

Changes in Biochemical Parameters and LDL-Subfractions Due to the Consumption of High-Fat Sheep's Milk Yogurt Depending on Pre-Intervention Visceral Fat Area

Martina Gažarová*¹, Petra Lenártová¹, Róbert Toman²

¹Slovak University of Agriculture in Nitra, Faculty of Agrobiolgy and Food Resources, Institute of Nutrition and Genomics, Slovak Republic

²Slovak University of Agriculture in Nitra, Faculty of Agrobiolgy and Food Resources, Institute of Animal Husbandry, Slovak Republic

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Yogurt is part of the healthy eating habits of many population groups, mainly because of its significant nutritional properties. However, it is also a source of fats, the intake of which is associated with certain health risks. The aim of the study was assessment of the impact of intervention on metabolic health depending on pre-intervention visceral fat area (VFA). Twenty women were divided into two groups depending on the pre-intervention visceral fat area determined using the MF-BIA method. Biochemical parameters were determined using the Biolis 24i Premium and lipoprotein subfractions using the Lipoprint® analyzer. With the exception of triglycerides, no significant pre-post intervention differences were found between the groups ($P > 0.05$). The group of women with over-limit values of VFA had higher values of all parameters (with the exception of HDL-C). There was a significant increase in T-C, LDL-C, but also HDL-C and a decrease in cardiovascular risk in both groups, and in the case of women with over-limit VFA, an increase in triglycerides and glucose, but a decrease in the LDL/HDL ratio. Mean LDL size did not change significantly and in both cases the primary non-atherogenic phenotype did not worsen ($P > 0.05$). Our findings confirm the need for a more comprehensive assessment of the health risks of consuming foods with a higher fat content, not just on the basis of one critical component. As total cholesterol increases, it is essential to assess what changes have occurred in low- and high-density lipoproteins, as well as their subfractions and particle size.

Keywords: yogurt, sheep's milk, cholesterol, lipoproteins, subfractions, milk fat

1 Introduction

The average daily consumption of milk and dairy products among adults ranges from 180 to 450 g per capita (Quann et al., 2015). This wide range is not surprising, as recommendations for daily intake of milk and milk products are country-specific and vary widely (Dror and Allen, 2014). Recently, however, a decrease in their consumption has been recorded in several countries (Quann et al., 2015). Whole milk contains 3.7% fat, while skimmed milk contains 0.3–1.8%. Many nutritional guidelines recommend 3–5 servings of dairy products per day, emphasizing the consumption of low-fat alternatives as important components of a healthy

diet. Fats in milk consist mainly of saturated fats (over 60%), whose impact on health has been the subject of studies for several decades. Therefore, the consumption of milk and milk products is associated with metabolic health (Lorková et al., 2015).

The health and nutritional properties of foods are commonly assessed based on their individual composition and nutrient content. This approach does not very well associate one nutrient with one specific health effect. Saturated fats, which also come from milk and dairy products, have been linked to higher cholesterol and poor cardiometabolic health. However, a growing body of evidence suggests that postprandial

***Corresponding Author:** Martina Gažarová, Slovak University of Agriculture in Nitra, Faculty of Agrobiolgy and Food Resources, Institute of Nutrition and Genomics, Tr. Andreja Hlinku 2, 949 76, Nitra, Slovakia
e-mail: martina.gazarova@uniag.sk ORCID: <https://orcid.org/0000-0001-8275-7311>

responses are strongly influenced by the food matrix (Thorning et al., 2017).

Due to the content of specific nutrients and its probiotic and other health-promoting properties, yogurt is considered a functional food. For the production of yogurt, sheep's milk is an excellent raw material, especially in terms of high protein content and dry matter content (Balthazar et al., 2017). Compared to cow's milk, it has a higher content of minerals, vitamins and fat. There are higher concentrations of short- and medium-chain fatty acids in sheep's milk (Raynal-Ljutovac et al., 2008). In addition, the fat in sheep's milk contains one of the highest concentrations of conjugated linoleic acid (Revilla et al., 2017). Balthazar et al. (2016) found that mono- and polyunsaturated fatty acids in sheep's milk may contribute to the prevention of cardiovascular diseases.

In predicting various health risks, not only the assessment of fat intake, but also the assessment of the amount of body fat and its distribution in different areas of the body is of great importance (Mangla et al., 2020). Even if the consumption of full-fat dairy products is no longer associated with obesity or excessive weight gain based on current knowledge (Gažarová et al., 2021), it remains questionable how the body reacts to the intake of full-fat products when the subject is already in a pre/obese state, or there is more visceral fat in his body than is physiologically necessary. Visceral obesity is strongly associated with higher levels of pro-inflammatory and inflammatory activities in the body, which results in a higher risk of metabolic morbidity and mortality than body fat in other parts of the body (Yu et al., 2019). Visceral obesity increases cardiovascular risk by enhancing direct (dyslipidemia, hypertension, and hyperglycemia) but also indirect mechanisms. Therefore, our effort and objective was to assess changes in selected biochemical parameters, lipid profile, and low-density lipoprotein subfractions after six weeks of consumption of high-fat yogurt made from sheep's milk in female participants depending on the amount of visceral fat before the intervention.

2 Material and Methods

2.1 Study Design and Characteristics of Participants

The study was approved by the Ethics Committee of the Specialized Hospital of St. Zoerardus Zobor in Nitra, Kláštorská 131, 94901 Nitra, Slovak Republic (study protocol no. 031219_2019) and in accordance with the guidelines of the Declaration of Helsinki. It was also approved by the Slovak University of Agriculture in Nitra, Institute of Animal Husbandry and Institute of Nutrition and Genomics, Slovak Republic. Written voluntary

consent to participate in the study was required from participants.

The principle of the study consisted in the daily consumption of high-fat yogurt made from sheep's milk for six weeks. Twenty female volunteers aged 40 to 67 were included in the study. The composition of the yogurt, as well as the methodical procedure, were described in more detail in Gažarová et al. (2023). For the purposes of this study, we divided the group of women according to the pre-intervention values of the visceral fat area (healthy VFA, non-healthy VFA). A more detailed description of the study group and both groups, as well as other methodological details, are described in Gažarová et al. (2024).

2.2 Body Composition Measurement

Methodological details regarding anthropometric measurement are given in the previous publication Gažarová et al. (2023) and Gažarová et al. (2024).

2.3 Blood Sampling and Determination of Biochemical Parameters

We focused on the lipid profile (total cholesterol, T-C; low-density lipoproteins, LDL-C; high-density lipoproteins, HDL-C; LDL-C/HDL-C ratio, CVD-risk factor, triglycerides, TG), glycemia, GLU; uric acid, UA; liver enzymes (aspartate aminotransferase, AST; alanine aminotransferase, ALT; gamma-glutamyl transferase, GGT; alkaline phosphatase, ALP). Analyzes were performed using a Biolis 24i Premium biochemical analyzer (Tokyo Boeki Machinery, Tokyo, Japan). We took blood samples at the beginning of the study, before the start of the intervention and after its immediate end after six weeks of consumption. Sampling was always done in the morning after at least 8 hours of fasting. For the purposes of the study, fasting venous blood was collected from the peripheral vein of the elbow fossa in a standard manner. We obtained two 2.7 ml tubes of ethylenediaminetetraacetic acid (EDTA) and one 7.5 ml serum gel tube. We centrifuged whole blood in EDTA at 1,800 rpm for 15 min and in serum gel tubes at 3,000 rpm for 10 min at 4 °C. After separating the blood serum and plasma, we stored the samples for the determination of lipoprotein subfractions at a temperature of -80 °C until analysis.

Lipoprotein subfractions – very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL-A, IDL-B, IDL-C), and LDL1-7 – were determined in blood serum using a Lipoprint® analyzer (Quantimetrix Corp., Redondo Beach, CA, USA) with the Quantimetrix Lipoprint System LDL Subfractions Kit "Lipoprint LDL Kit" (Quantimetrix, Redondo Beach, CA, USA) according to the procedure provided by the manufacturer. This method uses linear electrophoresis on a non-denaturing polyacrylamide

gel to separate and quantify lipoprotein subfractions. Based on LDL subfraction particle size, Lipoprint® reports the LDL phenotype as non-atherogenic phenotype A (size greater than 26.8 nm), *intermediate* phenotype AB (size 26.53–26.79 nm), and atherogenic phenotype B (size less than 26.5 nm) (Muñiz et al., 2024).

2.4 Statistical Analysis

Microsoft Office Excel 2016 (Los Angeles, CA, USA) in combination with XLSTAT (Version 2019) was used to process input data. Statistical analysis was carried out using the STATISTICA 13 (TIBCO Software, Inc., Palo Alto, CA, USA). The normality of variable distribution was checked with Shapiro-Wilk test. A descriptive analysis was carried out using the mean ± standard deviation.

Levels of statistical significance were determined at $P < 0.05$. With a one-factor variance analysis (ANOVA), we tested the differences between variables and compared using Fisher's Post Hoc Test.

3 Results and Discussion

As mentioned above, the women were divided based on the input values of the visceral fat area, with a critical value of 100 cm² determined. After six weeks of consumption of sheep's yogurt, we found significant changes, but with the exception of TG, these were only changes within the groups, not between them. As shown in Table 1a, in group with an optimal VFA value, T-C ($P < 0.05$), HDL-C ($P < 0.01$), LDL-C ($P < 0.05$) and CVD risk factor ($P < 0.01$) changed significantly. T-C ($P < 0.05$), HDL-C

Table 1a Interventional changes in biochemical parameters within and between groups

Parameters	Units	Pre-post	VFA < 100 cm ²					VFA > 100 cm ²					
			mean	±SD	min	max	P-value	mean	±SD	min	max	P-value	inter-group P-value
CVD risk factor	–	day 0	1.99	0.39	1.58	2.71	0.005	2.50	0.89	1.31	4.13	0.000	ns
		day 42	1.79	0.43	1.39	2.64		2.16	0.87	0.97	3.77		ns
TC	mmol/l	day 0	5.17	1.10	3.48	6.61	0.032	5.55	0.98	3.58	6.98	0.011	ns
		day 42	5.49	1.03	3.88	6.78		5.82	0.96	4.10	7.40		ns
HDL-C	mmol/l	day 0	1.73	0.30	1.10	2.05	0.008	1.63	0.33	1.21	2.48	0.000	ns
		day 42	1.97	0.29	1.42	2.23		1.91	0.39	1.42	2.98		ns
LDL-C	mmol/l	day 0	2.64	0.67	1.88	3.68	0.047	2.92	0.76	1.74	4.30	0.001	ns
		day 42	2.86	0.63	2.10	3.94		3.14	0.79	1.93	4.64		ns
LDL-C/HDL-C	mmol/l	day 0	1.53	0.31	1.14	2.07	0.116	1.86	0.66	0.96	3.16	0.000	ns
		day 42	1.47	0.32	1.14	2.02		1.72	0.63	0.88	2.99		ns
TG	mmol/l	day 0	0.83	0.32	0.59	1.47	0.690	1.13	0.47	0.58	1.91	0.032	ns
		day 42	0.79	0.20	0.54	1.10		1.41	0.61	0.83	3.01		< 0.05
GLU	mmol/l	day 0	4.66	0.74	4.00	6.20	0.578	4.92	0.54	4.10	5.80	0.010	ns
		day 42	4.77	0.33	4.40	5.40		5.22	0.70	4.30	7.00		ns
UA	µmol/l	day 0	266	78	177	362	0.642	314	86	212	459	0.094	ns
		day 42	253	46	179	311		281	83	147	438		ns
AST	µkat/l	day 0	0.33	0.09	0.23	0.48	0.768	0.34	0.07	0.27	0.47	0.283	ns
		day 42	0.32	0.05	0.25	0.41		0.36	0.08	0.26	0.53		ns
ALT	µkat/l	day 0	0.32	0.17	0.14	0.56	0.121	0.32	0.09	0.20	0.47	0.350	ns
		day 42	0.26	0.09	0.16	0.39		0.35	0.15	0.16	0.68		ns
GGT	µkat/l	day 0	0.30	0.10	0.19	0.41	0.576	0.43	0.25	0.21	1.02	0.310	ns
		day 42	0.31	0.08	0.21	0.41		0.46	0.26	0.22	1.03		ns
ALP	µkat/l	day 0	1.15	0.22	0.88	1.54	0.030	1.24	0.34	0.73	1.90	0.146	ns
		day 42	1.08	0.23	0.76	1.38		1.19	0.34	0.79	1.97		ns

Data are expressed as mean ± standard deviation; ns – non-significant difference; TC – total cholesterol; HDL-C – high density lipoproteins; LDL-C – low density lipoproteins; TG – triglycerides; GLU – glycaemia; UA – uric acid; AST – aspartate aminotransferase; ALT – alanine aminotransferase; GGT – gamma-glutamyl transferase; ALP – alkaline phosphatase

Table 1b Interventional changes in lipoprotein subfractions within and between groups

Parameters	Units	Prepost	VFA < 100 cm ²					VFA > 100 cm ²					
			mean	±SD	min	max	P-value	mean	±SD	min	max	P-value	inter-group P-value
VLDL	mmol/l	day 0	0.88	0.31	0.54	1.53	0.826	1.09	0.29	0.67	1.66	0.614	ns
		day 42	0.84	0.37	0.15	1.22		1.12	0.25	0.72	1.58		ns
IDL-A	mmol/l	day 0	0.75	0.19	0.54	1.11	0.137	0.71	0.17	0.44	0.98	0.020	ns
		day 42	0.57	0.34	0.13	0.96		0.63	0.13	0.44	0.78		ns
IDL-B	mmol/l	day 0	0.46	0.13	0.26	0.59	0.807	0.50	0.11	0.28	0.72	0.009	ns
		day 42	0.44	0.09	0.31	0.52		0.65	0.25	0.41	1.29		ns
IDL-C	mmol/l	day 0	0.48	0.14	0.31	0.70	0.017	0.53	0.19	0.28	1.03	0.001	ns
		day 42	0.63	0.18	0.39	0.83		0.67	0.17	0.39	1.01		ns
LDL 1	mmol/l	day 0	0.80	0.26	0.44	1.22	0.418	0.89	0.19	0.59	1.19	0.010	ns
		day 42	0.70	0.35	0.21	1.01		0.75	0.17	0.54	1.14		ns
LDL 2	mmol/l	day 0	0.27	0.15	0.05	0.41	0.679	0.38	0.22	0.13	0.80	0.508	ns
		day 42	0.24	0.12	0.10	0.44		0.36	0.27	0.13	0.96		ns
LDL 3–7	mmol/l	day 0	0.04	0.06	0.00	0.16	1.000	0.08	0.13	0.00	0.47	0.275	ns
		day 42	0.04	0.07	0.00	0.18		0.11	0.19	0.00	0.57		ns
Mean LDL size	nm	day 0	27.24	0.23	26.80	27.50	0.420	27.15	0.31	26.40	27.40	0.321	ns
		day 42	27.14	0.20	26.80	27.40		27.07	0.43	26.10	27.50		ns

Data are expressed as mean ± standard deviation; ns – non-significant difference; VLDL – very low density lipoproteins; IDL – intermediate density lipoprotein A/B/C; LDL 1–7 – low density lipoprotein 1–7

($P < 0.001$), LDL-C ($P < 0.01$) and CVD risk factor ($P < 0.001$) values, as well as the LDL-C/HDL-C ratio ($P < 0.001$), TG ($P < 0.05$) and GLU ($P < 0.05$) values significantly changed in women with an over-limit VFA. In both cases, there was an increase in T-C and LDL-C, but also HDL-C, but what is important, the values of the CVD risk factor and the ratio of LDL to HDL decreased. It is also important that, except for T-C, all values were within the range of reference values. We found a significant intergroup difference only in TG, when there was a significant increase in its values in the over-limit VFA group, even though the values were still within the norm.

As mentioned above, there was a significant increase in LDL-C values in both groups, but it is important to determine to what extent its subfractions and LDL particle size changed (Table 1b). In the group with optimal VFA values, we noted a significant change only in the case of intermediate density lipoprotein C/IDL-C ($P < 0.05$). In the group with over-limit VFA values, in addition to IDL-C ($P < 0.01$), we also found changes in IDL-A ($P < 0.05$), IDL-B ($P < 0.01$) and LDL1 ($P < 0.05$). While in the case of IDL-C the values changed from optimal to above the limit, in the case of IDL-A it was the opposite. Also important is the fact that there was no significant decrease in the size of LDL particles ($P > 0.05$).

In Table 2a, we present the pre-post intervention differences of biochemical parameters within the groups. In most cases, the same changes occurred in groups with an increasing (T-C, HDL-C, LDL-C, GLU) or decreasing (CVD risk factor, LDL/HDL ratio, UA) tendency. We found a different trend of changes in values between groups only in the case of TG.

In Table 2b, we present the pre-post intervention differences of lipoprotein subfractions within the groups. We found a decreasing trend in both groups in the case of IDL-A, LDL1 and LDL2, as well as mean LDL size, and rising trend in the case of IDL-C. We found a different development of values in the case of VLDL, IDL-B and LDL3-7, when there was an increase in values in the group with above-limit VFA values.

Based on the results, we conclude that six weeks of consuming high-fat sheep's milk yogurt did not produce fundamentally different effects between groups of women stratified by their baseline visceral fat area (VFA). The only significant intergroup difference was observed in triglyceride (TG) levels. In the group of women with elevated VFA, TG levels increased significantly ($P < 0.05$), accompanied by rising levels of VLDL ($P > 0.05$), IDL-C ($P < 0.01$), and LDL3–7 ($P > 0.05$). Overall, this trend can be considered unfavorable. In the case of the other

Table 2a Intervention differences of biochemical parameters within groups

Parameters	Units	Group	Mean diff.	±SD	Min of diff.	Max of diff.	Inter-group <i>P</i> -value
CVD risk factor	–	VFA < 100 cm ²	-0.20	0.12	-0.43	-0.07	0.072
		VFA > 100 cm ²	-0.34	0.21	-0.82	0.00	
TC	mmol/l	VFA < 100 cm ²	0.31	0.30	-0.03	0.84	0.769
		VFA > 100 cm ²	0.27	0.32	-0.30	0.64	
HDL-C	mmol/l	VFA < 100 cm ²	0.24	0.16	0.08	0.51	0.610
		VFA > 100 cm ²	0.28	0.13	0.11	0.50	
LDL-C	mmol/l	VFA < 100 cm ²	0.23	0.24	-0.10	0.67	0.975
		VFA > 100 cm ²	0.22	0.19	-0.06	0.53	
LDL-C/HDL-C	mmol/l	VFA < 100 cm ²	-0.07	0.10	-0.23	0.05	0.145
		VFA > 100 cm ²	-0.14	0.10	-0.28	0.02	
TG	mmol/l	VFA < 100 cm ²	-0.03	0.22	-0.37	0.24	0.039
		VFA > 100 cm ²	0.28	0.42	-0.24	1.43	
GLU	mmol/l	VFA < 100 cm ²	0.11	0.51	-0.80	0.70	0.416
		VFA > 100 cm ²	0.30	0.35	-0.10	1.20	
UA	μmol/l	VFA < 100 cm ²	-13.43	72.71	-116.00	81.00	0.562
		VFA > 100 cm ²	-33.15	65.72	-153.00	84.00	
AST	μkat/l	VFA < 100 cm ²	-0.01	0.05	-0.07	0.06	0.308
		VFA > 100 cm ²	0.02	0.08	-0.10	0.14	
ALT	μkat/l	VFA < 100 cm ²	-0.06	0.09	-0.21	0.06	0.066
		VFA > 100 cm ²	0.03	0.12	-0.10	0.21	
GGT	μkat/l	VFA < 100 cm ²	0.01	0.04	-0.06	0.06	0.537
		VFA > 100 cm ²	0.03	0.11	-0.19	0.24	
ALP	μkat/l	VFA < 100 cm ²	-0.08	0.07	-0.16	0.05	0.438
		VFA > 100 cm ²	-0.05	0.11	-0.26	0.11	

Data are expressed as mean ± standard deviation; TC – total cholesterol; HDL-C – high density lipoproteins; LDL-C – low density lipoproteins; TG – triglycerides; GLU – glycaemia; UA – uric acid; AST – aspartate aminotransferase; ALT – alanine aminotransferase; GGT – gamma-glutamyl transferase; ALP – alkaline phosphatase

Table 2b Intervention differences of lipoprotein subfractions within groups

Parameters	Units	Group	Mean diff.	±SD	Min of diff.	Max of diff.	Inter-group P-value
VLDL	mmol/l	VFA < 100 cm ²	-0.04	0.43	-0.65	0.39	0.696
		VFA > 100 cm ²	0.03	0.24	-0.49	0.36	
IDL-A	mmol/l	VFA < 100 cm ²	-0.18	0.28	-0.57	0.13	0.405
		VFA > 100 cm ²	-0.08	0.11	-0.26	0.16	
IDL-B	mmol/l	VFA < 100 cm ²	-0.01	0.15	-0.23	0.23	0.050
		VFA > 100 cm ²	0.15	0.17	-0.08	0.57	
IDL-C	mmol/l	VFA < 100 cm ²	0.15	0.12	0.00	0.39	0.942
		VFA > 100 cm ²	0.15	0.12	-0.03	0.36	
LDL 1	mmol/l	VFA < 100 cm ²	-0.10	0.30	-0.65	0.28	0.758
		VFA > 100 cm ²	-0.14	0.17	-0.47	0.16	
LDL 2	mmol/l	VFA < 100 cm ²	-0.02	0.13	-0.23	0.13	0.970
		VFA > 100 cm ²	-0.02	0.11	-0.21	0.16	
LDL 3–7	mmol/l	VFA < 100 cm ²	0.00	0.09	-0.13	0.13	0.430
		VFA > 100 cm ²	0.04	0.12	-0.05	0.41	
Mean LDL size	nm	VFA < 100 cm ²	-0.10	0.31	-0.40	0.40	0.870
		VFA > 100 cm ²	-0.08	0.27	-0.80	0.20	

Data are expressed as mean ± standard deviation; VLDL – very low density lipoproteins; IDL – intermediate density lipoprotein A/B/C; LDL 1–7 – low density lipoprotein 1–7

parameters, we did not find statistically significant intergroup differences. Despite the trend of increasing T-C and LDL-C values in both groups, there was an increase in HDL-C and a positive reduction in cardiovascular risk factor and LDL-C/HDL-C ratio. Equally important is the mean LDL size, which did not change significantly, even though there was a downward trend in the values. In both groups, the mean LDL size indicated a non-atherogenic phenotype A, which did not change even after 42 days of high-fat yogurt consumption.

Consumption of low-fat products is generally recommended precisely because of the strong association of increased fat intake with cardiovascular health risks. A meta-analysis by Rees et al. (2019) evaluating the Mediterranean diet showed, however, that as part of primary prevention, the positive effect on the lipid profile was minimal. A slight decrease in T-C was detected, but the concentrations of LDL-C, HDL-C and TG did not change fundamentally. Chiu et al. (2016) evaluated the effect of the Dietary Approaches to Stop Hypertension diet and its modified high-fat, low-carbohydrate version on hypertension and lipid profile. After three weeks, they found that the unmodified DASH diet reduced concentrations of IDL-C and LDL-C, including concentrations of large LDL particles, as well as HDL cholesterol and apolipoprotein I. The modified DASH reduced blood pressure, TG concentration, and increased the concentration of large LDL particles without affecting HDL. Large subfractions of LDL1 and LDL2 are associated with no or low cardiovascular risk

(Muñiz et al., 2024). According to Allaire et al. (2017), it is the size of LDL particles that becomes a non-causal risk factor for coronary heart disease.

It is generally known that a bad lipid profile represents an increased level of T-C and LDL-C, as well as triglycerides, and at the same time a decreased level of HDL-C (Khatana et al., 2020). For a long time, this condition appeared to be an important negative factor in the process of atherogenesis (Sharma and Ganguly, 2005). However, the discovery of lipoprotein subfractions questions the importance and relevance of cholesterol content in LDL-C and HDL-C (Arsenault et al., 2009; Superko et al., 2012). The influence of fats on the lipid profile of consumers is undeniable, but it is currently highly debatable whether an increase in LDL-C is also related to an increased cardiovascular risk. Numerous clinical studies have shown that the initiation and progression of atherosclerosis is determined by the number, size and modification of LDL particles. Sniderman pointed out the importance of not the total concentration of LDL-C, but LDL particles as an important determinant in the pathogenesis of cardiovascular diseases (Sniderman et al., 2022; Vekic et al., 2022).

Visceral adipose tissue is anatomically composed of a larger number of large and dysfunctional adipocytes. The high lipolytic activity of abdominal visceral fat releases free fatty acids, which are drained via the portal vein and accumulate in the liver. The liver increases lipid content and secretion of triglyceride-rich lipoproteins

(VLDL, Apo-B). Increased lipolysis and higher fatty acid levels lead to hyperglycemia, hyperinsulinemia, and hypertriglyceridemia (Misra and Vikram, 2003; Neeland et al., 2017).

A correlation analysis by Sam et al. (2024) demonstrated significant relationships between visceral adiposity and lipid profile. The authors found a significant positive correlation between VFA and total cholesterol ($r = 0.65$), LDL-C ($r = 0.58$), TG ($r = 0.52$) and conversely, a significant negative correlation was found between VFA and HDL-C ($r = -0.48$). This inverse relationship suggests that higher visceral fat is associated with lower HDL-C, which is known to have a protective role in cardiovascular health.

A study by Sukkriang et al. (2021) demonstrated significant positive correlations between TG and VFA in obese adults aged ≥ 40 years and with BMI ≥ 30 kg/m². In addition, their findings revealed a significant negative correlation between HDL-C and VFA in adults aged < 40 years and aged ≥ 40 years. The studies demonstrated a significant correlation between visceral adiposity and lipid profile, confirming the key role of visceral fat in the pathogenesis of metabolic disorders.

4 Conclusions

In conclusion, six weeks of high-fat sheep's yogurt consumption led to significant within-group changes in lipid and biochemical parameters, but only minimal differences between groups stratified by visceral fat area (VFA). Both groups showed increases in T-C and LDL-C, accompanied by a rise in HDL-C and a reduction in the CVD risk factor and LDL-C/HDL-C ratio, suggesting a potentially neutral overall effect on cardiovascular risk. Except for T-C, most parameters remained within reference ranges. A significant intergroup difference was observed only for triglycerides, which increased in women with elevated VFA, indicating a less favorable response in this subgroup. Changes in lipoprotein subfractions were modest, and importantly, LDL particle size remained unchanged, maintaining a non-atherogenic phenotype A in both groups. Overall, these findings suggest that high-fat sheep's yogurt does not substantially differentially impact lipid metabolism based on visceral adiposity, although the observed triglyceride increase in women with higher VFA may warrant further attention.

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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

Gažarová, Lenártová – conceptualization, methodology, software, validation, formal analysis, data curation, writing – original draft preparation, writing – review and editing. Toman – visualization, project administration, investigation supervision, funding acquisition. All authors have read and agreed to the published version of the manuscript.

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