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Fatty acids profile in plasma of patients with monoclonal gammopathy of undetermined significance (MGUS) and multiple myeloma (MM)

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Abstract: Background: Monoclonal gammopathies, including monoclonal gammopathy of undetermined significance (MGUS) and multiple myeloma (MM), are plasma-cell disorders linked to profound metabolic remodeling. Lipid metabolism, particularly the balance between n-3 and n-6 polyunsaturated fatty acids (PUFAs), plays a pivotal role in inflammation, membrane dynamics, and tumor progression.
Methods: Plasma fatty acid (FA) profiles were analyzed in patients with MGUS, MM, and healthy controls via gas chromatography. Desaturase indices (Δ5D, Δ6D, Δ9SCD1, Δ9SCD2) were calculated from FA ratios.
Results: MM patients exhibited significantly higher palmitic acid and lower n-3 PUFA levels (EPA, DHA) compared to controls ($p < 0.05$), reflecting a pro-inflammatory lipid milieu. MGUS patients showed increased oleic acid and markedly elevated Δ9-desaturase activity, suggesting enhanced monounsaturated FA synthesis during early plasma-cell transformation. The n-3/n-6 ratio decreased progressively from controls (1.0) to MGUS (0.5) and MM (0.4). Trans fatty acids were substantially elevated in MGUS and MM compared to the control group.
Conclusion: Altered plasma FA composition and desaturase activity indicate lipid metabolic reprogramming in MGUS and MM. The progressive reduction of the n-3/n-6 PUFA ratio underscores a shift toward a pro-inflammatory state that may promote malignant transformation. Lipidomic profiling could serve as an early biomarker of disease evolution, and dietary modulation of n-3 PUFAs may hold therapeutic potential.

Keywords: MGUS, multiple myeloma, fatty acids, lipid metabolism, inflammation, desaturase activity.

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Introduction

Monoclonal gammopathies encompass a spectrum of plasma cell disorders characterized by the clonal expansion of B cells producing monoclonal immunoglobulins. Among these, monoclonal gammopathy of undetermined significance (MGUS) is the most prevalent premalignant condition, affecting approximately 3–5% of adults over 50 years of age [1, 2]. MGUS progresses to multiple myeloma (MM) at an estimated rate of 1% per year, and MM itself accounts for about 1% of all malignancies and 13% of hematologic cancers [3]. Despite therapeutic advances, MM remains incurable mainly, with metabolic reprogramming emerging as a hallmark of disease persistence and drug resistance [4, 5].

Growing evidence indicates that lipid metabolism plays a crucial role in tumor biology, influencing membrane structure, energy homeostasis, and inflammatory signaling [6, 7]. Cancer cells often display enhanced *de novo* fatty acid (FA) synthesis and altered desaturation patterns to support membrane biogenesis, proliferation, and redox balance. Enzymes such as fatty acid synthase (FASN) and stearoyl-CoA desaturases (SCD1/SCD2) are frequently upregulated in malignant cells, promoting growth and chemoresistance [8–10].

In MM, metabolic remodeling extends beyond intrinsic tumor cell pathways to involve the bone marrow microenvironment. Bone marrow adipocytes release free fatty acids and adipokines that fuel myeloma cells, modulate immune function, and promote tumor progression [11–13]. Long-chain fatty acid uptake by myeloma cells suppresses $CD8^+$ T-cell metabolism and impairs anti-tumor immunity [14]. Moreover, alterations in polyunsaturated fatty acids (PUFAs) — particularly the balance between n-3 and n-6 series — have been linked to inflammatory tone and tumor-promoting signaling within the bone marrow niche [15, 16].

A deficiency of n-3 PUFAs (e.g., EPA, DHA) and excess of n-6 PUFAs (e.g., arachidonic acid) promote the synthesis of pro-inflammatory eicosanoids via COX and LOX pathways. At the same time, n-3–derived mediators such as resolvins and protectins that resolve inflammation and restore immune homeostasis [17–19]. Clinical and experimental studies indicate that modulating FA profiles through dietary or pharmacologic means can influence myeloma biology, sensitize cells to therapy, and mitigate systemic inflammation [20–30].

Despite growing insights, comparative data on systemic FA profiles in MGUS and MM remain limited. Understanding how lipid composition and desaturase activity differ between these entities may elucidate early biochemical events that precede malignant transformation.

Therefore, the present study aimed to characterize and compare plasma fatty acid composition and desaturase activities in patients with MGUS, MM, and healthy controls, with a particular focus on the n-3/n-6 balance as a potential indicator of inflammatory status and metabolic reprogramming.

Materials and Methods

Clinical samples

Plasma samples were collected from patients diagnosed with MGUS (n = 7), MM (n = 15), and age-matched healthy volunteers (n = 15) treated at the Department of Hematology, University Hospital in Kraków, Poland. All participants provided written informed consent. The study adhered to the Declaration of Helsinki and received approval from the Local Bioethics Committee.

Sample preparation

Venous blood was drawn into K_2-EDTA tubes and centrifuged (1500×g, 10 min) to separate plasma. To prevent lipid oxidation, 10 µL of 0.05% butylated hydroxytoluene (BHT) was added to each sample.

Lipid extraction and FA analysis

Total lipids were extracted using a chloroform–methanol mixture (2:1, v/v). Fatty acids were methylated with 14% boron trifluoride (BF_3) in methanol. Fatty acid methyl esters (FAMEs) were analyzed using gas chromatography (Agilent 6890N) equipped with a flame-ionization detector (FID) and a DB-23 column (60 m × 0.25 µm). The oven temperature was programmed from 140°C to 240°C. FAMEs were identified by retention times compared to commercial standards, and results were expressed as the percentage of total fatty acids. Results were expressed as relative percentages of total FAs. Total saturated (SFA), monounsaturated (MUFA), n-6 and n-3 PUFAs, trans-FAs, and n-3/n-6 ratios were calculated. Desaturase activity indices were calculated as follows: Δ5D = 20:4n-6/20:3n-6, Δ6D = 20:3n-6/18:2n-6, Δ9SCD1 = 16:1/16:0, and Δ9SCD2 = 18:1/18:0.

Statistical analysis

Group differences were tested using one-way ANOVA followed by Scheffé post hoc analysis (STATISTICA 13.1; StatSoft Inc., Tulsa, USA). Data are presented as mean ± SD, with significance defined as $p \leq 0.05$.

Results

Table 1 summarizes the plasma fatty acid composition across study groups. Healthy controls showed a predominance of palmitic, arachidonic, and linolenic acids, whereas MGUS patients exhibited higher levels of oleic acid. MM patients demonstrated significantly elevated palmitic acid and lower levels of n-3 fatty acids (EPA, DHA) compared to controls ($p \leq 0.05$). The n-3/n-6 ratio decreased progressively from controls (1.0) to MGUS (0.5) and MM (0.4) (Table 1).

Table 2 presents desaturases activity indices. MGUS patients exhibited significantly increased Δ9-desaturase (SCD1 and SCD2) activities, suggesting enhanced monounsaturated fatty acid synthesis, whereas MM patients showed lower Δ5-desaturase activity and reduced conversion of linoleic acid to arachidonic acid.

The analysis of plasma fatty acid (FA) profiles revealed significant differences among healthy controls, MGUS, and MM patients. In the control group, palmitic, arachidonic, and α-linolenic acids were the predominant species. Patients with MGUS exhibited a distinct shift toward higher levels of oleic acid (C18:1n-9), accompanied by a reduction in α-linolenic acid. In contrast, MM patients exhibited a marked accumulation of palmitic acid (C16:0) and a notable decrease in total n-3 polyunsaturated fatty acids (PUFAs), including eicosapentaenoic (EPA), docosapentaenoic (DPA) and docosahexaenoic (DHA) acids ($p < 0.05$) (Table 1).

When comparing FA classes, saturated fatty acids (SFA) were most abundant in MM (34.2%), moderate in controls (31.5%), and lowest in MGUS (21.5%). Conversely, unsaturated fatty acids (UFA) dominated in the MGUS group (78.5%), indicating enhanced desaturation activity.

Monounsaturated fatty acids (MUFA) were elevated in MM (27.6%) relative to controls (22.3%) and MGUS (19.2%), consistent with increased activity of stearoyl-CoA desaturases (Table 1, 2).

Of particular interest, trans-fatty acids (TFAs) were substantially higher in MGUS (3.1%) and MM (2.4%) compared with healthy controls (0.2%), suggesting increased lipid peroxidation or disturbed lipid turnover during disease progression.

The n-3/n-6 PUFA ratio showed a progressive decline across study groups. This reduction reflects a shift toward a pro-inflammatory lipid environment dominated by n-6–derived species such as arachidonic acid. Notably, MGUS patients displayed a twofold decrease in this ratio relative to controls, while MM patients demonstrated an even lower proportion of n-3 PUFAs, indicating further metabolic deterioration with disease advancement (Fig. 1).

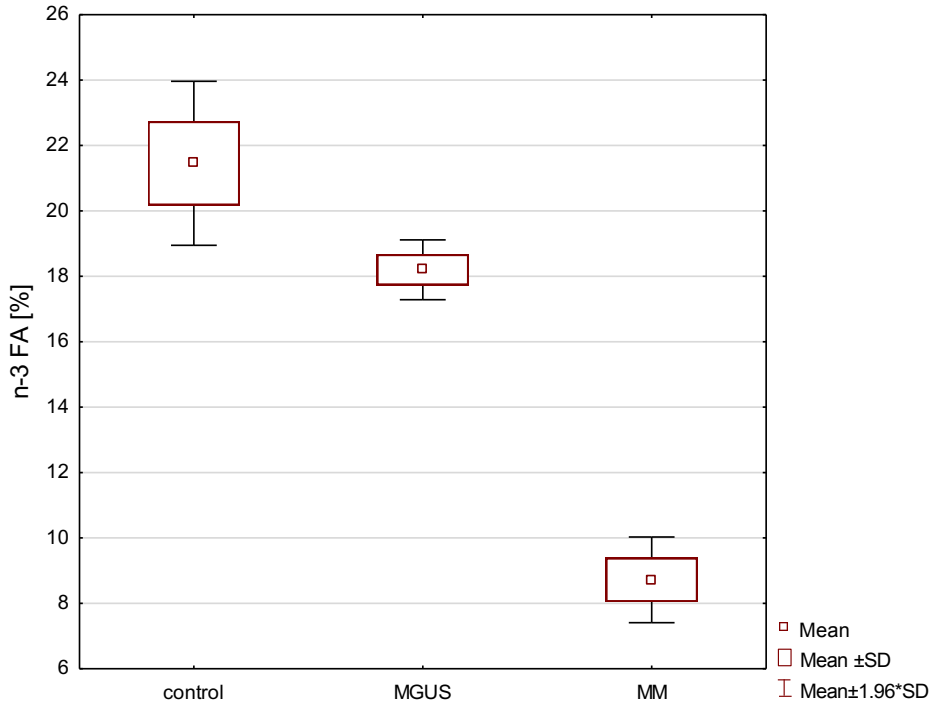

Fig. 1. Total n-3 fatty acids [%] in plasma of patients with MGUS and MM and in the control group.

Desaturase activity patterns differed markedly between groups. The Δ9-desaturase (SCD1) index was highest in MGUS (12.0), suggesting enhanced conversion of saturated to monounsaturated FAs and active membrane remodeling at the premalignant stage. Δ9SCD2 activity was also elevated in MGUS compared with controls and MM, reinforcing a trend toward MUFA synthesis (Table 2).

In contrast, Δ5-desaturase (D5) activity was strongly suppressed in MGUS (2.8) and MM (8.6) compared with controls (23.2), indicating impaired biosynthesis of arachidonic acid from its precursor. Δ6-desaturase (D6) activity remained low and did not differ significantly between groups. These enzymatic alterations suggest that both MGUS and MM are associated with disrupted FA desaturation control, leading to a pro-inflammatory and lipogenic plasma profile (Table 2).

Table 1. Fatty acid content [%] in plasma in the study groups. Means ± SD (min-max). SFA — saturated Fatty Acid, MUFA — Monounsaturated Fatty Acid, PUFA — Polyunsaturated Fatty Acid.

Fatty acid	Control	MGUS	MM
SFA			
Myristic C14:0	2.8^{a} ± 1.4 (0.8–5.1)	1.2^{b} ± 0.1 (1.1–1.3)	1.6^{b} ± 0.6 (0.8–2.8)
Pentadecanoic C15:0	2.0^{a} ± 2.1 (0.0–5.6)	1.7^{a} ± 0.2 (1.4–1.9)	0.4^{b} ± 0.1 (0.1–0.6)
Palmitic C16:0	15.2^{a} ± 4.2 (9.6–24.4)	12.2^{a} ± 0.4 (11.9–12.6)	22.4^{b} ± 2.8 (16.7–27.0)
Stearic C18:0	9.3^{a} ± 3.1 (3.8–15.0)	6.4^{a} ± 4.1 (1.7–9.4)	8.6^{a} ± 1.8 (6.2–13.6)
Arachidic C20:0	2.2^{a} ± 1.0 (0.3–2.2)	0.0	1.2^{b} ± 0.5 (0.4–1.7)
MUFA			
Myristoleic C14:1n-5	2.2^{a} ± 1.0 (0.0–3.7)	2.4^{a} ± 1.4 (1.3–4.0)	3.1^{a} ± 1.4 (1.4–6.0)
Pentadecanoic C15:1n-7	2.2^{a} ± 1.2 (0.0–4.7)	1.6^{a} ± 0.8 (0.7–2.0)	6.5^{b} ± 5.0 (1.6–15.1)
Palmitoleic C16:1n-7	8.5^{a} ± 2.3 (4.8–14.4)	1.1^{b} ± 0.4 (0.7–1.4)	2.2^{b} ± 1.1 (0.8–3.6)
Oleic C18:1 n-9	7.8^{a} ± 2.1 (4.8–13.0)	14.1^{b} ± 1.3 (13.3–15.5)	9.7^{a} ± 1.6 (7.4–12.6)
Vaccenic18:1n-7	0.0	0.0	0.7 ± 0.2 (0.4–1.1)
cis-11-eicosenoic C20:1n-11	1.3^{a} ± 1.2 (0.0–4.1)	0.0	2.9^{b} ± 1.3 (1.0–4.7)
Erucic C22:1n-9	0.3^{a} ± 0.7 (0.0–2.6)	0.0	1.5^{b} ± 0.9 (0.0–3.4)
Nervonic C24:1n-9	0.0	0.0	1.0 ± 0.5 (0.0–1.6)
PUFA n-6			
Linoleic C18:2n-6	3.6^{a} ± 1.9 (1.1–7.3)	9.1^{b} ± 1.2 (7.9–10.4)	7.7^{b} ± 2.6 (3.3–14.8)
γ-linolenicC18:3n-6	0.0	3.4^{a} ± 1.9 (1.2–4.8)	5.7^{b} ± 3.4 (1.7–15.2)
cis-11,14-eicosadienoic C20:2n-6	2.2^{a} ± 2.3 (0.0–7.8)	2.7^{a} ± 1.2 (1.4–3.8)	2.4^{a} ± 2.1 (0.5–5.8)
cis-8,11,14-eicosatrienoic C20:3n-6	1.4^{a} ± 2.8 (0.4–11.3)	2.0^{a} ± 0.5 (1.5–2.3)	1.3^{a} ± 1.1 (0.7–3.9)
Arachidonic C20:4n-6	14.1^{a} ± 5.2 (7.3–21.6)	14.3^{a} ± 1.2 (4.5–6.6)	8.6^{b}± 2.6 (4.0–11.8)
cis-13,16-docosadienoic C22:2n-6	0.3 ± 0.6 (0.0–1.9)	0.0	0.0
Adrenic C22:4n-6	1.4^{a} ± 0.6 (0.5–2.5)	1.3^{a} ± 0.8 (1.1–2.1)	1.0^{a} ± 0.4 (0.6–1.6)
PUFA n-3			
α-Linolenic	10.5^{a} ± 4.2 (1.3–19.5)	5.2^{b} ± 1.4 (3.6–6.0)	3.6^{c} ± 1.7 (0.0–5.1)
cis-11,14,17-eicosatrienoic	2.1^{a} ± 1.1 (0.9–5.0)	4.7^{b} ± 3.8 (2.5–9.1)	1.8^{a} ± 0.9 (0.0–3.1)
Eicosapentaenoic C20:5n-3	4.3^{a} ± 2.0 (2.3–9.9)	6.1^{a} ± 0.6 (5.5–6.8)	2.1^{b} ± 0.8 (0.8–3.4)
Docosapentaenoic C22:5n-3	2.1^{a} ± 1.0 (0.7–3.7)	0.4^{b} ± 0.2 (0.2–0.6)	0.3^{b} ± 0.2 (0.2–0.5)
Docosahexaenoic C22:6n-3	2.5^{a} ± 1.4 (0.2–5.9)	2.2^{a} ± 0.7 (1.8–3.0)	0.9^{b} ± 1.4 (1.2–5.1)

Table 1. Cont.

Fatty acid	Control	MGUS	MM
Trans FA			
Elaidic C18:1n-9t	$0.2^{a} \pm 0.4$ (0.0–1.1)	$1.5^{b} \pm 0.3$ (1.1–1.6)	$0.3^{a} \pm 0.4$ (0.0–1.3)
Linolelaidic C18:2n-6t	0.0	$1.6^{a} \pm 0.6$ (1.2–2.3)	$2.1^{b} \pm 0.2$ (0.4–1.1)
SFA Total	$31.5^{a} \pm 5.9$ (23.7–42.5)	$21.5^{b} \pm 4.4$ (16.5–24.3)	$34.2^{a} \pm 3.6$ (28.1–43.3)
UNSAT Total	$68.5^{a} \pm 5.1$ (57.5–76.3)	$78.5^{b} \pm 4.1$ (75.7–83.5)	$65.8^{a} \pm 3.1$ (56.7–71.9)
MUFA Total	$22.3^{a} \pm 4.2$ (14.2–32.4)	$19.2^{b} \pm 1.3$ (18.2–20.7)	$27.6^{c} \pm 4.2$ (21.5–35.1)
trans Total	$0.2^{a} \pm 0.4$ (0.0–1.1)	$3.1^{b} \pm 0.3$ (2.9–3.4)	$2.4^{c} \pm 0.8$ (1.4–3.6)
n-3/n-6 Ratio	$1.0^{a} \pm 0.3$ (0.4–1.5)	$0.5^{b} \pm 0.1$ (0.4–0.6)	$0.4^{b} \pm 0.2$ (0.2–0.7)

a, b, c — different letters indicate statistically significant differences, $p < 0.05$.

Table 2. Desaturases activity in patients with MGUS and MM and in the control group. Desaturase activity indices were calculated as follows: Δ5D = 20:4n-6/20:3n-6, Δ6D = 20:3n-6/18:2n-6, Δ9SCD1 = 16:1/16:0, and Δ9SCD2 = 18:1/18:0.

Desaturase	Control	MGUS	MM
D5	$23.2^{a} \pm 14.3$ (0.7–39.9)	$2.8^{b} \pm 1.5$ (2.0–4.6)	$8.6^{c} \pm 4.2$ (2.4–15.6)
D6	$0.4^{a} \pm 0.5$ (0.1–2.0)	$0.2^{a} \pm 0.0$ (0.2–0.3)	$0.2^{a} \pm 0.2$ (0.1–0.7)
D9SCD1	$0.6^{a} \pm 0.2$ (0.2–1.0)	$12.0^{b} \pm 5.5$ (8.6–18.4)	$0.1^{c} \pm 0,0$ (0.0–0.2)
D9SCD2	$0.9^{a} \pm 0.3$ (0.4–1.6)	$4.0^{b} \pm 4.3$ (1.4–9.0)	$1.2^{a} \pm 0.3$ (0.8–1.7)

a, b, c — different letters indicate statistically significant differences, $p < 0.05$.
Desaturase activity indices were calculated as follows: Δ5D = 20:4n-6/20:3n-6, Δ6D = 20:3n-6/18:2n-6, Δ9SCD1 = 16:1/16:0, and Δ9SCD2 = 18:1/18:0.

Discussion

This study demonstrates that both MGUS and MM are associated with significant alterations in plasma fatty acid composition, reflecting metabolic and inflammatory dysregulation. The elevated palmitic acid and reduced n-3 PUFA levels observed in MM are consistent with enhanced *de novo* lipogenesis and chronic inflammation described in previous reports [4, 15, 17]. These findings support the concept that lipid metabolism plays a role in malignant plasma-cell transformation and tumor progression.

In MGUS, increased Δ9-desaturase (SCD1/SCD2) activity suggests an early metabolic adaptation that favors membrane fluidity and proliferation. Elevated monounsaturated fatty acid synthesis may represent an intermediate stage of metabolic reprogramming preceding full malignancy [5, 6, 20]. The progressive decline in the n-3/n-6 ratio from controls to MGUS and MM underscores a transition toward a pro-inflammatory lipid phenotype, characterized by excessive n-6 PUFA-derived eicosanoids and impaired production of anti-inflammatory lipid mediators [9, 13, 14, 18].

Emerging evidence suggests that myeloma cells heavily rely on fatty acid uptake and storage to sustain growth and survival. Bone marrow adipocytes serve as active lipid reservoirs, providing

long-chain fatty acids that promote tumor persistence and immune evasion [2–4, 20]. This metabolic crosstalk between tumor cells and adipocytes establishes a lipid-rich microenvironment that favors angiogenesis, inflammation, and drug resistance [1, 9, 12].

Moreover, reduced Δ5-desaturase activity observed in both MGUS and MM may impair the conversion of linoleic to arachidonic acid, further disrupting PUFA homeostasis. Conversely, increased Δ9-desaturase activity enhances MUFA synthesis, supporting membrane remodeling and redox stability, processes known to facilitate cancer cell survival [5, 6, 8]. Together, these findings suggest that desaturase activity shifts toward lipid saturation and MUFA enrichment as MGUS progresses to MM.

Therapeutically, lipid metabolism has emerged as a promising target. Inhibition of fatty acid synthase (FASN) or stearoyl-CoA desaturase (SCD1) has been shown to induce apoptosis and sensitize myeloma cells to chemotherapy [6–8]. Recent data indicate that dietary modulation, particularly increasing n-3 PUFA intake, can attenuate inflammation and potentially delay disease progression [10, 20–30]. These interventions align with the concept that restoring metabolic balance could complement standard antimyeloma therapy [27–30].

The present study is limited by a small cohort size and a cross-sectional design, which preclude causal inference. Dietary habits and lifestyle factors influencing FA profiles were not fully controlled. Nevertheless, the observed differences provide valuable insight into early metabolic changes in plasma-cell disorders and support further lipidomic investigation on a larger scale.

In conclusion, our results reinforce the notion that dysregulated lipid metabolism and a disturbed n-3/n-6 balance are integral to the pathophysiology of MGUS and MM. The integration of lipidomic profiling into clinical assessment may provide novel biomarkers for early detection and monitoring of disease progression. Moreover, targeting lipid metabolic pathways, through pharmacological inhibition or dietary strategies, holds potential to modify disease trajectory and improve therapeutic outcomes.

Authors contributions

The conception and design of the study, J.G.A., A.J.; acquisition of data, J.G.A., A.J., K.S., A.P; analysis and interpretation of data, J.G.A., K.S.; drafting the article J.G.A.; final approval of the version to be submitted: all Authors.

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Conflict of interest

The authors otherwise disclose no conflicts of interest.

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