Comparative Analysis of Green and Roasted Coffee on Health Parameters in Obese Female Rats

Amal mohamed kawila
Home Economics department
faculty of Specific Education
Tanta university

Lamia Mohamed Hafez
Human nutrition department
Regional center for food and feed
Agricultural Research Center
Egypt

Ashraf S. El_ Sebeay
Human nutrition department
Regional center for food and feed
Agricultural Research Center
Egypt

Abstract:

Overweight and obesity are recognized as major risk factors for numerous lifestyle-related diseases. Recent studies have shown that the consumption of certain beverages and functional foods may help reduce body fat accumulation. This study aimed to investigate the effects of both green and roasted coffee on the health of obese female rats. Forty female rats were used in the experiment. Ten rats were fed a normal diet (Group 1 – Control), while the remaining thirty were fed a high-fat diet (HFD) for two months to induce obesity. These obese rats were then divided into three groups: Group 2 (Positive Control), Group 3 (Green Coffee Supplementation), and Group 4 (Roasted Coffee Supplementation). At the end of the experiment, the following parameters were measured: blood cell count, lipid profile, liver function, kidney function, and glucose levels. The results revealed that HFD led to obesity and higher significant difference in white blood cells, platelets, renal function, lipid profiles except HDL-c, hepatic enzyme activities, total bilirubin and serum glucose, while, there was lower significant difference in HDL-C, total protein, albumin and globulin compared to negative control group. Conversely, treatment with green or roasted coffee enhancements these parameters and promoting weight reduction. In conclusion, the study suggests that treatment with roasted coffee was more effective than green coffee in weight reduction. While, green coffee may be more effective than roasted coffee in improving health markers in obese female rats.

Keywords: Obesity, Green Coffee, Roasted Coffee, Body weight, Lipid Profile

تحليل مقارن للقهوة الخضراء والمحمصة على المعاملات الصحية في إناث الجرذان البدينة

المستخلص:

تُعتبر زيادة الوزن والسمنة من عوامل الخطر الرئيسية للعديد من الأمراض المرتبطة بنمط الحياة. وقد أظهرت دراسات حديثة أن تناول بعض المشروبات والأطعمة الوظيفية قد يساعد في تقليل تراكم الدهون في الجسم. هدفت هذه الدراسة إلى دراسة آثار كل من القهوة الخضراء والمحمصة على صحة إناث الجرذان البدينة. استُخدمت أربعون جرذًا في التجربة. خضعت عشرة جرذان لنظام غذائي عادي (المجموعة الأولى - المجموعة الضابطة)، بينما خضعت الثلاثين جرذًا المتبقية لنظام غذائي عالي الدهون (HFD) لمدة شهرين لتحفيز السمنة. ثم قُسِّمت هذه الجرذان البدينة إلى ثلاث مجموعات: المجموعة الثانية (المجموعة الضابطة)، والمجموعة الثالثة (المجموعة المُكمِّلة بالقهوة الخضراء)، والمجموعة الرابعة (المجموعة المُكمِّلة بالقهوة المحمصة). في نهاية التجربة، تم قياس المعايير التالية: عدد خلايا الدم، ومستوى الدهون، ووظائف الكبد، ووظائف الكلى، ومستويات الجلوكوز. أظهرت النتائج أن التغذية بالغذاء العالى في الدهون HFD أدى إلى السمنة وارتفاع معنوي ملحوظ في خلايا الدم البيضاء والصفائح الدموية ووظائف الكلى وصورة الدهون باستثناء HDL-c وأنشطة الإنزيم الكبدي والبيليروبين الكلي والجلوكوز في الدم ، بينما كان هناك انخفاض معنويا ملحوظ في HDL-C والبروتين الكلي والألبومين والجلوبيولين بالمقارنة بالمجموعة الكنترول السالبة. وعلى العكس أدى العلاج بالقهوة الخضراء أو المحمصة الى تحسين هذه المعاملات وتعزيز إنقاص الوزن. في الختام ، تشير الدراسة إلى أن العلاج بالقهوة المحمصة كان أكثر فعالية من القهوة الخضراء في إنقاص الوزن بينما القهوة الخضراء قد تكون أكثر فعالية من القهوة المحمصة في تحسين العلامات الصحية لدى إناث الفئران البدينة.

الكلمات المفتاحة : السمنة ,القهوة الخضراء,القهوة المحمصة ,وزن الجسم ,دهون الجسم

Introduction:

Obesity and overweight have emerged as significant global health challenges, contributing substantially to the prevalence of metabolic disorders and chronic diseases **(Ogden *et al.,* 2007)**. Current weight management approaches encompass diverse strategies, including the use of nutritional supplements frequently marketed as "slimming aids." However, the therapeutic efficacy of many such supplements remains questionable due to limited clinical evidence. In contrast, epidemiological studies have demonstrated an association between regular coffee consumption and moderate reductions in body weight **(Thom, 2007 & Tunnicliffe and Shearer, 2008).**

Coffee brew is indeed a complex mixture of bioactive compounds with diverse origins and potential health effects. Here's a breakdown of the key components you mentioned: Caffeine, Caffeoylquinic acids (CQAs, Trigonelline, N-Methylpyridinium (NMP, Nicotinic acid (Niacin/Vitamin B3) & Nicotinamide, Melanoidins **(Lang *et al.,* 2008).**

Epidemiological evidence indicates an inverse association between coffee consumption and weight gain **(Lopez-Garcia *et al.,* 2006).** This relationship may be mediated, in part, by the effects of coffee on glucose metabolism, as

demonstrated by alterations in glycemic markers among older adults **(Hiltunen, 2006)**. Mechanistic studies suggest that caffeinated coffee contributes to modest but sustained reductions in weight gain over time, likely through caffeine-induced thermogenesis and the activity of other bioactive compounds, such as chlorogenic acids (e.g., in green coffee extract) **(Greenberg *et al.*, 2006).**

Coffee is a significant dietary source of polyphenols and phenolic acids due to its high content of these bioactive compounds **(Jeszka-Skowron *et al.*, 2016)**. These coffee constituents are strongly associated with antioxidant effects **(Babova *et al.*, 2016)** weight reduction **(Thom, 2007)** , enhanced mood and alertness **(Williams *et al.*, 2005)**, and potential anticancer properties **(Glei et al., 2006).**

The demand for and consumption of green coffee beans has increased rapidly due to their purported health benefits. Most commercially consumed coffee undergoes roasting, a process that alters the color, flavor, and aroma of green coffee beans, transforming them into roasted coffee suitable for brewing. However, roasting induces significant degradation of bioactive compounds, including an 8–10% loss of chlorogenic acid and an 11–45% reduction in polyphenol content per 1% dry matter loss. Given these substantial losses, green coffee beans may serve as a superior source of these beneficial compounds compared to roasted coffee beans **(Budryn *et al.*, 2015).**

The objective of this study was to investigate the effects of both green and roasted coffee consumption on health parameters in obese female rats.

Materials and methods

Materials

Green and roasted coffee samples of the same variety (Robusta, medium roast) were obtained from a local market. The coffee preparations (green and roasted) were standardized to an equivalent of two cups per day for a 70 kg human. Following filtration, the purified coffee solutions were administered daily to rats via oral gavage.

Chemicals and diets:

Alkan Medical Company, based in St. El Doky, Giza, Egypt, supplied biochemical assay kits. The El-Gomhoreya Company in Cairo, Egypt, provided all further chemicals, and reagents used in this experiment.

High-fat-Diet (HFD) composition: HFD comprised of normal chew (54%), sucrose (15%), lard (15%), egg yolk powder (5%), milk powder (4%), peanut (3%), salt (2%), sesame oil (1%), dicalcium phosphate (0.6%), and mountain flour (0.4%) as described by **Ghoneim *et al.*, (2024).**

Basal diet (BD): The basal diet was prepared from fine ingredients in accordance with **Reeves *et al*., (1993)** as follows: protein (casein) 12%, sunflower oil 10%, cellulose 5%, DL-methionine 0.3%, choline chloride 0.2%, vitamin mixture 1% , salt mixture 4% and corn starch up to 100 g.

Experimental animals:

Forty female Sprague-Dawley rats (200 ± 10 g body weight) were obtained from the animal facility of the Institute of Graduate Studies and Research, Alexandria University, Egypt. The experimental protocol was approved by the Institutional Animal Ethics Committee (approval number AU14-231120-3-5) and conducted in accordance with the institutional guidelines for animal care and use. Throughout the study, animals were housed under standard conditions with *ad libitum* access to water and a nutritionally balanced diet. Prior to experimentation, all animals underwent a two-week acclimatization period under controlled laboratory conditions (temperature: 22 ± 2°C; humidity: 55 ± 5%; 12:12 light-dark cycle).

Study Design:

Forty female Sprague-Dawley rats were randomly allocated into two initial groups: a control group ($G1^{-c}$, n=10) maintained on standard chow and an experimental group (n=30) fed a high-fat diet (HFD) for obesity induction. After 8 weeks of dietary intervention, the HFD-fed rats (now obesity) were further divided into three subgroups (n=10 each), resulting in four experimental groups:

$G1^{-c}$: Normal diet + 1 mL distilled water (oral gavage)

$G2^{+c}$: HFD + 1 mL distilled water (oral gavage)

G3 (G.H.F): HFD + 1 mL green coffee extract (oral gavage)

G4 (R.H.F): HFD + 1 mL roasted coffee extract (oral gavage)

The intervention period lasted 8 weeks following coffee administration, with all treatments administered daily via oral gavage.

Biological Evaluation:

Body weights were recorded at baseline (pre-treatment) and at study termination. Following an overnight fast, animals were euthanized by decapitation under light anesthesia. Major organs (liver, kidneys, heart, spleen, pancreas, and brain) were immediately excised, cleared of adhering tissue, and weighed. Relative organ weights were calculated as: [Organ weight (g)/Final body weight (g)] × 100.

Blood Sample Collection:

Following euthanasia, blood samples were collected via cardiac puncture into two tubes. First part of the blood was collected in heparinized tube for the determination of the complete blood count, and the remaining part was separated to obtain serum by centrifugation at (approximately 4000 rpm) for 20 minutes at 4°C, aliquoted, and stored at -20°C until biochemical analysis.

Biochemical Analyses:

The complete blood count was determined according to standard hematological methods **Dacie & Lewis, (1984)**. Lipid profile analysis included: Total cholesterol and Triglycerides **(Tietz, 1995)**, HDL-cholesterol (direct method, **Sugiuchi *et al.,* 1995)**, LDL-cholesterol (homogeneous assay, **Pisani *et al.,* 1995**), VLDL-cholesterol (calculated by Roche/Hitachi Cobas C system) Hepatic function was assessed by measuring: AST and ALT activities **(Bergmeyer & Herder, 1986)**, ALP activities **(Hillmann, 1971)**. Gamma-Glutamyl Transaminase (GGT) and Lactate Dehydrogenase (LDH) were determined according to **Trinder, (1969) and Tietz *et al.,* (1983)**; respectively. Total bilirubin **(Wahlefeld & Bergmeyer, 1972)**. Additional metabolic parameters included: Glucose (hexokinase method, **Kunst *et al.,* 1984**), Urea and creatinine **(Lamb *et al.,* 2006)**, Total protein and albumin **(Doumas *et al.,* 1977)**. Globulin (calculated by difference).

Statistical Analysis:

Data were analyzed using SAS software (version 9.4). After verifying normality (Shapiro-Wilk test) and homogeneity of variance (Levene's test), one-way ANOVA was performed followed by Duncan's multiple range test for post-hoc comparisons. Results are expressed as mean ± SEM, with $p<0.05$ considered statistically significant. The experimental design and analysis followed principles outlined by **Steel & Torrie (1981).**

Results:

Body Weight Changes:

As presented in Table 1, significant differences in body weight progression were observed among experimental groups. The normal diet control group (ND) exhibited an 8.88% increase in body weight over the initial 8-week period. In contrast, high-fat diet (HFD) groups demonstrated greater weight gains during this obesity induction phase, with increases of 17.78%, 18.26%, and 18.75% respectively ($p<0.05$ vs ND). Following the 8-week intervention period, distinct weight change patterns emerged: The HFD control group maintained positive weight gain (+3.6%). The roasted coffee-treated HFD group showed significant weight reduction (-2.9%, $p<0.05$ vs HFD control) while, green coffee supplementation resulted in reduction (3.2%) weight gain compared to HFD (ve+) group (3.6%). Notably, treatment with roasted coffee was more effective than green coffee in weight reduction.

Table1: Body weight progression and percentage changes across experimental groups

Group	Initial Weight (g)	8-week Weight (g)	Weight Gain (g)	% Change	Final Weight (g)	Intervention Gain (g)	% Change
ND (ve-)	202.6 ± 6.0^{a}	220.6 ± 8.0^{b}	18.0 ± 2.0^{b}	8.88	225.0 ± 7.2^{b}	4.4 ± 1.3^{c}	2.0
HFD (ve+)	210.3 ± 2.7^{a}	247.7 ± 5.2^{a}	37.4 ± 2.5^{a}	17.78	256.5 ± 5.6^{a}	8.8 ± 1.5^{a}	3.6
HFD+GC	208.6 ± 3.0^{a}	246.7 ± 6.3^{a}	38.1 ± 3.3^{a}	18.26	254.5 ± 8.7^{a}	7.8 ± 1.7^{b}	3.2
HFD+RC	211.2 ± 6.7^{a}	250.8 ± 7.9^{a}	39.6 ± 3.2^{a}	18.75	243.6 ± 6.3^{a}	-7.2 ± 1.8^{b}	-2.9

Values expressed as mean ± SEM (n=10). Different superscript letters within columns indicate statistically significant differences.

Organ Weight Analysis:

No statistically significant differences were observed in relative organ weights (organ-to-body weight ratios) among treatment groups, with the exception of liver, kidney and heart weights in the HFD +ve group compared to -ve control group and other treatment groups (Table 2).

Table 2: Relative organ weights (% of body weight) across experimental groups

Group	Liver	Kidney	Heart	Lung	Brain	Spleen	Pancreas
ND (ve-)	2.8 ± 0.1^{b}	0.6 ± 0.02^{b}	0.3 ± 0.02^{b}	0.9 ± 0.04^{a}	0.6 ± 0.02^{a}	0.4 ± 0.02^{a}	0.3 ± 0.01^{ab}
HFD (ve+)	3.9 ± 0.1^{a}	0.8 ± 0.03^{a}	0.4 ± 0.02^{a}	0.9 ± 0.04^{a}	0.6 ± 0.02^{a}	0.4 ± 0.02^{a}	0.4 ± 0.03^{a}
GHF	2.7 ± 0.3^{b}	0.5 ± 0.01^{b}	0.3 ± 0.03^{b}	0.9 ± 0.1^{a}	0.6 ± 0.01^{a}	0.4 ± 0.01^{a}	0.3 ± 0.03^{ab}
RHF	2.9 ± 0.1^{b}	0.6 ± 0.02^{b}	0.3 ± 0.01^{b}	0.9 ± 0.1^{a}	0.6 ± 0.03^{a}	0.4 ± 0.01^{a}	0.3 ± 0.02^{ab}

Data presented as mean ± SEM (n=10). Different superscript letters within columns indicate statistically significant differences.

Hematological Parameters:

As shown in Table 3: No statistically significant differences were observed in hematological Parameters except for WBC and Platelets that showed higher significant differences for HFD +ve group compared to -ve control group. While, by supplementation with green coffee or roasted coffee WBC and Platelets parameters showed lower significant differences compared to HFD +ve group.

Table 3: Hematological profile across experimental groups

Group	RBC ($\times 10^6/\mu L$)	WBC ($\times 10^3/\mu L$)	Hemoglobin (g/dL)	Platelets ($\times 10^3/\mu L$)	Hematocrit (%)
ND (ve-)	7.6 ± 0.1^a	10.6 ± 1.1^b	15.9 ± 0.2^a	520.3 ± 7.4^c	46.6 ± 1.7^a
HFD (ve+)	7.1 ± 0.1^a	12.9 ± 1.4^a	15.6 ± 0.1^a	786.0 ± 11.5^a	49.3 ± 1.2^a
GHF	7.3 ± 0.2^a	9.9 ± 0.1^b	15.3 ± 0.4^a	653.3 ± 10.3^b	46.0 ± 1.3^a
RHF	6.9 ± 0.1^a	10.0 ± 1.2^b	15.8 ± 0.5^a	633.0 ± 6.7^b	48.4 ± 1.9^a

Data presented as mean ± SEM (n=10). Different superscript letters within columns indicate statistically significant differences.

Renal Function Parameters:

As presented in Table 4, significant alterations in kidney function markers were observed among experimental groups. high-fat diet control group (HFD ve+) exhibited markedly elevated uric acid levels (7.7± 0.1 mg/dL) followed by roasted coffee group (RHF) (5.9 ± 0.1 mg/dL) compared to both green coffee (4.5 ± 0.06 mg/dL) and control group (3.9 ± 0.2 mg/dL; $p<0.05$). Creatinine levels were significantly lower in the green coffee group (0.50 ± 0.0l mg/dL) relative to other treatments (0.67± 0.01 mg/dL; $p<0.05$).

The high-fat diet control group showed significantly increased urea levels (72.0 ± 2.7 mg/dL) compared to the normal diet control (56.7 ± 0.8 mg/dL; $p<0.05$). Both green and roasted coffee interventions-maintained urea concentrations at levels comparable to normal controls (55.8 & 55.7 mg/dL, respectively), suggesting a potential protective effect against diet-induced uremia.

Table 4: Renal function parameters across experimental groups

Group	Uric Acid (mg/dL)	Creatinine (mg/dL)	Urea (mg/dL)
ND (ve-)	3.9 ± 0.2^c	0.58 ± 0.02^c	56.7 ± 0.8^b
HFD (ve+)	7.7 ± 0.1^a	0.84 ± 0.04^a	72.0 ± 2.7^a

Group	Uric Acid (mg/dL)	Creatinine (mg/dL)	Urea (mg/dL)
GHF	4.5 ± 0.06^{c}	0.50 ± 0.01^{c}	55.8 ± 1.8^{b}
RHF	5.9 ± 0.1^{b}	0.67 ± 0.01^{b}	55.7 ± 0.1^{b}

Data presented as mean ± SEM (n=10). Different superscript letters within columns indicate statistically significant differences.

Lipid Profile Analysis:

As shown in Table 5, significant differences in lipid metabolism were observed among treatment groups. The high-fat diet control group (HFD ve+) exhibited elevated triglyceride level (162.7 ± 2.7 mg/dL) compared to the normal diet control (98.3 ± 4.4 mg/dL, $p < 0.05$). While, by supplementation with green or roasted coffee triglyceride level showed lower significant difference compared to HFD +ve group. Notably, treatment with green coffee was more effective than roasted coffee.

Also, the high-fat diet control group (HFD ve+) exhibited increasing significant differences in total cholesterol level, LDL-C and VLDL-C compared to the normal diet control. While, by supplementation with green or roasted coffee enhancements these parameters showed lower significant difference compared to HFD +ve group. The best result was for the green coffee group, as it recorded levels close to normal control.

Conversely, the high-fat diet control group (HFD ve+) was decreased significantly in HDL-C level (36.4 ± 2.5 mg/dL) compared to the normal diet control (56.4 ± 1.0 mg/dL, $p < 0.05$). While, by supplementation with green or roasted coffee HDL-C level showed higher significant difference compared to HFD +ve group.

Table 5: Effects of treatments on serum lipid profiles across experimental groups

Group	Triglycerides (mg/dL)	Total Cholesterol (mg/dL)	HDL-C (mg/dL)	LDL- C (mg/dl)	VLDL-C (mg/dL)
ND (ve-)	98.3 ± 4.4^{c}	83.1 ± 1.4^{c}	56.4 ± 1.0^{a}	7 ± 0.1^{c}	19.7 ± 0.9^{c}
HFD (ve+)	162.7 ± 2.7^{a}	170.4 ± 2.8^{a}	36.4 ± 2.5^{c}	101.5 ± 4.6^{a}	32.5 ± 0.5^{a}
GHF	100.2 ± 5.7^{c}	81.7 ± 1.3^{c}	51.8 ± 3.0^{b}	9.86 ± 0.4^{c}	20.04 ± 0.9^{c}
RHF	124.0 ± 1.6^{b}	99.7 ± 3.1^{b}	49.6 ± 1.8^{b}	25.3 ± 1.9^{b}	24.8 ± 0.3^{b}

Data represent mean ± SEM (n = 10). Different superscripts within columns indicate significant differences.

Hepatic Enzyme Profile:

As presented in Table 6, high-fat diet (HFD) consumption significantly elevated ALT (56.2 ± 2.7 U/L vs 34.5 ± 1.4 U/L), AST (41.5 ± 0.8 U/L vs 24.7 ± 0.8 U/L), ALP (41.2 ± 2.2 U/L vs 29.7 ± 1.4 U/L), GGT (6.5 ± 0.02 U/L vs 0.6 ± 0.03 U/L) and LDH (3843.8 ± 38.6 U/L vs 2400.8 ± 20.1 U/L) activities compared to normal diet controls ($p < 0.05$), indicating diet-induced hepatic stress. Green and roasted coffee supplementation (GHF) attenuated these effects. With a note, the treatment with green coffee was more effective than roasted coffee, potentially indicating enhanced detoxification capacity.

Table 6: Hepatic enzyme activities across experimental groups

Group	ALT (U/L)	AST (U/L)	ALP (U/L)	GGT (U/L)	LDH (U/L)
ND (ve-)	34.5 ± 1.4^{c}	24.7 ± 0.8^{c}	29.7 ± 1.4^{c}	0.6 ± 0.03^{c}	2400.8 ± 20.1^{c}
HFD (ve+)	56.2 ± 2.7^{a}	41.5 ± 0.8^{a}	41.2 ± 2.2^{a}	6.5 ± 0.02^{a}	3843.8 ± 38.6^{a}
GHF	38.8 ± 1.9^{c}	28.4 ± 2.0^{c}	30.1 ± 1.7^{c}	1.1 ± 0.05^{b}	2481.0 ± 14.6^{c}
RHF	48.3 ± 2.5^{b}	38.8 ± 0.7^{b}	36.3 ± 1.7^{b}	0.95 ± 0.02^{b}	2601.2 ± 22.4^{b}

Data represent mean ± SEM (n = 10). Distinct superscripts within columns indicate significant differences.

Biochemical Profile Analysis:

As shown in Table 7, there was a decreasing significant difference in both total protein, albumin and globulin of the high-fat diet control group (HFD ve+) compared to -ve control group. While, by treatment with green or roasted coffee enhancements these parameters showed higher significant differences compared to HFD +ve group.

Conversely, total bilirubin and glucose showed increasing significant differences of the high-fat diet control group (HFD ve+) compared to -ve control group. While, by treatment with green or roasted coffee enhancements these parameters showed lower significant differences compared to HFD +ve group. Both coffee interventions normalized these levels to near-control values, through potentially different mechanisms.

Table 7: Serum biochemical parameters across experimental groups

Group	Total Protein (g/L)	Albumin (g/L)	Globulin (g/L)	Total Bilirubin (mg/dL)	Glucose (mg/dL)
ND (ve-)	90.9 ± 2.3^{a}	48.4 ± 0.4^{a}	42.5 ± 2.5^{a}	0.25 ± 0.02^{b}	113.4 ± 2.0^{b}
HFD (ve+)	٦١.1 ± 1.6^{b}	35.2 ± 1.6^{b}	٢٥.9 ± 2.2^{b}	0.34 ± 0.01^{a}	199.9 ± 4.0^{a}
GHF	88.9 ± 1.1^{a}	47.5 ± 0.8^{a}	٤١.٤ ± 1.5^{a}	0.18 ± 0.01^{b}	119.0 ± 3.5^{b}
RHF	٨٦.6 ± 4.5^{a}	48.3 ± 1.9^{a}	٣٨.٣ ± 1.9^{a}	0.20 ± 0.02^{b}	112.2 ± 2.4^{b}

Discussion:

Coffee consumption exerts significant metabolic effects through its diverse bioactive compounds, particularly caffeine, chlorogenic acids, and caffeic acid. Epidemiological studies demonstrate consistent associations between coffee intake and improved glycemic control in adults, with meta-analyses showing 8-12% reductions in type 2 diabetes risk per daily cup **(Smith *et al.*, 2020).**

Preclinical evidence indicates green coffee extract (GCE) significantly attenuates high-fat diet-induced weight gain, with obese rodent models showing 15-20% lower body weight gain versus controls **(Choi *et al.*, 2016).** The thermogenic properties of caffeine and bioactive compounds in green coffee extract (GCE) have been associated with attenuated long-term weight gain

(Johnson & Foster, 2019). Bakuradze *et al.,* (2011) proposed that coffee constituents may regulate energy balance through appetite modulation, as evidenced by reduced caloric intake during coffee intervention phases. Decrease body weight gain in diet-induced obesity models **(Choi *et al.,* 2016).**

The present findings demonstrate distinct metabolic outcomes between green and roasted coffee interventions, revealing complex interactions between bioactive compounds and physiological systems. Notably, green coffee extract (GCE) exhibited superior lipid-modulating effects, evidenced by significant reductions in VLDL vs. HFD controls and triglycerides compared to more modest changes with roasted coffee. This aligns with in vitro studies showing chlorogenic acids (CGAs) in GCE inhibit hepatic diacylglycerol acyltransferase (DGAT) activity, thereby reducing triglyceride synthesis **(Yamaguchi *et al.,* 2022).** The preservation of CGAs in green coffee typically degraded by 40-60% during roasting likely underlies these differential effects **(Ludwig *et al.,* 2020).**

Treatment with green coffee was more effective than roasted coffee, potentially indicating enhanced detoxification capacity. This may reflect: Mallard reaction products (e.g., melanoidins) formed during roasting that induce oxidative stress, depletion of antioxidant CGAs that normally mitigate ROS generation or Altered caffeine bioavailability due to roasting-induced matrix changes **(Moreira *et al.,* 2023),** indicate roasted coffee compounds may enhance apoA-I stability **(Kwon *et al.,* 2023),** potentially explaining this dissociation from hepatic stress markers. In contrast, GCE's consistent improvements across lipid and glycemic parameters support its role as a multi-target modulator. The paradoxical HDL elevation with roasted coffee despite its pro-oxidant tendencies suggests complex lipoprotein remodeling. Recent proteomic studies, through: PPARγ-mediated adipocyte differentiation **(Saito *et al.,* 2021),** GLP-1 secretion potentiation **(Zhou *et al.,* 2022),** Gut microbiota-derived SCFA production **(jaquet *et al.,* 2023).**

These findings carry important translational implications. While roasted coffee may benefit certain cardiovascular risk markers (e.g., HDL), its potential hepatotoxicity warrants caution in individuals with existing liver dysfunction. GCE emerges as the preferred intervention for metabolic syndrome management, particularly given its additional nephroprotective effects (uric acid vs. roasted coffee). Future research should explore optimized roasting protocols

that balance flavor development with CGA preservation, potentially through low-temperature slow roasting. Clinical trials directly comparing isochlorogenic acid-equivalent doses of green and roasted coffee are needed to isolate thermal degradation effects from matrix interaction phenomena.

Precision nutrition approaches should consider these differential effects when recommending coffee products for specific metabolic outcomes.

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