

Abnormal octadeca-carbon fatty acids distribution in erythrocyte membrane phospholipids of patients with gastrointestinal tumor

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Abstract

Fatty acid (FA) composition is closely associated with tumorigenesis and neoplasm metastasis. This study was designed to investigate the differences of phospholipid FA (PLFA) composition in erythrocyte and platelet cell membranes in both gastrointestinal (GI) tumor patients and healthy controls.

In this prospective study, 50 GI tumor patients and 33 healthy volunteers were recruited between the years 2013 and 2015. Blood samples were collected from healthy volunteers and patients, and FA composition was assessed using gas chromatography-mass spectrometer (GC-MS), and data were analyzed by multifactor regression analysis.

Compared with healthy controls, the percentages of C18:0 (stearic acid, SA), C22:6 (docosahexaenoic acid, DHA), and n-3 polyunsaturated FAs (n-3 PUFA) were significantly increased, while C18:1 (oleic acid, OA), C18:2 (linoleic acid, LA), and monounsaturated FAs (MUFA) decreased in erythrocyte membranes of GI tumor patients. Also, patient's platelets revealed higher levels of C20:4 (arachidonic acid, AA) and DHA, and lower levels of OA and MUFA.

Our study displayed a remarkable change in the FA composition of erythrocyte and platelet membranes in GI tumor patients as compared with healthy controls. The octadeca-carbon FAs (SA, OA, and LA) in erythrocyte membranes could serve as a potential indicator for GI tumor detection.

Abbreviations: AA = arachidonic acid, ALA = α -linolenic acid, ANOVA = analysis of variance, BMI = body mass index, CRC = colorectal cancer, DHA = docosahexaenoic acid, DHLA = dihomogamma-linolenic acid, DPA = docosapentaenoic acid, ECG = electrocardiogram, EPA = eicosapentaenoic acid, FA = fatty acid, GC = gastric cancer, GC-MS = gas-chromatography-mass spectrometry, GI = gastrointestinal, GLA = gamma-linolenic acid, LA = linoleic acid, MUFA = monounsaturated fatty acids, NRS = nutritional risk screening, OA = oleic acid, PA = palmitic acid, PLFA = phospholipid fatty acid, PN = parenteral nutritional, PUFA = polyunsaturated fatty acids, SA = stearic acid, SFA = saturated fatty acids.

Keywords: erythrocyte membrane phospholipid, fatty acid composition, gastrointestinal tumor, octadeca-carbon fatty acids, platelet membrane phospholipid

1. Introduction

Gastrointestinal (GI) cancers represent a number of malignancies affecting multiple organs of the digestive tract, including esophagus, gall bladder, liver, pancreas, stomach, small intestine,

large intestine, colon, rectum, and anus. Such tumors result in a large number of cancer-related mortalities annually worldwide. In particular, gastric cancer (GC) and colorectal cancer (CRC) are the second and fourth leading cause of cancer-related deaths, respectively.^[1]

Although a high-fat diet has long been associated with an increased risk of developing certain types of tumors, morbidity does not show any relation with gross dietary fat intake, as assessed by epidemiological investigations. However, a close association between the intake of different types of FA and tumorigenesis has been observed.^[2-6] Studies analyzing the relationship between FA and GI tumors have shown that high content of OA, α -linolenic acid (ALA), and dihomogamma-linolenic acid (DHLA) in the plasma may increase the risk of GC.^[7] Furthermore, DHA found in erythrocyte membranes has shown a negative correlation with GC risk.^[8] In contrast, DHA in the plasma has shown positive correlation with tumorigenesis and development of CRC,^[9] while ALA found in subcutaneous adipose tissues has displayed a negative correlation with CRC risk.^[10] In addition, tumor tissues display decreased LA and increased DHLA levels, compared with surrounding normal tissues.^[11] Furthermore, some additional studies have reported that administration of PUFAs (polyunsaturated fatty acids) and n-3 PUFA prevented the occurrence of gastric and CRC, while SFA and n-6 PUFA promoted development of such

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malignancies.^[12–15] However, such findings are not consistent across all studies^[16–20] and thus emphasize the need for further exploration of the possible link between fatty acids (FAs) and GI tumors.

Therefore, in the present study, we have examined the differences in the FA composition of erythrocyte and platelet membranes from GI tumor patients and healthy controls, in order to identify any correlation between FAs and the presence of GI tumors. Overall, this study may aid in the diagnosis and therapy of GI tumor patients.

2. Methods

2.1. Research subjects

The study was approved by the Ethics committee of Chinese PLA General Hospital and Peking Union Medical College Hospital. All procedures involving human participants were in accordance with the ethical standards of the institutional and national research committee and 1964 Helsinki declaration with all amendments. The informed consent forms were signed by all the subjects. Between June 2013 to March 2014, we recruited 50 GI tumor patients (GC, 37; CRC, 13) undergoing elective ablation of stomach, small intestine, and colorectal carcinomas. The following inclusion criteria was used: age, 18 to 75 years; body weight, 45 to 75 kg; nutritional risk screening (NRS) 2002 score of ≥ 3 ; hemoglobin (Hb) level of ≥ 80 g/L; alanine aminotransferase, total bilirubin, direct bilirubin levels ≤ 1.5 times of upper limits, normal creatinine level; no metabolic, infectious or psychiatric diseases such as pyrexia, hyperthyroidism, hypothyroidism; no chemoradiotherapy or parenteral nutrition support 3 weeks before surgery. Subjects falling into any of the following criteria were excluded: intraoperative hemorrhage >1000 mL; intraoperative or postoperative transfusion of blood or blood products; patients with serious organ function impairment such as congestive heart failure, symptomatic coronary heart disease, or arrhythmia not responding to drug treatment; patients with a history of serious cerebrovascular disease; participants in another research trial carried out concurrently; patients considered not suitable for the study by researchers; and pregnant or lactating women. In addition, 33 healthy volunteers, recruited between November 2014 and July 2015, were also included in the study as a control group. The inclusion criteria for healthy volunteers was as follows: no history of tumors or metabolic disorders; no serious diseases or any measurable clinical manifestations; no allergies or surgical history; normal results in a physical examination including vital signs, electrocardiogram, blood test, hepatic and renal functions as well as serum lipid levels; and having a normal diet and sleep pattern. The characteristics of patients and healthy control subjects are summarized in Table 1.

2.2. Blood collection, processing, and measurement

The blood (5 mL) was drawn from a vein after overnight fasting, and stored at 4°C to be processed within 4 hours. FAs extraction and detection were performed using previously described methods.^[21,22] Briefly, the collected samples were first centrifuged at room temperature, and the supernatants were collected. Next, an equal volume of HEP buffer (140 mM NaCl, 2.7 mM KCl, 3.8 mM HEPES, 5 mM ethylenedis (oxyethylenitrilo) tetraacetic acid, pH 7.4) was added to the supernatant along with 1 μ M prostaglandin E1. After subsequent centrifugation and washing, platelets were collected. The bottom layer of blood samples was processed using a human peripheral blood leukocyte

Table 1

Characteristics of healthy controls and GI tumor patients.

	GI tumor patients (n = 50)	Control (n = 33)	P
Age, y	57.4 (33–75)	36.0 (19–58)	<.001
Sex, M/F (%)	23/27 (54.0%)	21/12 (36.4%)	.11
BMI, kg/m ²	23.4 \pm 2.4 (16.5–28.8)	22.3 \pm 1.9 (18.8–26.5)	.038
Smoking status, n (%)			.83
Never-smoker	46 (92.0)	29 (87.9)	
Casual smokers	1 (2.0)	1 (3.0)	
Regular smokers	3 (6.0)	3 (9.1)	
Alcohol intake, n (%)			.20
Nondrinker	42 (84.0)	23 (69.7)	
Casual drinkers	4 (8.0)	7 (21.2)	
Regular drinkers	4 (8.0)	3 (9.1)	
Physical activity, n (%)			.58
Low intensity	9 (18.0)	8 (24.2)	
Medium intensity	27 (54.0)	14 (42.4)	
High intensity	14 (28.0)	11 (33.3)	
Educational status, n (%)			.054
Primary school or below	21 (42.0)	6 (18.2)	
Middle school	15 (30.0)	11 (33.3)	
University diploma or above	14 (28.0)	16 (48.5)	
Family tumor history, n (%)			.51
Family CRC history	4 (8.0)	1 (3.0)	
Family GC history	5 (10.0)	2 (6.1)	
Anatomic site distribution, n (%)			
Stomach	37 (74.0)	—	
Intestine	13 (26.0)	—	

BMI = body mass index, CRC = colorectal cancer, GC = gastric cancer.

separation kit and erythrocyte separation kit to purify leukocytes and erythrocytes, which were then preserved at -80°C . Membrane phospholipids from the blood cells were extracted using a chloroform-methanol (3:1, vol/vol) solution. PLFAs were then methyl esterified using methylbenzene and methyl alcohol agents. FAs were separated by GC-MS using a Trace 1300 gas chromatograph (Thermo Fisher Scientific, Waltham, MA) with a SP-2560 capillary column (100 m \times 0.25 mm \times 0.2 μ m) as the chromatographic column. A Trace ISQ mass spectrometer was used for FA identification. The gaseous phase temperature programming procedures were used as follows: Helium as the carrier gas, and the initial temperature was set at 140°C for 5 minutes, with the temperature increasing to 150°C at 10°C/min rate, and then to 190°C at 3°C/min rate, and then to 220°C for 1 minute at 2°C/min rate, and then to 240°C for 15 minutes at 4°C/min rate. Finally, the injection temperature was set to 250°C.

2.3. Statistical analysis

FA composition was represented as a percentage, and the values of total SFAs (saturated FA) including C8:0, C10:0, C12:0, C14:0, C16:0, C18:0, C20:0, C22:0, C24:0, MUFAs (monounsaturated FAs) including C14:1(n-5), C16:1(n-7), C18:1(n-9), C20:1(n-9), C22:1(n-9), C24:1(n-9); n-6 PUFAs including C18:2(n-6), C18:3(n-6), C20:2(n-6), C20:3(n-6), C20:4(n-6), C22:2(n-6), n-3 PUFAs including C18:3(n-3), C20:3(n-3), C20:5(n-3), C22:5(n-3), C22:6(n-3), along with the ratio of n-6 to n-3 PUFA (n-6/n-3), were calculated. The differences in the FA composition between the blood cell membranes of tumor patients and healthy volunteers were analyzed using a regression model, where FA percentage was the dependent variable, while gender, age, body

mass index (BMI), and tumor were independent variables. In addition, multiple-factor analysis was carried out on each of the variable to determine its influence on FA composition. All statistical analyses were performed using SAS9.1 statistical software (SAS Institute Inc., Cary, NC) and a *P* value of <.05 represented statistically significant difference.

3. Results

3.1. Comparison of GI tumor patient characteristics with healthy controls

GC and CRC patients displayed a high morbidity and mortality and share similar potential tumor markers and therapeutic targets. In western countries, the synchronization incidence of both tumor types is as high as 35.8%.^[2,3] However, previous studies have shown some differences in FA composition between GC and CRC patients, and this could be attributed to the comparison of different sample types including racial differences and varying detection methods. However, in this study, we have undertaken for the first time, the direct comparison of FAs composition in erythrocyte and platelet membranes of GC and CRC patients using GC-MS in Chinese Han population. Our results indicated that there was no significant difference in the FA

composition of erythrocyte membranes from tumor patients. Furthermore, there was also no difference in platelets composition (Supplementary Table 1, <http://links.lww.com/MD/B735>). Thus, we combined GC and CRC patients into 1 group labeled as GI tumor group for further analysis. The basic characteristics of GI tumor patients and healthy controls are summarized in Table 1. Upon analysis, we observed significant differences in age and BMI among both groups. Patients in the tumor group were older in age and had a higher BMI. These 2 characteristics were analyzed by multiple-factor analysis, comparing the FA composition of erythrocyte and platelet membranes in GI tumor patients and healthy controls.

3.2. Comparison of the fatty acid profiles of erythrocyte membranes in healthy controls and GI tumor patients

The levels of FAs in erythrocyte membranes of 50 patients and 33 healthy controls are summarized in Table 2. Both groups displayed high quantities of SFA, followed by PUFA and MUFA. In addition, individual FAs such as C16:0, stearic acid (SA), oleic acid (OA), linoleic acid (LA), arachidonic acid (AA), and docosahexaenoic acid (DHA) were relatively abundant. Multiple-factor analysis indicated that differences due to grouping alone (tumor group vs control group) were linked to ten FAs,

Table 2
Fatty acid composition (mean percentage) of erythrocyte and platelet membranes of GI tumor patients and healthy controls.

FA name	Erythrocyte		Platelets	
	Control n=33	GI tumor patients n=50	Control n=33	GI tumor patients n=50
C8:0	0.02±0.04	0.06±0.04	0.08±0.15	0.07±0.05
C10:0	0.05±0.02	0.04±0.05	0.19±0.13	0.07±0.08
C12:0	0.05±0.02	0.07±0.02	0.18±0.11	0.10±0.04
C14:0	0.18±0.03	0.28±0.09	0.28±0.12	0.32±0.15
C16:0	27.26±4.36	28.32±2.25	21.85±4.25	23.22±3.95
C18:0	14.45±2.79	18.45±3.28	24.22±4.03	25.57±4.10
C20:0	0.17±0.04	0.28±0.30	1.11±0.18	1.08±0.41
C22:0	0.53±0.13	0.56±0.12	1.08±0.21	0.96±0.26
C24:0	2.48±0.49	1.75±0.41	1.05±0.20	0.57±0.18
C14:1 (n=5)	0.02±0.05	0.02±0.06	0.04±0.06	0.02±0.05
C16:1 (n=7)	0.18±0.06	0.17±0.05	0.22±0.14	0.21±0.07
C18:1 (n=9)	13.19±1.61	11.50±1.08	12.79±1.83	11.11±1.70
C20:1 (n=9)	0.27±0.12	0.21±0.04	0.70±0.29	0.60±0.16
C22:1 (n=9)	0.38±0.15	0.58±0.24	1.19±0.78	0.88±0.33
C24:1 (n=9)	1.88±0.81	1.64±0.35	0.64±0.25	0.59±0.16
C18:2 (n=6)	15.35±2.35	11.60±1.86	7.64±1.69	6.54±1.30
C18:3 (n=6)	0.03±0.01	0.04±0.02	0.05±0.08	0.05±0.02
C20:2 (n=6)	0.37±0.09	0.33±0.07	0.47±0.13	0.46±0.12
C20:3 (n=6)	1.36±0.44	1.33±0.36	1.08±0.40	1.11±0.32
C20:4 (n=6)	15.13±2.32	14.81±2.88	21.70±5.12	23.24±4.37
C22:2 (n=6)	0.08±0.02	0.10±0.06	0.06±0.03	0.07±0.08
C18:3 (n=3)	0.14±0.06	0.12±0.07	0.12±0.09	0.07±0.04
C20:3 (n=3)	0.04±0.01	0.03±0.01	0.07±0.05	0.03±0.01
C20:5 (n=3)	0.31±0.18	0.44±0.34	0.19±0.13	0.24±0.17
C22:5 (n=3)	1.60±0.40	1.75±0.58	1.41±0.64	1.08±0.31
C22:6 (n=3)	4.39±1.11	5.44±1.66	1.42±0.58	1.66±0.55
n-6 PUFA	32.32±4.27	28.21±3.36	3.21±0.98	3.08±0.85
n-3 PUFA	6.48±1.62	7.78±2.24	31.00±6.66	31.47±5.58
n-6/n-3	5.24±1.16	3.88±1.02	10.37±3.21	10.79±2.83
SFA	45.19±6.62	49.81±4.67	50.04±7.96	51.96±7.48
MUFA	15.91±2.32	14.12±1.26	15.58±2.26	13.40±1.82
PUFA	38.79±5.33	36.00±4.02	34.21±6.93	34.55±6.00
MUFA/SFA	0.36±0.09	0.29±0.05	0.32±0.08	0.27±0.07
PUFA/SFA	0.89±0.20	0.74±0.16	0.72±0.24	0.69±0.21

FA = fatty acids, GI = gastrointestinal, MUFA = monounsaturated fatty acids, PUFA = polyunsaturated fatty acids, SFA = saturated fatty acids.

Table 3
Multiple-factor analysis of erythrocyte and platelet of GI tumor patients and healthy controls.

Independent variable	Erythrocyte				Platelet			
	FA name	Regression coefficient	Standard error	P	FA name	Regression coefficient	Standard error	P
Grouping	C8:0	-0.0288	0.0137	.0382	C10:0	0.1520	0.0306	.0000
	C12:0	-0.0258	0.0073	.0007	C12:0	0.0849	0.0234	.0005
	C14:0	-0.0875	0.0240	.0005	C24:0	0.4356	0.0612	.0000
	C18:0	-2.8755	1.0069	.0055	C18:1 (n-9)	1.8846	0.5765	.0016
	C24:0	0.6665	0.1453	.0000	C20:4 (n-6)	-3.7467	1.4983	.0145
	C18:1 (n-9)	1.7561	0.4321	.0001	C20:3 (n-3)	0.0388	0.0107	.0005
	C22:1 (n-9)	-0.2342	0.0683	.0010	C22:6 (n-3)	-0.4620	0.1821	.0132
	C18:2 (n-6)	2.4688	0.6453	.0003	MUFA	2.3738	0.6648	.0006
	C22:6 (n-3)	-1.0163	0.4784	.0369				
	n-3 PUFA	-0.1425	0.6577	.0338				
	MUFA	1.8491	0.5742	.0019				
Sex	C20:2 (n-6)	-0.0384	0.0162	.0199	C22:1 (n-9)	-0.2729	0.1164	.0217
	C20:3 (n-6)	-0.1937	0.0890	.0327	C18:3 (n-3)	-0.0332	0.0144	.0236
	C20:3 (n-3)	-0.0096	0.0028	.0009	C22:5 (n-3)	-0.2309	0.1039	.0285
	C22:5 (n-3)	-0.2726	0.1150	.0202	n-3 PUFA	-0.5498	0.1964	.0065
Age				n-6/n-3	0.1346	0.0651	.0420	
	C18:2 (n-6)	-0.0478	0.0205	.0221	C18:2 (n-6)	-0.0343	0.0146	.0205
	n-6 PUFA	-0.0789	0.0377	.0398	C20:4 (n-6)	-0.1087	0.0475	.0249
				n-6 PUFA	-0.1485	0.0607	.0167	
BMI				PUFA	0.1592	0.0641	.0151	
	C16:0	0.3864	0.1607	.0186	C14:1 (n-5)	0.0054	0.0027	.0203
	C14:1 (n-5)	0.0059	0.0026	.0268	C24:1 (n-9)	-0.0212	0.0100	.0373
	C20:2 (n-6)	-0.0087	0.0036	.0182				
	SFA	0.5975	0.2741	.0323				
	PUFA	-0.4959	0.2255	.0309				

BMI = body mass index, FA = fatty acids, MUFA = monounsaturated fatty acids, PUFA = polyunsaturated fatty acids, SFA = saturated fatty acids.

including C8:0, C12:0, C14:0, SA, C24:0, OA, C22:1, DHA, MUFA, and n-3 PUFA (Table 3). Age and grouping both affected LA content; however, grouping had a more significant effect than age, as analyzed by regression coefficient (2.4688 vs -0.0478) analysis. This indicated that differences in LA levels may be due to grouping alone. Overall, we observed significant differences in 11 FAs between tumor and healthy control groups, with the tumor group demonstrating higher levels of C8:0, C12:0, C14:0, SA, C22:1, DHA, and n-3 PUFA, and lower levels of C24:0, OA, LA, and MUFA. Moreover, on the basis of the proportion of FAs in erythrocytes and the differences between 2 groups, changes in octadeca-carbon FAs levels were most obvious (Fig. 1A).

3.3. Comparison of the fatty acid profiles of platelet membranes in healthy controls and GI tumor patients

The composition of FAs in platelet membranes has been summarized in Table 2. SFA was abundantly found in platelets, followed by PUFA and MUFA. Individual FAs involving C16:0, SA, OA, LA, and AA were also abundant. In addition, grouping demonstrated differences in 7 FAs, including C10:0, C12:0, C24:0, OA, C20:3 (n-3), DHA, and MUFA, as assessed by multiple-factor analysis (Table 3). Furthermore, the difference in AA content maybe due to grouping alone, as the effect of grouping on AA was much larger than age (correlation coefficient, -3.7467 vs -0.1087). In summary, GI tumor

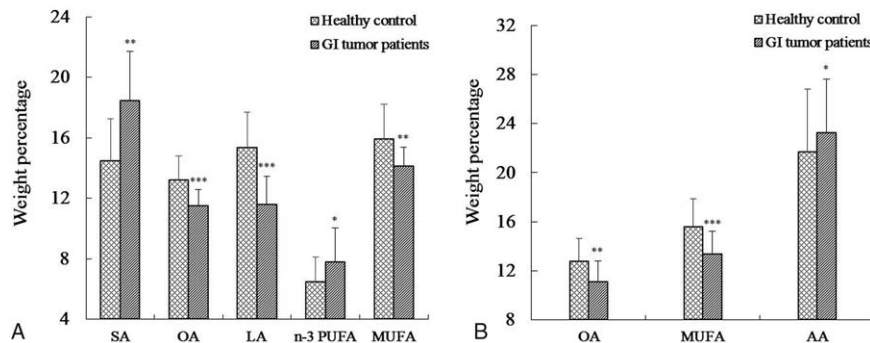


Figure 1. Fatty acid composition of erythrocytes (A) and platelets (B) in healthy controls and GI tumor patients. GI = gastrointestinal, LA = linoleic acid, MUFA = monounsaturated fatty acids, OA = oleic acid, PUFA = polyunsaturated fatty acids, SFA = saturated fatty acids. *P < .05; **P < .01; ***P < .001, versus healthy controls.

patients showed lower levels of C10:0, C12:0, C24:0, OA, C20:3 (n-3), MUFA, and higher levels of AA and DHA FAs in the platelet membranes, than healthy controls (Fig. 1B).

4. Discussion

Our results indicated the differences in the FAs composition of erythrocyte membranes from GI tumor patients and healthy controls, especially 18-carbon FAs (an increase in SA and decreases in OA and LA contents). These differences were consistent with previously published studies analyzing other solid tumors, especially a reduction in LA levels. Compared with healthy individuals, erythrocyte membranes of lung cancer patients demonstrated lower levels of LA, along with plasma from liver and pancreatic cancer patients displaying similar profiles. In addition, liver, breast, and colon cancer tissues also displayed significantly lower levels of LA in comparison to surrounding normal tissues. In contrast, the plasma from hematological tumor patients, such as multiple myeloma, displayed markedly elevated levels of LA.^[24-27] Moreover, our study showed a significant increase in the levels of SA FAs, while a decrease in OA levels in erythrocyte membranes of GI tumor patients. Surprisingly, our findings were somewhat contradictory to the results observed in various other cancers. For example, non-small cell lung cancer patients showed higher levels of OA and SA in the erythrocyte membranes than healthy subjects. In contrast, myeloma patients displayed lower levels of OA and SA in the erythrocyte membranes.^[28] Further, levels of SA FA significantly decreased, while OA levels increased in the erythrocyte membranes of patients with gallbladder primary carcinoma. Thus, we can say that typically there is always a change in the levels of octadeca-carbon FAs in multiple tumors, but GI tumors showed an opposing trend.

In addition, a variety of studies involving multiple tumor types have consistently demonstrated higher levels of SFA in erythrocyte membranes.^[8,24,28-30] Indeed, there is a positive correlation between CRC risk and SFA levels. However, PUFA levels show a negative correlation with such cancers.^[28] Consistent with these reports, our study also showed a significant increase in SFA levels and a remarkable decrease in PUFA levels in erythrocyte membranes from GI tumor patients. The decrease in PUFA content suggests that lipid peroxidation of PUFA may occur in erythrocyte membranes, and can trigger cellular fluidity and reduce the permeability of cell membranes.

PUFA consists of n-3 PUFA and n-6 PUFA, and both of these compete for the same metabolic enzymes, but have different physiological functions. The detailed examination of previously published studies regarding n-3 PUFA in GI tumor patients indicated inconsistent results. For example, some studies have reported increased levels of n-3 PUFA in GC and CRC patients, and indicated a positive correlation with tumorigenesis.^[7,8] In contrast, a select few studies have demonstrated a negative correlation of n-3 PUFA with tumor development, with decreased levels in tumor patients.^[8,31] Similar results were also observed with n-6 PUFA in GI tumors, with levels decreasing in erythrocyte membranes.^[8,28,29] However, we observed a significant increase in n-3 PUFA levels and a decrease in n-6 PUFA. To explain these results, we hypothesized that excessive dietary intake of n-6 PUFA may increase incidence of tumor, whereas n-3 PUFA could have an opposite effect on tumorigenesis. Patients with tumor increasingly utilize n-6 PUFA, and thus, its levels decrease, while n-3 PUFA levels remain high in parallel.

Furthermore, despite our observation of lower levels of LA (a precursor of FA AA) in the erythrocyte membranes of tumor patients in comparison to the control group, we did not observe any overall differences between AA levels of patients and healthy controls. The tentative explanation may be that lower level of LA upregulate $\Delta 6D$ enzymatic activity, which in turn increased the in vivo synthesis of AA. Moreover, with respect to the changes in AA content, previous studies to date have not drawn any consensus in patients with tumors.^[24,26,29,31,32]

Moreover, our study also investigated for the first time the FA profiles of platelets in GI tumor patients. We observed higher levels of AA FA in the platelets of GI tumor patients, than healthy controls. In parallel, we also observed changes in OA, DHA, and MUFA levels in platelets. The previous studies have demonstrated that lipid changes in platelets may play a role in platelet activation and increase thrombotic risk. Further, changes in AA and other FA profiles may be associated with platelet function in GI tumor progression and warrant further investigation.

Our results also indicated that the majority of FA profiles were significantly different between erythrocytes and platelets, except for some such as C8:0, C10:0, C14:1 (n-5), C22:2 (n-6), C20:3 (n-3); however, their content levels were extremely low. The total content of SFA and PUFA was approximately the same, with only a modest difference in MUFA levels.

On the basis of our results, we concluded that there were significant differences in the FA composition of both erythrocyte and platelet membranes between GI tumor patients and healthy controls. The octadeca-carbon FAs (SA, OA, and LA) in erythrocyte membranes may serve as indicators for GI tumor detection. However, further studies exploring the effects of n-3 PUFA and AA on GI tumors are warranted. In particular, our future studies would be designed to compare the FA composition of tumor and nontumor cells, with an aim to specifically explore the role of FAs from octadeca-carbon species in GI tumorigenesis.

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