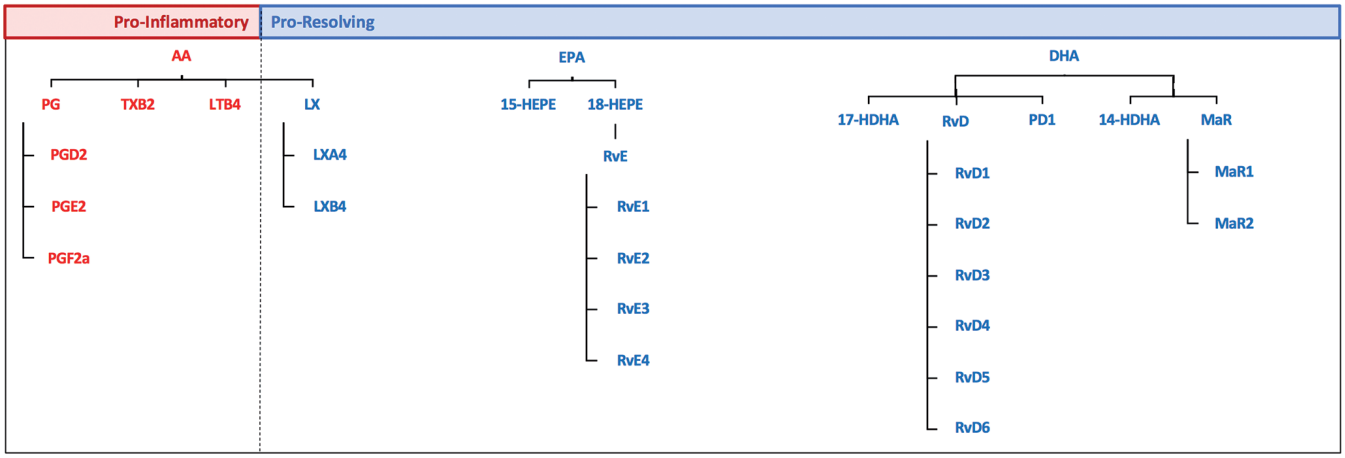
Supplementary Table S1. Baseline characteristics of SLE patients and age, sex, and race matched controls.

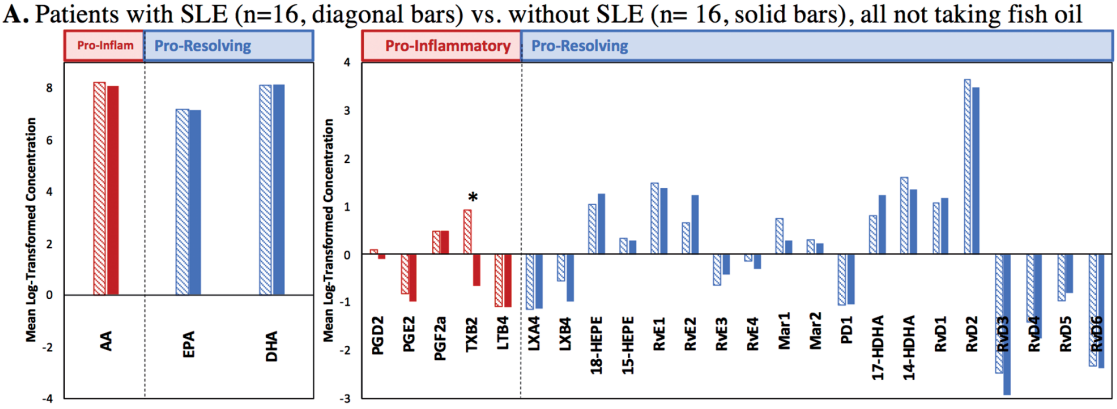
Characteristic	Not taking Fish Oil		Taking Fish Oil	
	SLE (n=16)	Matched controls (n=16)	SLE (n=16)	Matched controls (n=16)
Age, mean (SD)	43.97 (10.81)	43.30 (10.27)	43.26 (10.59)	43.41 (10.28)
Sex, female, %	16 (100.0)	16 (100.0)	16 (100.0)	16 (100.0)
Race, %				
White	13 (81.25)	14 (87.50)	14 (87.50)	15 (93.75)
Non-white	2 (12.50)	2 (12.50)	1 (6.25)	1 (6.25)
Missing	1 (6.25)	0 (0.00)	1 (6.25)	0 (0.00)
Smoking, %				
Current	2 (12.50)	0 (0.00)	0 (0.00)	2 (13.33)
Past	4 (25.00)	4 (25.00)	2 (12.50)	3 (18.75)
Never	10 (62.50)	10 (62.50)	14 (87.50)	11 (73.33)
Unknown	0 (0.00)	2 (12.50)	0 (0.00)	0 (0.00)
BMI, kg/m^2, mean (SD)	28.36 (7.38)	26.12 (7.40)	24.78 (3.75)	27.46 (5.64)
Statin use[1], %	2 (12.50)	0 (0.00)	0 (0.00)	0 (0.00)
Current prednisone use, %	3 (18.75)	0 (0.00)	3 (18.75)	1 (6.25)
HCQ use, %	12 (75.00)	0 (0.00)	15 (93.75)	0 (0.00)
Other immunosuppressant medication use[2], %	2 (12.50)	2 (12.50)	1 (6.25)	0 (0.00)
SLEDAI-2K, mean (SD)	1.58 (3.26)		3.08 (3.26)	
Elevated CRP or ESR, %	4 (28.57)		7 (46.67)	
Positive anti-dsDNA, %	2 (13.33)		7 (43.75)	
Hypocomplementaemia, %	4 (30.77)		6 (42.86)	
Lupus nephritis, %	1 (6.25)		4 (25.00)	

BMI: Body Mass Index; HCQ: hydroxychloroquine; SD: standard deviation; SLE: systemic lupus erythematosus
[1]Pravastatin, atorvastatin, lovastatin, rosuvastatin, simvastatin
[2]Methotrexate, leflunomide, sulfasalazine, cyclophosphamide, cyclosporine, mycophenolate mofetil, etanercept, adalimumab, rituximab, infliximab, belimumab, tofacitinib, tocilizumab.

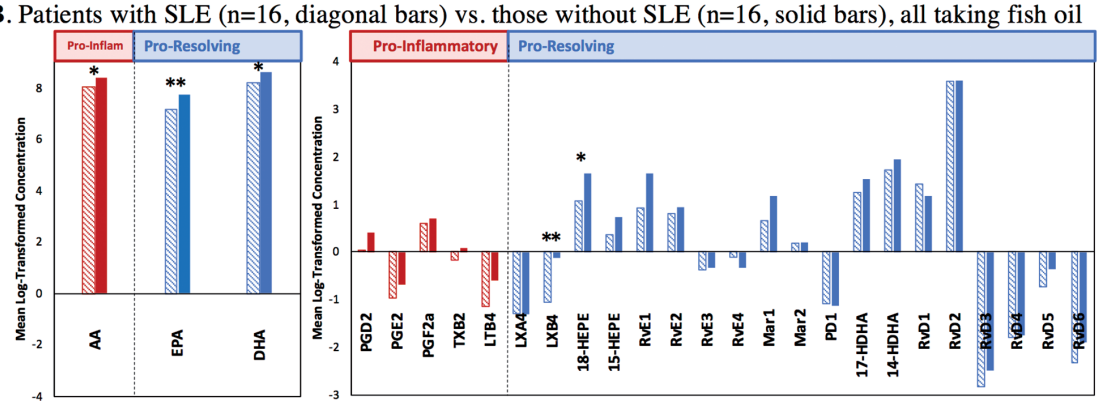


Supplementary Fig. S1. Proinflammatory (red) and pro-resolving (blue) lipid mediators assayed in this study**.** Liquid chromatography (LC)-tandem mass spectrometry (MS) was performed to quantify 27 targeted LM, their precursors and individual pathway markers. Plasma samples were defrosted from -80°C on ice. For purpose of quantification and recovery of the lipid mediators, deuterium labeled internal standards including d8-5S-HETE, d5-Resolvin D2 (RvD2), d5-lipoxin A (LXA_4), d4-Leukotriene B_4 (LTB_4), and d4-Prostaglandin E_2 (PGE_2) (500 pg each, Cayman Chemical) in 1 mL of methanol were added to each sample for determining extraction recoveries. Samples were then held in methanol at – 20°C for 45 min to allow protein precipitation. After centrifugation at 1000 g for 10 min at 4 °C, supernatants were collected and were taken to solid phase extraction using an automated LM extractor (Extrahera, Biotage) according to optimized methods. Briefly, samples were acidified to an apparent pH 3.5 and loaded onto Isolute SPE C18 columns (100 mg, 3 mL, Biotage). C18 columns were rapidly neutralized with double-distilled water and washed with hexane. Lipid mediators were eluted with methyl formate. These lipid mediators-containing fractions were then brought to dryness under a gentle stream of nitrogen gas using the automated evaporation system (TurboVap LV, Biotage) and immediately resuspended in methanol-water mixture (50:50, v/v). The LC-MS/MS system consisted of a QTRAP 5500 (AB Sciex) equipped with a Shimadzu LC-20AD HPLC that was carried out at the University of Toronto (Toronto, Canada) equipped with a poroshell 120 EC-C18 column (100 mm x 4.6 mm x 2.7 μm; Agilent Technologies) and kept in a column oven regulated at 50 °C. Lipid mediators were eluted at a flow rate of 0.5 mL/min with a gradient of methanol/water/acetic acid that increased from 55:45:0.01 (v/v/v) to 98:2:0.01 (v/v/v). Lipid mediators were detected (limits of detection ≈ 0.1 pg) using targeted multiple reaction monitoring and enhanced product ion in a negative mode (University of Toronto, Toronto, Canada). Criteria used to identify and quantify LMs included MS-MS matching to at least six diagnostic ion fragments per molecule as described in English *et al*. (30), matching retention time to authentic and synthetic standards, and individual linear calibration curves for each LM ($r^2 > 0.98$). Samples were run > twice and average results reported. Coefficients of variance (CV) were <20% for low and <15% for moderate abundance lipids. LC-MS/MS acquisition was performed with the Analyst software 1.6.2 (AB Sciex) and quantification was performed using MultiQuant software 3.0.2 (AB Sciex).
AA: arachidonic acid; EPA: eicosapentaenoic acid; DHA: docosahexaenoic acid; LTB_4: leukotriene B_4; LXA4: lipoxin A_4; LXB_4: lipoxin B_4; MaR1-2: maresin 1-2; PGD_2: prostaglandin D_2; PGE_2: prostaglandin E_2; PGF2a: prostaglandin F2 alpha; TXB_2: thromboxane B_2; PD1: protectin D1; RvD1-6: resolvin D-6; RvE1-4: resolvin E1-4; 14-HDHA: 14-hydroxy-docosahexaenoic acid; 15-HEPE: 15-hydroxyeicosapentaenoic acid; 17-HDHA: 17-hydroxy-docosahexaenoic acid; 18-HEPE: 18-hydroxyeicosapentaenoic acid.

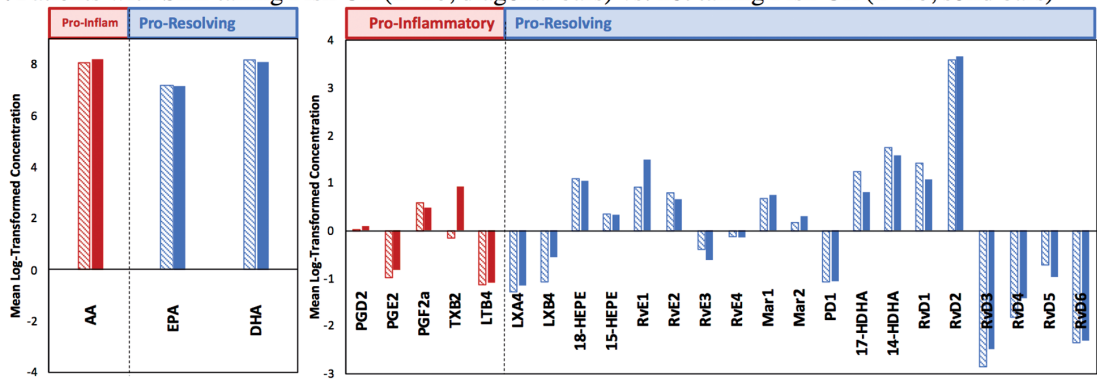
A. Patients with SLE (n=16, diagonal bars) vs. without SLE (n= 16, solid bars), all not taking fish oil



B. Patients with SLE (n=16, diagonal bars) vs. those without SLE (n=16, solid bars), all taking fish oil



C. Patients with SLE taking Fish Oil (n=16, diagonal bars) vs. not taking Fish Oil (n=16, solid bars)



D. Patients without SLE taking (n=16, diagonal bars) vs. not taking fish oil (n=16, solid bars)

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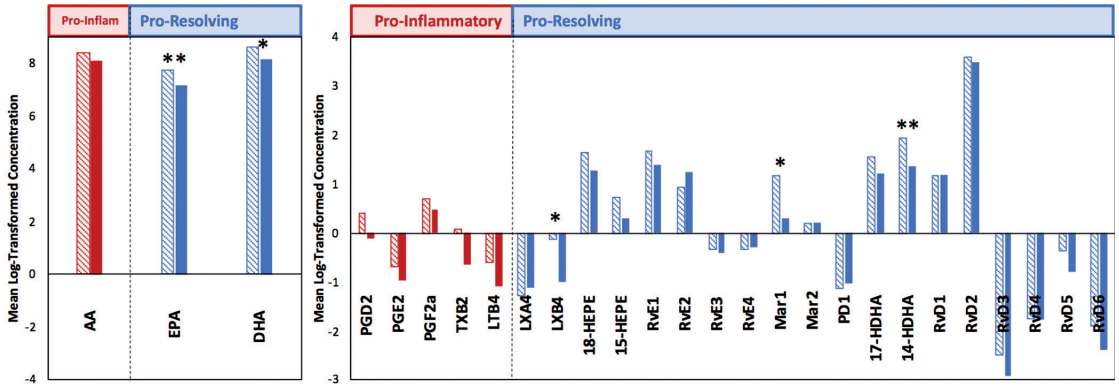

Supplementary Fig. S2A-D. Bivariable analyses of 27 individual eicosanoid lipid mediators assayed by liquid chromatography- tandem mass spectrometry in the blood of 64 matched patients with and without SLE, taking and not taking fish oil supplements.
Mean log-transformed concentrations (log pg/ml). *p<0.05, **p<0.01.
AA, arachidonic acid; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; LTB4, Leukotriene B4; LXA4, Lipoxin A4; LXB4, Lipoxin B4; MaR1-2, maresin 1-2; PGD2, Prostaglandin D2; PGE2, Prostaglandin E2; PGF2a, Prostaglandin F2 alpha; TXB2, Thromboxane B2; PD1, protectin D1; RvD1-6, resolvin D-6; RvE1-4, resolvin E1-4; 14-HDHA, 14-hydroxy-docosahexaenoic acid; 15-HEPE, 15-hydroxyeicosapentaenoic acid; 17-HDHA, 17-hydroxy-docosahexaenoic acid; 18-HEPE, 18-hydroxyeicosapentaenoic acid.