

Supplementary files

A) Isolation of Peripheral Blood Mononuclear Cells

The cells were isolated from whole blood taken from patients and the control group after receiving informed consent and ethics clearance (M170440). After separating the plasma, the blood was diluted in a 1:1 ratio using Roswell Park Memorial Institute (RPMI) medium without foetal bovine serum (Sigma Aldrich, St Louis, MI, USA). The diluted blood was layered onto ficoll at a 2:1 ratio. This was followed by centrifugation at $1734 \times g$, for 30 min, RT with the brakes off. The PBMCs were transferred to a new 50 mL falcon tube and washed ($932 \times g$, 10 min RT) with 30 mL of RPMI (Sigma Aldrich). Red blood cells were lysed using ammonium--chloride--potassium, or ACK (150 mM NH_4Cl , 10 mM KHCO_3 , 0.1 mM EDTA, pH 7.2) for 5 min before washing with 15 mL of RPMI ($277 \times g$, RT, 10 minutes). The cells were resuspended and maintained in RPMI supplemented with 10% FBS, gentamicin, and antibiotic/antimycotic solution (complete RPMI). This was followed by stimulation for 2 h with $2 \mu\text{g/mL}$ of phytohemagglutinin-protein (PHA-P) also known as lectin from *Phaseolus vulgaris* (Sigma Aldrich, St Louis, MI, USA) before treatment with compounds.

B) Effect of compounds on IL-6

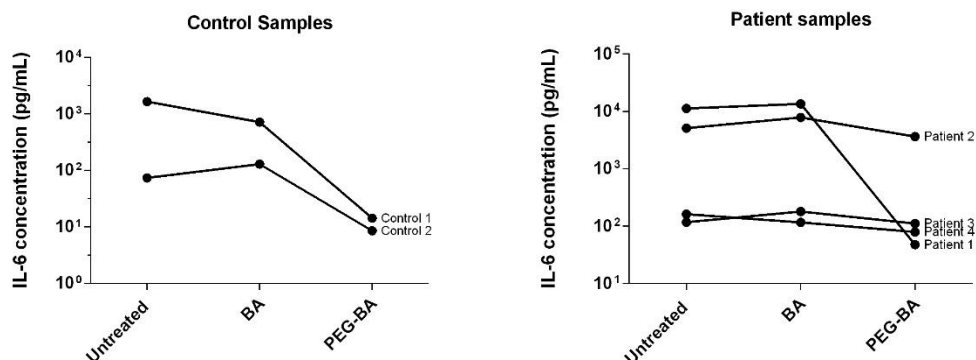


Figure S1: Line graph showing the effect of BA and PEG-BA on IL-6. A notable inhibition of IL-6 by PEG-BA is shown. The y-axis scale is Log₁₀, $n = 2$ controls, $n = 4$ patients. Although there was a consistent decrease in IL-6 concentration when patient samples were treated with BA versus PEG-BA, it is evident that the responses for patient 3 and 4 were much reduced. This could be attributed to the varying degrees of inflammation occurring due to differences in cancer severity, i.e., locally advanced versus metastatic.

C) Effect of compounds on Th1Th2Th17 cytokines

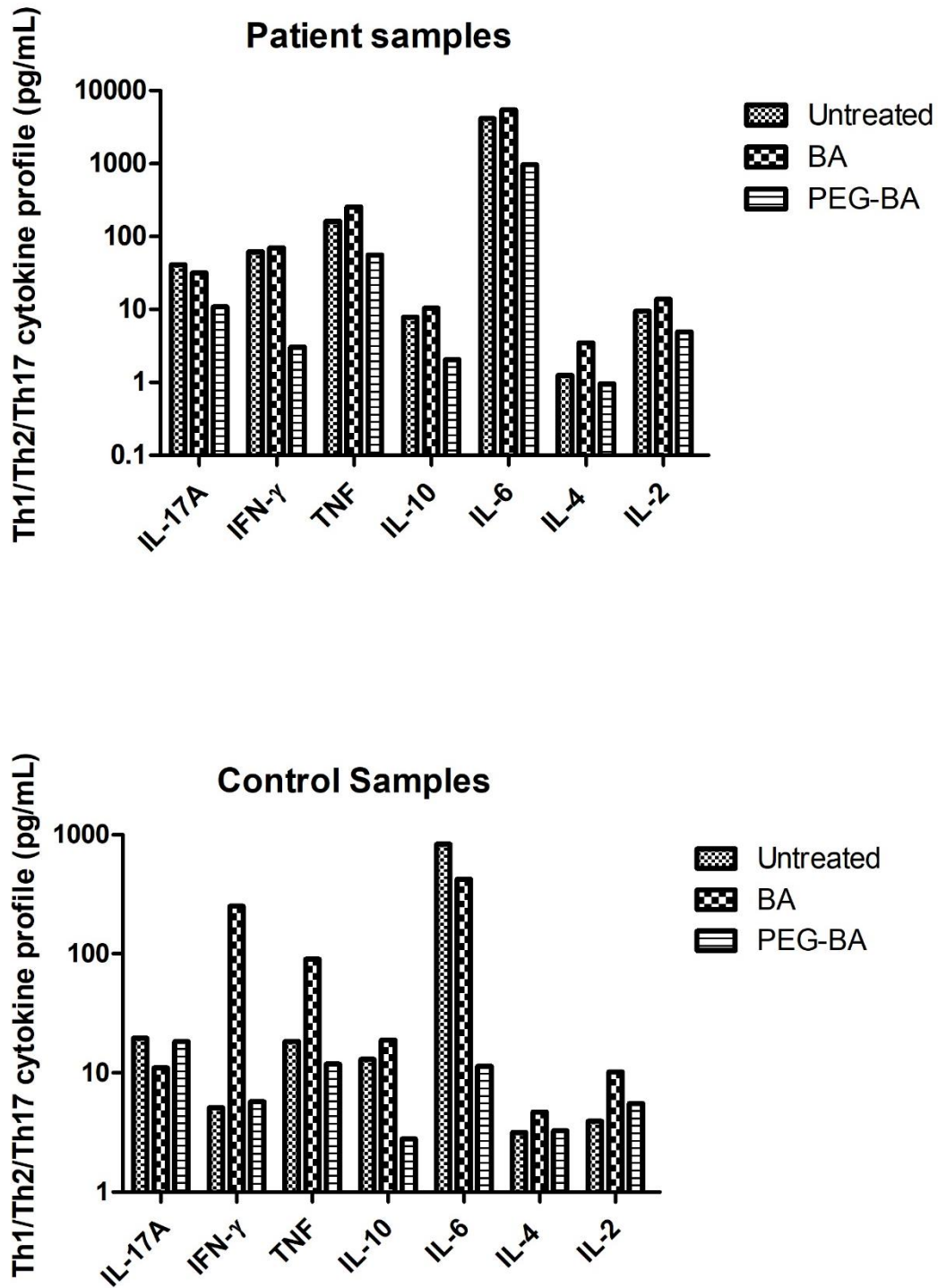


Figure S2: Effect of BA and PEG-BA on IL-17A, IFN- γ , TNF- α , IL-10, IL-6, IL-4 and IL-2. A notable increase in the inhibition of IL-17A, IFN- γ , TNF- α , IL-10 and IL-6 by PEG-BA was shown compared to that induced by BA mainly in the patient samples. The y-axis scale is Log10, $n = 2$ controls, $n = 4$ patients.

D) RT2 profiler plate layout

	1	2	3	4	5	6	7	8	9	10	11	12
A	ACSL3	ACSL4	ACSL5	ADM	ARNT	ATF4	AXIN2	BAX	BBC3	BCL2	BCL2A1	BCL2L1
B	BIRC3	BMP2	BMP4	BTG2	Ct9	CCL5	CCND1	CCND2	CDKN1A	CDKN1B	CEBPD	CPT2
C	CSF1	DAB2	EGFR	EMP1	EPO	FABP1	FAS	FCER2	FOSL1	FTH1	GADD45A	GADD45B
D	GATA3	GCLC	GCLM	GSR	HERPUD1	HES1	HES5	HEY1	HEY2	HEYL	HMOX1	ICAM1
E	ID1	IFNG	IFRD1	IRF1	JAG1	LDHA	LFNG	LRG1	MCL1	MMP7	MYC	NOTCH1
F	NGO1	OLR1	PCNA	PPARD	PITCH1	RB1	SERPINE1	SLC27A4	SLC2A1	SOC3	SORBS1	SQSTM1
G	STAT1	TNF	TNFSF10	TXN	TXNRD1	VEGFA	WISP1	WNT1	WNT2B	WNT3A	WNT5A	WNT6
H	ACTB	B2M	GAPDH	HPRT1	RPLP0	HGDC	RTC	RTC	RTC	PPC	PPC	PPC

Figure S3: The 96-plate layout of the human signal transduction RT² profiler PCR array panel with the position of the genes.

Table S1: Full names of dysregulated genes

Gene symbol	Gene name
<i>WISP1</i>	WNT1-inducible-signaling pathway protein 1
<i>TXNRD1</i>	Thioredoxin reductase 1
<i>ACTB</i>	Actin Beta
<i>GADD45B</i>	Growth arrest and DNA-damage-inducible, beta
<i>AXIN2</i>	Axin-related protein
<i>STAT1</i>	Signal transducer and activator of transcription 1
<i>ACSL4</i>	Acyl-CoA Synthetase Long Chain Family Member 4
<i>FTH1</i>	Ferritin heavy chain 1
<i>NQO1</i>	NAD(P)H dehydrogenase [quinone] 1
<i>VEGFA</i>	Vascular Endothelial Growth Factor A
<i>ADM</i>	Adrenomedullin
<i>LRG1</i>	Leucine-rich alpha-2-glycoprotein
<i>GATA3</i>	GATA binding protein 3
<i>CCND1</i>	B-cell leukemia/lymphoma 1
<i>CEBPD</i>	CCAAT Enhancer Binding Protein Delta
<i>CDKN1A</i>	Cyclin Dependent Kinase Inhibitor 1A
<i>BTG2</i>	BTG Anti-Proliferation Factor 2
<i>HES5</i>	Hes Family BHLH Transcription Factor 5
<i>WNT3A</i>	Wingless-related integration site 3 A
<i>BCL2</i>	B-cell lymphoma 2
<i>ACSL4</i>	Acyl-CoA Synthetase Long Chain Family Member 4
<i>SLC2A1</i>	Solute carrier family 2 member 1
<i>GAPDH</i>	Glyceraldehyde 3-phosphate dehydrogenase