	结构表征、制备工艺、体外消化/发酵、检测方法、风味品质等
其他-安全性	4	急性/亚急性/亚慢性毒理及致突变试验
无关/无效	5	主题不符或下载失败的伪 PDF/HTML 文件
合计	**62**	—

人体试验（5 篇）

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
人体试验 Weight Reduction Effect of Puerh Tea in Male Patients with Metabolic Syndrome	Yang TY 等，2014	Phytother Res 28:1096	RCT n=70 代谢综合征，普洱茶提取物 1.5 g/d×3 月	男性亚组体重/BMI 显著下降（−2.05 kg），全人群/女性无显著；血脂血糖无改善
人体试验 Reduction of body fat and improved lipid profile (Puer tea extract)	Jensen GS 等，2016	Clin Interv Aging 11:367	RCT n=59 高胆固醇北美非亚裔，普洱茶提取物 3 g/d×20 w	减重约 1 kg、DEXA 总脂肪/gynoid 区减脂、TC 自第 4 周下降、TG 约−20%
人体试验 Effects and mechanisms of	Ding XY 等，2025	Int J Biol Macromol	HUA 小鼠 + 34 名男性受试者	TB 280 mg/kg 降尿酸并修复肾损伤；临床 12

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
theabrownin from black tea in improving hyperuricemia		293:139373	口服 TB 12 周	周后血尿酸较基线降约 7%
人体试验 Sex-Specific Regulation of Glycemic Homeostasis by Theabrownin from Pu-erh Tea	Li Y 等，2026	Adv Sci 13:e19337	IGT/正常女性人群餐后血糖观察 + 高血糖小鼠	TB 降低女性餐后血糖；雌激素上调小肠 MUC2 增强 α-葡萄糖苷酶非竞争性可逆抑制
人体试验 Theabrownin from Pu-erh tea attenuates hypercholesterolemia	Huang F 等，2019	Nat Commun 10:4971	HFD 小鼠 225 mg/kg 纯化 TB×8 w + 13 名健康男性即溶普洱×4 w	血清/肝脏 TC、TG 显著降低；FMT 证实依赖菌群；BSH 菌↓→FXR-FGF15↓→CYP7B1↑→粪便 BA 排泄↑

动物实验（15 篇）

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
动物实验 Age-related cognitive decline mice	Lei MJ 等，2026	Foods 15:1587	自然衰老 C57BL/6J 小鼠，ABX+FMT+代谢物回补	Y-maze 交替率 51.91%→71.59%，经菌群→乙酸/3-HB→肠-脑轴改善认知
动物实验 Theabrownins improve burn-induced kidney injury	Gao Y 等，2025	Phytomedicine 140:156609	小鼠烫伤 AKI + HK2 细胞验证	降低 60 h 死亡率；GAA/FA↑恢复 GPX4，抑制凋亡与铁死亡
动物实验 Tibetan tea	Zhang CX 等，2025	Food Res Int 202:115555	斑马鱼胚胎乙醇致畸模型	100 μg/mL 逆转脊柱畸形；SOD↑、MDA↓、调

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
theabrownin ameliorates ethanol-induced FASD				节磷脂与支链氨基酸代谢
动物实验 Theabrownin + zearalenone suppress colitis-associated CRC	Leung HKM 等，2025	Sci Rep 15:26386	AOM/DSS CAC 小鼠	TZ 提高存活率、减少肿瘤数；PI3K/AKT↓；乙酸/丙酸与肿瘤数负相关
动物实验 Amelioration of colitis by donor microbiota modulated by tea ingredients	Wei K 等，2026	Food Res Int 229:118473	DSS 结肠炎小鼠 FMT	TB 调制菌群→降低 DAI、恢复肠屏障、升高 SCFAs 与次级胆汁酸
动物实验 Comparative study anti-obesity white vs dark tea	Zhou F 等，2025	Food Res Int	HFD 肥胖 SD 大鼠，黑茶 vs 白茶	黑茶（TB 10.97%）降体重/血脂/LPS，上调肠屏障蛋白与抗氧化酶
动物实验 Comparable neuroprotection efficacy of raw vs ripened Pu-erh	Peng LY 等，2026	npj Sci Food	D-gal 诱导衰老小鼠	熟普洱（TB 1035 mg/g）与原料普洱同等改善认知，经肠-脑轴抑制脑氧化应激
动物实验 Does thermal processing improve intestinal antioxidant function	Fan XQ 等，2025	Food Chem 496:146847	氧化鱼油诱导肠道氧化应激大鼠	TB 热稳定，BCL2/BAX 比 +25.6%，维持肠道抗氧化
动物实验 Theabrownin	Li JJ 等，2026	Foods	SD 大鼠氟药代动力学	TB 延缓氟吸收、降低 Cmax 35.38%，促进氟

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
modulates fluoride bioavailability in rats				经粪便排泄
动物实验 普洱茶和茶褐素调节小鼠“菌-肠-肝-脑”轴缓解代谢综合征	侯艳，2023	云南农业大学博士论文	db/db 小鼠 TB 1.215 g/kg×12 w	首次系统提出 TB 经菌-肠-肝-脑轴缓解 MS，调节肝脏节律基因与 SCFA 受体
动物实验 茶褐素的制备、结构表征及其对高脂小鼠糖脂代谢的调节作用	张凤，2024	云南农业大学硕士论文	高脂 C57BL/6J 小鼠 560/1120 mg/kg×12 w	三种工艺 TB 均降 TG/TC/LDL-C；H-TBs-a 增加产丁酸颤螺菌属 Oscillospira
动物实验 普洱茶膳食纤维结构特性及改善便秘作用机理	白晓丽，2022	天津科技大学博士论文	洛哌丁胺便秘小鼠 + TDF 结构表征	TDF 含 TB 9.77%，经菌群-SCFA-肠神经递质轴改善便秘
动物实验 Structural characteristics and nonvolatile metabolites of theabrownins	Feng Zhang 等，2025	Food Chem 463:141317	三种工艺 TB 结构表征 + 高脂小鼠体内	560 mg/kg 调节糖脂；1120 mg/kg 增加降脂相关菌群
动物实验 Unlocking the healing power of Jinhua white tea (IBD)	Lin Y 等，2025	Food Res Int 213:116555	DSS 诱导 IBD 小鼠	JWT 经 NF-κB/MAPK 缓解结肠炎，恢复菌群与宿主代谢
动物实验 Dietary theabrownins improve freeze-	—，2026	Foods 15:462	草鱼饲料添加 0.02%/0.06% TB	改善冻融品质；文中提及 TB 生物利用度低

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
thaw quality of grass carp				

细胞实验（2 篇）

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
细胞实验 Theabrownin triggers DNA damage to suppress human osteosarcoma	Jin WD 等，2018	J Cell Mol Med	U2OS 细胞 + 斑马鱼移植瘤	TB 诱导 DNA 损伤激活 p53 通路诱导凋亡，IC50≈44 μg/mL（48 h）
细胞实验 Monascus-fermented Pu-erh tea and HepG2 lipid-lowering	Chen XM 等，2025	Foods	红曲霉发酵普洱 + HepG2 油红 O 模型	发酵使 TB 增加 29.82%，发酵茶在 HepG2 中降脂效果优于未发酵茶

综述（3 篇）

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
综述 Theabrownin: The rich hue of Chinese dark tea	Liu C 等，2025	Food Res Int	叙述性综述	汇总四条代谢轴（胆汁酸-FXR、SCFA-PPARγ/NF-κB、AhR/IL-22、AMPK-PGC1α）；普洱 TB 含量约 11%
综述 Therapeutic	Wang B 等，2024	Biomed Pharmacother	ASK1 药理综述	TB 作为天然 ASK1 激活剂诱导肺癌细胞凋亡的引文线索

文献标题/文件名	第一作者·年份	期刊/单位	研究模型	核心发现
potential of ASK1 activators				
综述 Tea consumption and diabetes: pharmacological perspectives	Erukainure OL 等，2025	Plants	五类茶抗糖尿病综述	归纳 TB 改善血糖、胰岛素敏感性、血脂并重塑菌群

其他-体外/化学表征、安全性及无关文件

以下 37 篇文献（28 篇体外/化学表征 + 4 篇安全性 + 5 篇无关）主要为结构、制备、检测、工艺、风味、安全性背景资料，未逐一展开，但已在第 1–2 节相关位置引用其关键数据。

类别	代表性文献/文件名	第一作者·年份	期刊/单位	与综述的相关要点
体外/表征	Structural Characterization and Gut Microbiota Modulation of Theabrownins	Liu H 等，2026	J Food Sci	上消化道不吸收、结肠产 SCFA、富集双歧杆菌
体外/表征	Detection of composition of tea by degradation method	Hu S 等，2025	Food Chem	TB 分子量一般 >3.5 kDa；两种制备法共有 86 种降解产物
体外/表征	不同制备工艺粗茶褐素的结构表征及组成分析	张凤等，2024	食品学报类	三种工艺 TB 均为无定形聚合物，XRD 显示结构差异
体外/表征	普洱速溶茶粉中茶褐素类物质的化学组成分析	杨柳，2025	食品科学	>300 kDa 组分占 36.83%；TB 为多糖-多酚复合体系
体外/表征	Efficient preparation and structural	—，2025	Food Chem	透析 vs 有机溶剂法提取比较

类别	代表性文献/文件名	第一作者·年份	期刊/单位	与综述的相关要点
	characterization of theabrownins			
体外/表征	Enzymatic oxidation preparation of theabrownins	—，2023	J Integr Agric	酶氧化制 TBs，对 E. coli MIC=1.25 mg/mL
体外/表征	Ganoderma-fermented green tea metabolomics	Liu X 等，2025	Foods	发酵使 TB 增加 10.21 倍；体外结合甘氨/牛磺胆盐
体外/表征	Storage metabolomics of dark tea	Li H 等，2025	Food Chem	TB 含量在储存第 5 年达峰；Pseudomonas/Aspergillus 驱动合成
体外/表征	Flavor/quality, fermentation, detection, processing 类文献（约 18 篇）	—	Food Chem / Foods 等	工艺优化、品质标志物、快速检测方法、风味形成
安全性	Acute and subacute safety of black tea extract	Kim S 等，2022	Toxics	HTE LD_{50}=21.68 g/kg；4000 mg/kg/d 轻度肾毒性
安全性	Acute and subchronic toxicity of Pu-erh extract	Wu SC 等，2011	J Ethnopharmacol	LD_{50}>10 g/kg、NOAEL=5000 mg/kg/d
安全性	Evaluation of toxicity of green tea catechins	Isbrucker RA 等，2008	Food Chem Toxicol	儿茶素 NOAEL≈764–820 mg/kg/d，作前体安全性对照
安全性	云南普洱茶特征成分的功能与毒理学评价	龚加顺，2007	茶叶科学	TB 复合物 LD_{50}>10 g/kg 实际无毒、无致突变
无关	Methylstat glioma / MAFLD mechanisms	—	—	主题不符或下载失败（Cloudflare 验证 HTML

类别	代表性文献/文件名	第一作者·年份	期刊/单位	与综述的相关要点
	/ PCBs renal injury / TB-whey coacervate / TB-potato starch complex			伪 PDF），已剔除

7. 说明与局限

- **文献覆盖**：本梳理基于原文件夹 63 份 PDF 及用户本次补充的 3 篇新文献，共 66 份 PDF；去重后 62 篇文献条目，其中 57 篇与茶褐素功效、代谢、结构或安全性相关，5 篇为无关/无效文件。
- **新增核心文献**：Huang 2019（*Nat Commun*）填补了茶褐素降胆固醇的完整机制链（菌群-胆汁酸-FXR-FGF15-CYP7B1-粪便排泄），应作为机制章节的核心引用； Jensen 2016 与 Yang 2014 提供了早期/北美人群普洱茶提取物的人体减重证据，但需注意其干预物并非纯化茶褐素。
- **“功效”≠“临床治疗”**：人体直接证据目前包括 Ding 2025（高尿酸）、Li 2026（血糖）、Huang 2019（胆固醇）； Yang 2014、Jensen 2016 为普洱茶提取物人体 RCT。动物与细胞证据丰富，但人群异质性、剂量换算、长期安全性仍需更多临床验证。
- **代谢途径缺口**：茶褐素上消化道不吸收、结肠菌群发酵产 SCFA 已有直接证据；Huang 2019 提供了“粪便胆汁酸排泄增加”的直接证据，但茶褐素原形及特征代谢物经尿液、胆汁、粪便的定量排泄研究仍不足，建议后续补充同位素示踪或血清/尿液/胆汁代谢组学研究。配套报告见《茶褐素排泄途径补充文献检索.html》。
- **图片位置**：报告图片均为从对应 PDF 渲染的页面快照，页码为 PDF 阅读器页码；图中 Figure/图号、图注与正文位置可直接用于引用核对。

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