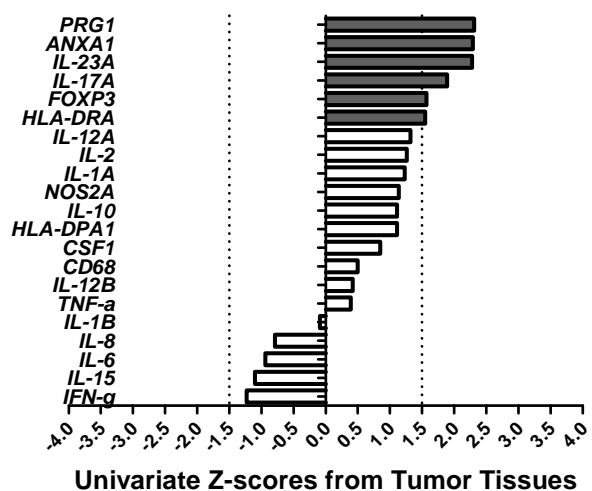
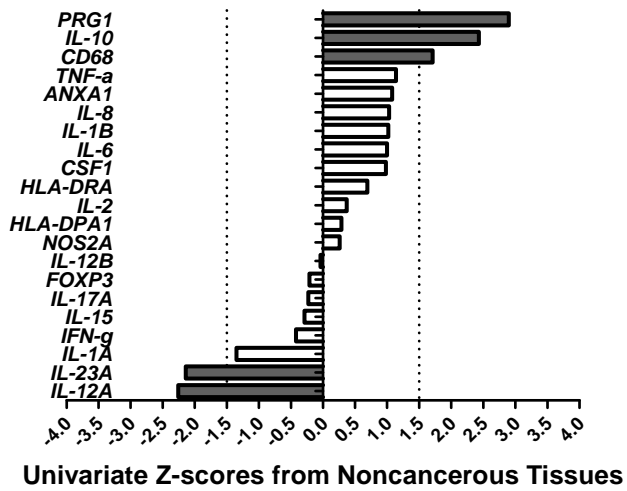


Z-scores from univariate Cox regression analysis on expression levels of inflammatory genes in Hong Kong Training Cohort.



Supplemental figure 1: Univariate analysis of the expression of 23 inflammatory genes in noncancerous or tumor tissue with cancer-specific mortality. The genes are ranked based on their predictive power (univariate Z-score). Dashed lines indicate $|Z\text{-score}|=1.5$. Black bars represent $|Z\text{-score}|>1.5$. This criterion was used for selecting genes to include in the multivariate Cox regression model used to calculate IRS.

Supplemental Table 1: Inflammatory Genes that are differentially expressed in tumors vs. non-cancerous tissue, by cohort or colon adenomas

Genes Decreased in Tumors	<u>Hong Kong Cohort</u>		<u>NCI-Maryland Cohort</u>		<u>Adenoma Study</u>	
	Fold Change ¹	P-value ²	Fold Change ¹	P-value ²	Fold Change ¹	P-value ²
<i>IL-2</i>	0.23	P < 1 X 10 ⁻⁷	0.30	P < 1 X 10 ⁻⁷	0.54	P = 0.007
<i>IL-15</i>	0.42	P < 1 X 10 ⁻⁷	0.67	P = 0.006	0.79	P = 0.27
<i>IL-10</i>	0.46	P < 1 X 10 ⁻⁷	0.74	P = 0.006	0.61	P = 0.09
<i>IL-12A</i>	0.58	P = 1 X 10 ⁻⁷	0.57	P = 3 X 10 ⁻⁷	0.62	P = 0.009
<i>CSF1</i>	0.58	P < 1 X 10 ⁻⁷	0.54	P = 2 X 10 ⁻⁶	0.61	P = 0.01
<i>HLA-DPA1</i>	0.67	P < 1 X 10 ⁻⁷	0.69	P = 7 X 10 ⁻⁵	0.61	P = 0.003
<i>HLA-DRA</i>	0.69	P < 1 X 10 ⁻⁷	0.84	P = 0.03	0.75	P = 0.04
<i>PRG1</i>	0.80	P = 3 X 10 ⁻⁷	1.09	P = 0.19	0.69	P = 0.02
<i>TNF-a</i>	0.83	P = 0.03	0.59	P = 1 X 10 ⁻⁶	0.92	P = 0.60
<i>CD68</i>	0.90	P = 0.02	0.76	P = 0.0005	0.85	P = 0.25
Genes Increased in Tumors						
	Fold Change ¹	P-value ²	Fold Change ¹	P-value ²	Fold Change ¹	P-value ²
<i>IL-6</i>	2.06	P = 8 X 10 ⁻⁶	2.41	P = 0.0001	1.50	P = 0.44
<i>IFN-γ</i>	2.37	P < 1 X 10 ⁻⁷	1.87	P = 0.005	1.14	P = 0.53
<i>IL-17A</i>	2.71	P < 1 X 10 ⁻⁷	2.22	P = 0.02	4.36	P = 0.01
<i>FOXP3</i>	3.33	P < 1 X 10 ⁻⁷	1.65	P = 0.0003	1.60	P = 0.04
<i>IL-1B</i>	3.36	P < 1 X 10 ⁻⁷	1.88	P = 1 X 10 ⁻⁵	2.00	P = 0.001
<i>IL-1A</i>	5.21	P < 1 X 10 ⁻⁷	3.40	P < 1 X 10 ⁻⁷	6.20	P = 2.7 X 10 ⁻⁵
<i>IL-23A</i>	7.90	P < 1 X 10 ⁻⁷	4.37	P < 1 X 10 ⁻⁷	6.47	P = 3.7 X 10 ⁻⁴
<i>IL-8</i>	13.12	P < 1 X 10 ⁻⁷	11.09	P < 1 X 10 ⁻⁷	7.93	P = 9.0 X 10 ⁻⁷

¹Fold change in tumors vs. non-cancerous tissues or adenoma vs. nonadenoma tissue where appropriate.

²Paired T-test. Genes included in this table were differentially expressed (P < 0.01) in at least one cohort and are organized from low to high based on fold change in the Hong Kong Cohort. Patient samples used included all that passed qRT-PCR quality control (n=113 for Hong Kong Cohort, n=73 for NCI-Maryland Cohort and n=18 for adenoma study)

Supplemental Table 2: Association of *miR-21* expression with inflammatory genes *IL-6*, *IL-12a*, *IL-8*, *IL-10*, and *NOS2a*.

	<u>Nontumor Tissue (n=113)</u>		<u>Tumor Tissue (n=113)</u>		<u>Combined (n=226)</u>	
	Regression Coefficient	p-value	Regression Coefficient	p-value	Regression Coefficient	p-value
<i>IL-6</i>	0.13	0.004	0.13	0.01	0.13	<0.0005 *
<i>IL-12a</i>	-0.26	0.018	-0.19	<0.0005	-0.21	<0.0005 *
<i>IL-8</i>	0.21	<0.0005	0.05	0.209	0.13	<0.0005 *
<i>IL-10</i>	0.32	0.012	0.14	0.053	0.21	0.002 *
<i>NOS2a</i>	-0.30	0.001	-0.06	0.105	-0.12	0.003 *
<i>IL-1b</i>	0.29	0.001	0.02	0.616	0.12	0.006
<i>IL-17a</i>	-0.04	0.645	-0.12	0.001	-0.10	0.012
<i>PRG1</i>	0.39	0.040	0.15	0.207	0.26	0.019
<i>ANXA1</i>	0.05	0.742	0.20	0.012	0.15	0.063
<i>IL2</i>	-0.21	0.050	-0.04	0.509	-0.11	0.065
<i>CD68</i>	-0.22	0.187	0.11	0.385	-0.27	0.140
<i>CSF1</i>	-0.33	0.043	0.11	0.359	-0.12	0.241
<i>TNF</i>	0.51	0.489	0.12	0.273	0.06	0.264
<i>IL23a</i>	-0.21	0.071	0.00	0.960	-0.05	0.315
<i>IFNG</i>	0.02	0.817	-0.09	0.090	-0.05	0.331
<i>HLADRA</i>	-0.41	0.043	0.12	0.300	-0.09	0.408
<i>IL1a</i>	0.07	0.435	0.00	0.934	0.02	0.651
<i>IL15</i>	0.11	0.401	-0.01	0.877	0.03	0.653
<i>FOXP3</i>	-0.02	0.850	-0.02	0.808	-0.02	0.768
<i>HLADPA1</i>	-0.10	0.563	0.11	0.354	0.00	0.985

* = statistically significant with Holm-Bonferonni method of adjustment for multiple comparisons. Coefficients are shown for linear regression models of *miR-21* expression in nontumor tissue and tumor tissue separately with the expression of inflammatory genes. Combined models include both nontumor and tumor tissue and are adjusted for tumor status. Only the combined models are evaluated for significance.