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Coffee and lipid profile: from theory to everyday practice

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Abstract

Lipid disorders have been the most common cause of atherosclerotic cardiovascular diseases in Poland for years. The latest data indicate that about 20 million people in Poland have hypercholesterolaemia. Nutritional habits have a very significant impact on the lipid profile. Therefore, considering the fact that coffee is an important component of the diet of Poles (on average, 1–2 cups of coffee are consumed in our country per inhabitant per day, and 66% of Poles declare regular consumption), its impact on the lipid profile cannot be overlooked. Coffee contains over 1000 chemical compounds, of which kahweol and cafestol are the most important in the context of lipidology. These are compounds that can have a hyperlipidaemic effect. On the other hand, compounds such as caffeine, chlorogenic acid, trigonelline, and melanoidins are characterized by antioxidant activity, which can limit lipid peroxidation. The effect of consuming coffee prepared in different ways has been analysed in numerous clinical studies. This article summarizes the current knowledge on the effects of coffee on the lipid profile and risk of atherosclerosis.

Key words: coffee, lipid disorders, atherosclerotic cardiovascular diseases

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Introduction

For years, lipid disorders have been ranked as the most common atherosclerotic cardiovascular disease (ASCVD) risk factor worldwide [1].

The most common lipid disorder in Poland is hypercholesterolemia, involving elevated levels of LDL-C (low-density lipoprotein cholesterol) fraction above the recommended values for a given cardiovascular risk group [2]. The WOBASZ II study (Multi-centre National Population Health Examination Survey), which included 5947 subjects aged 20–99, revealed that hypercholesterolemia was present in 67.1% of them (64.3% of women and 70.3% of men, respectively) [3]. These results indicate that the number of patients with hypercholesterolemia in Poland may be as high as 20 million.

Dietary habits have a very important effect on lipid profile [4]. Therefore, given that coffee is an important component of the Polish diet (on average, 1–2 cups of coffee per capita/day are consumed in our country and 66% of Poles declare regular consumption of coffee), its effect on lipid profile cannot be ignored.

Coffee and lipid profile

A systematic review and meta-analysis of randomised clinical trials by Schoeneck and Iggman concluded the effects of different dietary components on LDL-C levels. In the case of coffee, its effect on serum LDL-C levels was found to be dependent on whether filter or non-filter coffee was consumed. Consumption of filter coffee *versus* non-consumption of this beverage was not significantly associated with

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changes in serum LDL-C levels (MD = 0.03 mmol/L; 95% CI: –0.05 to 0.11). There was no significant difference between coffee and tea consumption in relation to LDL-C (MD = 0.14 mmol/L; 95% CI: –0.01 to 0.28). The comparison of filter coffee consumption and non-filter coffee consumption revealed that the latter increased serum LDL-C levels (MD = –0.39 mmol/L; 95% CI: –0.49 to –0.30). There was no significant effect on LDL-C of black coffee *versus* decaffeinated coffee (MD = –0.02 mmol/L; 95% CI: –0.08 to 0.04), nor on consumption of more roasted *versus* less roasted coffee (MD = 0.07 mmol/L; 95% CI: –0.08 to 0.23). Based on the results of this meta-analysis, the effect of coffee consumption on LDL-C depends on whether the brew is filter or non-filter [5].

A study by Miranda et al. [6], involving 4736 Brazilians, assessed the effect of the intensity of coffee consumption (≤ 1, 1–3 and >3 cups/day; cup = 50 mL espresso) on lipid profile. After including other risk factors, it was found that consumption of up to 3 cups of coffee a day did not alter the lipid profile (total cholesterol [β = 2.67; 95% CI: –0.10 to 5.41], triglycerides [β = 5.61; 95% CI: –0.71 to 11.93], LDL-C [β = 2.13; 95% CI: –0.13 to 4.40], HDL-C [β = 0.11; 95% CI: –0.89 to 1.11] and triglyceride-rich lipoproteins [β = 5.26; 95% CI: –0.46 to 10.97]). Consumption of >3 cups of coffee a day was associated with some lipoprotein-increasing effects (total cholesterol [β = 4.13; 95% CI: 0.81 to 7.45], triglycerides [β = 9.53; 95% CI: 1.65–17.42], LDL-C [β = 2.39; 95% CI: –0.37 to 5.14], HDL-C [β = 0.44; 95% CI: –0.75 to 1.64] and triglyceride-rich lipoproteins [β = 8.42; 95% CI: 1.24–15.60]). The results of this study indicate that the consumption of 1–3 cups of coffee a day does not affect the lipid profile [6].

A randomised clinical trial by Gonçalinho et al. [7], involving 53 healthy subjects analysed the effect of consuming 450–600 mL/day of filter Arabica or a filter blend of Arabica and Robusta on sirtuin-1, homocysteine and lipid levels. It should be borne in mind that Arabica is considered the oldest and the most high-quality type of coffee, originating in Ethiopia. It is cultivated in mountainous areas, at approximately 20–25 degrees Celsius, contains less caffeine than Robusta, while it may contain more fats and sugars than Robusta (fats in Arabica beans are approximately 6–9% while for Robusta they are approximately 3–7%, and sugars are 15–17% of Arabica beans compared to 10–11.5% for Robusta). Robusta, or Congolese coffee, originates from Central Africa but is also grown in other areas of the world (mainly in the intertropical zone). After 8 weeks of intervention, it was revealed that consumption of Arabica or a blend of Arabica and Robusta significantly increased sirtuin-1 levels (0.51 to 0.58 ng/mL; p = 0.004, and from 0.40 to 0.49 ng/mL; p = 0.003) but had no effect on homocysteine levels. In terms of the lipid profile, it was found that consumption of a blend of Arabica and Robusta was associated with increases in total cholesterol (from 4.70 to 5.17 mmol/L; p < 0.001), LDL-C (from 2.98 to 3.32 mmol/L; p < 0.001) and HDL-C (from 1.26 to 1.36 mmol/L; p < 0.001). In this study, coffee consumption did not affect triglyceride levels. The observed differences in terms of the effects of pure Arabica and its blend with Robusta are probably due to the different polyphenol content (more caffeine, less polyphenols in Robusta). The results of this study indicate that, with a view to the lipid profile, filter Arabica coffee should be preferred [7]. A study by Gebeyehu et al. [8], involving 70 healthy subjects, assessed the effect of consuming 100% filter Ethiopian Arabica on lipid profile. Consumption of filter Arabica was found to be associated with a reduction in triglyceride levels (p < 0.01), while having a non-significant effect on total cholesterol and LDL-C levels [8]. The results of a study by Svatun et al. [9], involving 21 083 subjects from the Tromsø Study in Northern Norway, cannot be overlooked. This study analysed the effect of coffee consumption on serum total cholesterol levels. It was found that consumption of 1–2 cups of espresso or filter coffee a day did not have a statistically significant effect on serum total cholesterol levels (in contrast to consumption of 3–5 cups of these brews a day). Consumption of 1–2 cups of boiled coffee/day revealed how significant the effect on an increase in serum total cholesterol levels was. Instant coffee at 1–2 cups/day significantly increased serum total cholesterol levels in men; this effect was not observed in women [9]. The results of this study indicate that there should be a preference to consume espresso or filter coffee at 1–2 cups/day, bearing in mind the lipid profile.

Zhou and Hyppönen's study of 36 2571 subjects from the UK Biobank database assessed the effect of regular coffee consumption on lipid profile. There was a dose-dependent slight increase in LDL-C (1–2 cups of coffee/day: β = 0.06 mmol/L; 95% CI: 0.05–0.07; > 6 cups of coffee/day: β = 0.13 mmol/L; 95% CI: 0.11–0.15) and this effect did not differ significantly by the type of coffee consumed: ground, decaffeinated or instant. HDL-C also revealed increased levels (1–2 cups of coffee/day: β = 0.01 mmol/L; 95% CI: 0.01–0.01; > 6 cups of coffee/day: β = 0.01 mmol/L; 95% CI: 0.01–0.02), however, this effect only applied to ground and instant coffee. Coffee consumption was also dose-dependently associated with increased total cholesterol levels (1–2 cups of coffee/day: β = 0.08 mmol/L; 95% CI: 0.07–0.09; > 6 cups of coffee/day: β = 0.15 mmol/L; 95% CI: 0.13–0.18), regardless of the type of brew consumed. In terms of triglycerides, the information is more optimistic since consumption of coffee did not affect or could even gently reduce their levels (1–2 cups of coffee/day: β = 0.01 mmol/L; 95% CI: 0.00–0.02; > 6 cups of coffee/day: β = –0.07 mmol/l; 95% CI: –0.09 to –0.05), and this effect was common across all analysed types of coffee. The effect of coffee consumption on apolipoprotein B (apoB) and apolipoprotein A1 (apoA1) levels was also analysed. There was a dose-dependent increase in apoB levels

(1–2 cups of coffee/day: β = 0.01 g/L; 95% CI: 0.01–0.01; > 6 cups of coffee/day: β = 0.02 g/l; 95% CI: 0.02–0.03), which was independent of coffee type. In terms of apoA1, coffee consumption may or may not have increased its levels (1–2 cups of coffee/day: β = 0.01 g/L; 95% CI: 0.00––0.01; > 6 cups of coffee/day: β = 0.00 g/L; 95% CI: –0.01 to 0.01), with the most beneficial effect observed when ground coffee was consumed, followed by instant coffee. Another study found a positive association between coffee consumption and LDL-C/total cholesterol/ /apoB. The results of this prospective study indicate that coffee consumption may be associated with increased LDL-C, total cholesterol and apoB levels. **Nevertheless, it should be emphasised that in terms of Polish conditions, where 1–3 cups of coffee/day are consumed on average, an increase in LDL-C of approximately 2 mg/dL and total cholesterol of 3 mg/dL can be expected, which is not clinically significant.** Furthermore, as the authors of the study point out, an important limiting factor is that the respondents reported coffee consumption in a questionnaire. It is also impossible to assess exactly what the kahweol and cafestol content of coffees consumed by the subjects was. The importance of this issue is indicated, for example, by the fact that consumption of instant coffee increased LDL-C and total cholesterol levels to a lesser extent compared to ground coffee, while this is the coffee that contains less kahweol and cafestol [10].

A meta-analysis of 12 randomised clinical trials by Du et al. [11], involving 1182 subjects, concluded the effects of coffee consumption on the risk of dyslipidemia. The results of this meta-analysis are shown in Table 1.

The results of this meta-analysis indicate that consumption of higher amounts of coffee may be associated with increased levels of specific lipid fractions. A dose-effect analysis revealed that consumption of 1–3 cups of coffee/ /day (preferably filter coffee) had no effect on LDL-C, HDL-C and triglyceride levels, whereas it had a borderline effect on total cholesterol levels [11]. The results obtained in this meta-analysis are in line with those obtained several years ago in a meta-analysis of 12 randomised clinical trials by Cai et al. [12]. It was found that consumption of filter coffee had a slight effect on total cholesterol levels (difference: 3.6 mg/dL; 95% CI: 0.6–6.6), while it had no significant effect on LDL-C and triglycerides. Moreover, a dose-dependent effect was also found. Consumption of up to 6 cups of coffee/day had little effect on total cholesterol levels (difference: 4.2 mg/dL; 95% CI: 1.3–7.1) and no effect on LDL-C and triglycerides [12]. Moreover, these results are fully in line with the results of the meta-analysis review of the effects of coffee on human health by Poole et al. It was found that consumption of non-filter coffee significantly increased total cholesterol, LDL-C and triglyceride levels, while consumption of filter brew increased total cholesterol levels only slightly. Decaffeinated coffee consumption was not associated with changes in lipid profile [13]. The results of this meta-analysis indicate that the consumption of 1–3 cups of coffee/day, preferably filter, remains safe from the point of view of the risk of lipid disorders.

A systematic review by Penson et al. [14], involving 640 subjects, analysed the effect of coffee consumption on lipoprotein (a) levels. It was found that consumption of filter coffee might be associated with a reduction in lipoprotein (a) levels, while non-filter coffee had the opposite effect. The authors indicate that the effect of coffee consumption on lipoprotein (a) depends on how coffee is prepared [14].

In terms of lipid profile, the less investigated green coffee should be mentioned. A meta-analysis of 17 randomised clinical trials by Ding et al. [15], revealed that decaffeinated coffee consumption was associated with a reduction in total cholesterol levels (weighted mean difference [WMD] = –4.51 mg/dL; 95% CI: –6.90 to –2.13), increased HDL-C levels (WMD = 2.64 mg/dL; 95% CI: 2.21 to 3.07), decreased LDL-C levels (WMD = –4.38 mg/dL; 95% CI: –6.45 to –2.32), and a non-significant effect on triglyceride levels (WMD = –4. 34 mg/dL; 95% CI: –9.00 to 0.32) [15]. The results of this meta-analysis indicate that green coffee consumption has some hypolipemic effects.

In summary, the results of recent studies and meta-analyses indicate that the consumption of 1–3 cups of coffee/ /day, preferably filter coffee, has no effect on lipid profile.

From coffee through lipid profile to atherosclerosis

The strong interest in the effect of coffee consumption on lipid disorders is also associated with previous observations showing that consumption of this beverage was associated with a higher risk of coronary artery disease (CAD). A very interesting paper by Shirai et al. [16] reviewed studies evaluating the effect of coffee consumption on CAD risk, published from 1990 to 2018. The analysis covered more than one million subjects from 147 countries. Interestingly, it was found that the assessment of the association between coffee consumption and the risk of CAD and death in CAD over the period 1990–2018 changed from unfavourable to favourable [16]. There are several explanations for this. A multi-centre study by Tverdal et al. [17], involving more than 500 000 subjects who were observed for 20 years, found that cardiovascular disease (CVD) mortality was higher in those who consumed non-filter coffee than those who consumed filter coffee. Furthermore, a similar relationship was found in terms of CAD risk [17]. The explanation for these differences lies in the way the coffee is prepared, as filtering the brew leads to a reduction in kahweol and cafestol, i.e., diterpenoids with hyperlipidemic effects [18]. There was a significant difference in terms of the effect of

Table 1. Effect of coffee consumption on lipid profile — results of a meta-analysis by Du et al. 2020 [11]

Lipid fraction [mmol/L]	Study group/subgroup	Number of RCT	Effect (WMD [95% CI]); mmol/L
Total cholesterol	Overall effect	12	0.21 (0.04–0.39)
	Filter coffee	3	0.10 (0.17–0.37)
	Boiled coffee	3	0.30 (0.06–0.53)
	Instant coffee	2	0.08 (0.06–0.21)
	1–3 cups/day	3	0.11 (0.03–0.23)
	3–5 cups/day	5	0.14 (0.03–0.31)
	≥ 6 cups/day	4	0.52 (0.40–0.54)
	≤ 6 weeks	5	0.24 (0.06–0.41)
	> 6 weeks	7	0.20 (0.04–0.36)
LDL-C	Overall effect	10	0.14 (0.05–0.24)
	Filter coffee	2	0.12 (0.24–0.47)
	Boiled coffee	3	0.14 (0.08–0.46)
	1–3 cups/day	2	0.10 (–0.17 to 0.36)
	3–5 cups/day	5	0.12 (0.06–0.30)
	≥ 6 cups/day	4	0.43 (0.19–0.67)
	≤ 6 weeks	3	0.13 (0.00–0.25)
	> 6 weeks	7	0.18 (0.03–0.33)
HDL-C	Overall effect	10	–0.01 (–0.06 to –0.04)
	Filter coffee	2	–0.02 (–0.12 to 0.09)
	Boiled coffee	2	–0.05 (–0.15 to 0.05)
	1–3 cups/day	2	–0.01 (–0.12 to 0.11)
	3–5 cups/day	5	–0.02 (–0.06 to 0.02)
	≥ 6 cups/day	3	0.00 (–0.07 to 0.07)
	≤ 6 weeks	4	–0.01 (–0.04 to 0.02)
	> 6 weeks	6	–0.04 (–0.10 to 0.03)
Triglycerides	Overall effect	7	0.12 (0.03–0.20)
	Boiled coffee	2	0.25 (0.08–0.41)
	Decaffeinated coffee	2	0.00 (–0.09 to 0.09)
	1–3 cups/day	2	0.04 (–0.26 to 0.50)
	3–5 cups/day	3	0.12 (0.01–0.24)
	≥ 6 cups/day	2	0.25 (0.16–0.34)
	≤ 6 weeks	3	0.08 (0.02–0.18)
	> 6 weeks	4	0.15 (0.02–0.33)

CI — confidence interval; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol; RCT — randomized controlled trial; WMD — weighted mean difference

filter and non-filter coffee on lipid profile in the previously discussed meta-analysis by Du et al. (Table 1) [11], as well as the meta-analysis by Cai et al. [12]. The evidence in the 1990s of the effect of coffee filtering in reducing adverse changes in lipid profile led to the spread of this method of preparing this brew. Currently, filtering coffee using a paper filter is common in many parts of the world, especially in high-income countries [16]. In recent years, some coffee drinkers may have switched from non-filter (Turkish) coffee to filter coffee [16].

In terms of atherosclerosis, it is useful to examine the effect of coffee consumption on the risk of peripheral artery disease (PAD), which is a very good model for research into this process. A study by Hoek et al. [19] assessed the relationship between different dietary components and the risk of PAD. Participants of the Million-Veteran-Program

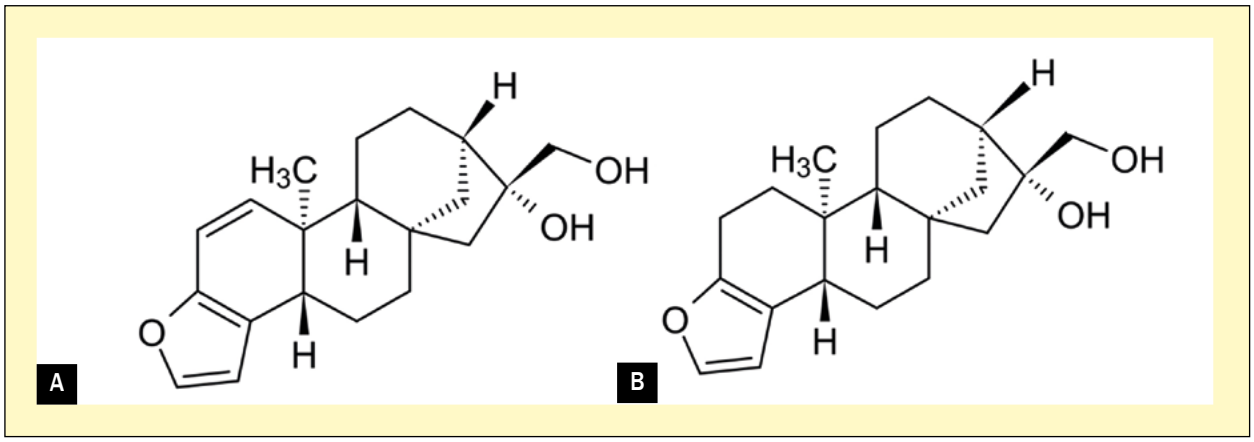

Figure 1. Chemical structure of diterpenoids: **A.** kahweol and **B.** cafestol

(MVP) genome-wide association studies (cases: 31 307, controls: 211 753) and the GoLEAD-SUMMIT genome-wide association studies (cases: 12 086, controls: 449 548) were included in the study. There was no significant cause--and-effect relationship between coffee consumption and the risk of PAD (MVP, OR = 1.19; 95% CI: 0.92–1.54 and GoLEAD-SUMMIT, OR = 1.13; 95% CI: 0.75–1.69) [19].

In conclusion, the consumption of filter coffee does not affect, and may even be beneficial in preventing the process of atherosclerosis.

Chemical explanation of the difference in terms of the effect of coffee consumption on lipid profile

The way coffee is prepared significantly influences the effects on changes in lipid profile. In the course of filtering coffee, excess diterpenoids — kahweol and cafestol — are removed (Figure 1) [18, 20].

Compared to traditionally brewed Turkish coffee, i.e. non-filter coffee, the kahweol and cafestol content in filter coffee is not significant. It should also be borne in mind that the content of kahweol and cafestol depends on the type of coffee [20].

Kahweol and cafestol have hyperlipidemic effects (especially cafestol in humans). In hepatocytes, these compounds reduce the number of receptors for LDL-C (down--regulation), while in plasma they increase cholesteryl ester transfer protein and phospholipid transfer protein levels [18]. Furthermore, a mixture of kahweol and cafestol can reduce LCAT (lecithin: cholesterol acyltransferase) activity [17]. Kahweol and cafestol, through activation of the nuclear receptors FXR and PXR, can reduce the synthesis of sterol 27-hydroxylase and oxysterol 7-αhydroxylase, thereby reducing the conversion of cholesterol into bile acids [18]. It should be noted that with long-term coffee consumption, the hyperlipidemic effect of these diterpenoids is reduced [18]. Interestingly, in addition to the adverse hyperlipidemic effects of these diterpenoids, they show several beneficial effects, such as anti-inflammatory effects, anti-cancer effects, anti-diabetic effects, and anti-osteoporotic effects [18]. Hence the widespread recommendation to prefer filter coffee, which seems justified in those with uncontrolled, severe hypercholesterolemia but is controversial in other populations. It cannot be ruled out that many of the pleiotropic benefits that are observed with regular coffee consumption may result precisely from the presence of kahweol and cafestol in coffee.

Consumption of caffeinated coffee was found to increase lipid levels compared to decaffeinated brew [13]. Caffeine — through antagonism to certain adenosine receptor subtypes, reduction of phosphodiesterase activity in adipocytes and increased secretion of catecholamines — enhances lipolysis, resulting in the release of free fatty acids into the circulation [21]. This can have an adverse effect with a sedentary lifestyle and poor eating habits. The released free fatty acids are then not re-deposited in adipose tissue, however, they can serve to produce *de novo* triglycerides and subsequently very low-density lipoprotein and LDL-C [22]. In conclusion, the effect of caffeine on lipid profile depends not so much on its consumption but on eating habits and lifestyle of a given person.

Interestingly, *in vitro* and *in vivo* studies by Ontawong et al. [23] revealed that coffee pulp — an aqueous extract of coffee bean waste from the first stage of coffee production — acted similarly to ezetimibe, i.e., it reduced the activity of Niemann-Pick C1-Like 1 (NPC1L1) protein [23]. Furthermore, an *in vivo* study revealed that coffee polyphenols inhibited diet-induced fat accumulation by reducing the expression (down-regulation) of sterol regulatory element-binding transcription factor 1c (SREBP-1c) protein [24]. Caffeine, chlorogenic acid, trigonelline, melanoidins,

and kahweol and cafestol, as a result of their antioxidant properties, can also reduce lipid peroxidation and thus the formation of highly proatherogenic oxidised LDL fractions [25].

The resultant effect of coffee on lipid profile depends on the content of kahweol, cafestol, as well as other biologically active compounds such as chlorogenic acid (high content in green coffee, the consumption of which had a beneficial effect on lipid profile) or trigonelline, which have beneficial effects on lipid metabolism [26].

According to consumer research conducted by the SW Research Agencja Badań Rynku i Opinii on behalf of the Nespresso Poland brand, more than half of Poles consume non-filter coffee (39% ground coffee; 14% coffee from an espresso machine, 11% coffee from a moka pot), i.e. coffee with a higher diterpenoid content. Therefore, it seems useful to raise the issue of the effect of coffee on lipid profile in Polish society.

Coffee and lipids through the prism of guidelines/recommendations of scientific societies

The European Society of Cardiology CVD prevention guidelines (2021) indicate that consumption of non-filter coffee may increase LDL-C and the risk of ASCVD [27].

The Interdisciplinary Expert Position Statement supported by the Cardiovascular Pharmacotherapy Section of the Polish Cardiac Society on the treatment of dyslipidemia in Poland (Sopot Declaration IV) indicated that the consumption of decaffeinated and filter coffee does not affect the lipid profile, while the consumption of non-filter coffee may have a moderate to high hyperlipidemic effect [28].

Conclusions

Lipid disorders are a significant global problem. The main risk factors for their occurrence are poor eating habits and a sedentary lifestyle. Coffee consumption plays an important role in the diet of Poles. Results from large studies and meta-analyses in recent years indicate that consumption of 1–3 cups of filter coffee is safe in terms of the risk of lipid disorders. It should be stressed, however, that this is black coffee (espresso) without added sugar or milk. It should also be noted that dietary habits have been changing in recent years, with a trend towards drinking a higher average number of cups of coffee a day, even 3–5/day, which may be associated with a slight increase in cholesterol levels but still without clinical significance, especially for patients at low and moderate cardiovascular risk.

When considering the effect of coffee on lipid profile, several principles are useful to follow:

The effect of chronic coffee consumption, in the average number of cups/day typical of Poland, does not appear to be clinically relevant.

Although nearly 20 million Poles suffer from lipid disorders, proper treatment of these should in no way interfere with a coffee-drinking habit of 1–3 cups a day.

For a small group of individuals with uncontrolled high lipid values, a preference for filter, kahweol- and cafestol-free, optimally pure Arabica coffee could be recommended.

However, the widespread application of this principle is questionable due to the potential benefits of the pleiotropic effects of kahweol and cafestol contained in coffee on other physiological activities in addition to the effect on lipid profile.

Conflict of interest

None declared.

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