Supplementary Figure Legends:

Supplementary Figure S1. Malignant Characteristics of Invasive Adenocarcinoma in K-ras^{LA1} Mice

(A – G) Representative images of H&E-stained sections from K-ras^{LA1} mice with invasive adenocarcinoma demonstrating invasion of pleural surface (A), intravasation into vasculature (B), implantation on intercostal muscle (C), invasion into hilar node (D), invasion into heart (E), and metastases to liver (F) and kidney (G).

Scale bars denote 100 μm.

Supplementary Figure S2. Radiation Effects on the Incidence of Various Endpoints in K-ras^{LA1} Mice

Overall incidences of lung tumors extending into bronchial airways (A), pneumonia (B), lymphoma (C), leukemia (D), lymphoma and leukemia combined (E), myeloproliferative disease (F), and focal liver hyperplasia and hepatoma in combined (G).

Supplementary Figure S3. Comparative Genomic Analyses and Classifier Isolation from Irradiated Versus Unirradiated Control K-ras^{LA1} Mice.

(A) Flowchart of overall analyses including integration of human cancer microarray datasets.

(B) Clustering of mouse samples using raw expression values and *lumi* package detected single outlier for exclusion from further analysis.

(C) Box plots of expression intensities from raw data (left panel) or after background correction and quantile normalization using *MBCB* package (right panel).

(D) Beta-uniform mixture model analysis of p-values (left panels) and hierarchical clustering using *classComparison* package with *t*-tests comparing with 632-gene set.

Supplementary Figure S4. Comparative Genomic Analysis of Whole Lungs Reveals Unique Gene Classifiers Capable of Specifying Individual Experimental Cohorts

(A) Schematic representation of experimental design.

(B) Hierarchical clustering and associated heatmap demonstrating capacity of 632 genes to segregate experimental cohorts. (ANOVA; $p < 0.05$).

(C) Principal component analysis validates capability of 632 genes to segregate experimental cohorts.

(D – L) Hierarchical clustering (D – F), k-means clustering (G – I), and principal component analysis (J – L) demonstrate robust capacity of unique gene classifiers to identify and segregate the designated experimental cohort from the other two cohorts.

Black = Control; Red = Acute; Blue = Fractionated.

Supplementary Figure S5. Only "Fractionated" Classifier Demonstrates Clinical Relevance for Lung Cancer Patient Survival

(A – I) Lung adenocarcinoma patient samples were partitioned into two groups using k-mean clustering and classifiers identifying unirradiated K-ras^{LA1} mice (A – C) or those irradiated with an acute (D – F) or fractionated dose (G – I) of 1.0 Gy ^{56}Fe- particles. Kaplan-Meier survival plots using overall survival from each cluster demonstrates clinical relevance of "fractionated" classifier.

Red and black lines denote high-risk and low-risk patients respectively. Hazard ratios and 95% confidence intervals are relative to high-risk patients.

Supplementary Figure S6. "Fractionated" Classifier Capable of Predicting Overall Survival in Patients with Breast, but not Lung Squamous Cell Cancer

Breast adenocarcinoma and lung squamous cell cancer patient samples were partitioned into two groups using k-means clustering and the 45-gene "fractionated" classifier. Analysis of overall survival from patients in each partitioned group demonstrates capacity of "fractionated" to identify patients with decreased survival with breast (A – C) and not lung squamous cell (D – E) carcinoma.

Red and black lines denote high-risk and low-risk patients respectively. Hazard ratios and 95% confidence intervals are relative to high-risk patients.

Supplementary Figure S7. Cox Regression Analysis Exposes 6 Genes Within "Fractionated" Classifier Which Retain Predictive Capacity

6 genes result from univariate Cox regression analysis ($p < 0.01$) using SPORE dataset and the 45-gene "fractionated" classifier. Patient samples from lung adenocarcinoma (A – C), squamous cell carcinoma, (F – G), and breast adenocarcinoma (C – E) were partitioned into two groups using 6-genes. Kaplan-Meier survival analysis using overall survival from each partitioned group demonstrates predictive capacity of "fractionated" for lung and breast adenocarcinoma (A – E), and not lung squamous cell carcinoma (F – G), was retained.

Red and black lines denote high-risk and low-risk patients respectively. Hazard ratios and 95% confidence intervals are relative to high-risk patients.

Supplementary Methods:

Microarray and Survival Analyses

All analysis was done using R 2.15.1 (http://www.R-project.org/) and tools in Bioconductor (http://www.bioconductor.org/) unless otherwise stated (1, 2). Mouse microarrays were performed using Illumina® MouseWG-6 v2.0 Expression BeadChips (Illumina). Samples were labeled and hybridized using Illumina® TotalPrep™ kit (Ambion). Arrays were scanned using Illumina® Beadstation 500 BeadArray reader and data acquisitioned with BeadStudio (Illumina®). One sample was identified as an outlier using the *lumi* package, and removed (Figure S3B) (3). Remaining samples were background corrected (non-parametric) and quantile normalized using the Model-based Background Correction for Beadarray algorithm (*MBCB* package) (Figure S3C) (4). K-ras^{LA1} expression data have been deposited under the accession number GSE42233 in the National Center for Biotechnology Information's Gene Expression Omnibus (GEO) public repository (http://www.ncbi.nlm.nih.gov/geo/). A variance filter was applied to retain only probes in which the inter-variation between any two classes was greater than the intra-variation in either class. This resulted in 4580 of 45,281 probes representing 4311 genes. Our analysis was limited to a final set of 1495 genes after cross-species gene mapping using AILUN (http://ailun.stanford.edu/) and cross-platform gene mapping by Entrez ID (5). An ANOVA, resulting in 632 genes, was performed using the *multtest* package (6); FDR was controlled using the Benjamini & Hochberg adjustment ($\alpha = .05$). Hierarchical clustering of samples and clustering of genes for the heatmap (Figure S4B) were implemented using packages *classDiscovery* (http://bioinformatics.mdanderson.org/Software/OOMPA) (7) and *pheatmap* (http://CRAN.R-project.org/package=pheatmap) (8). Principal component analysis (Figure S4C) using R's *prcomp* was implemented without centering or scaling since all

datasets were previously standardized. For each pair of groups (Control vs. Acute, Control vs. Fractionated, and Acute vs. Fractionated), we performed *t*-tests in which FDR was controlled using the beta-uniform mixture modeling described by Pounds and Morris and implemented using the *classComparison* package (http://bioinformatics.mdanderson.org/Software/OOMPA) (9, 10). Classifiers were assigned using the set of common overlapping genes from the t-tests for each group assignment. Heatmaps for classifiers (Figures S4D - F) as previously described above. MacQueen's k-means clustering algorithm (k=2) and classical multidimensional scaling were implemented to create ordination plots using the *vegan* package (http://CRAN.R-project.org/package=vegan) (Figures S4G - I) (11, 12). Principal component analysis (Figures S4J - L) was performed as described above. Network analysis was performed using Ingenuity Pathway Analysis (Figures 4A - D & 7A; Ingenuity® Systems, www.ingenuity.com).

The NCI's Director's Challenge Consortium lung adenocarcinoma dataset (n=442) was downloaded from caArray (https://array.nci.nih.gov/caarray/home.action) (13). Raw data was background corrected using default parameters and quantile normalized using RMAExpress (http://rmaexpress.bmbolstad.com/) (14, 15). For the UT-Lung SPORE lung dataset (GSE41271, n=209), only samples annotated as adenocarcinoma stage I-III (n =151) or squamous cell carcinoma (n=57) and not having received neo-adjuvant therapy were utilized. Two additional lung datasets were downloaded from GEO (http://www.ncbi.nlm.nih.gov/geo/). Raw data for the Aichi lung adenocarcinoma dataset (GSE13213, n=117) (16) was background corrected and quantile normalized using the *limma* package (17), and processed data was downloaded for the Raponi squamous cell

carcinoma dataset (GSE4573, n=130) (18). The following breast datasets were also downloaded from GEO: processed data from Miller et al (GSE3454, n=236) (19), Pawitan el at (GSE1456, n=159) (20), and the RData file for Loi et al (GSE6532, n=380) (21). All breast datasets were limited to patients with complete annotations for disease specific survival time and status. We used the median PC1 method described by Venet et al to classify patients into two groups (22). For each of the classifiers, using the SPORE dataset, the first principal component (PC1) was computed and the dataset was split using the median of PC1. Association between overall survival to each classifier was evaluated in the three lung adenocarcinoma datasets by log-rank comparison of survival curves using Kaplan-Meier estimators computed in R with the *survival* package (Figure 3) (http://CRAN.R-project.org/package=survival) (23). The 'predict' method for *prcomp* was used with the "fractionated" classifier to predict the NCI and Aichi datasets using the first principal component computed from the SPORE dataset. The "fractionated" classifier was also tested using the aforementioned squamous cell and breast carcinoma datasets (Figure 6) with median PC1 method. To further evaluate the association of each classifier with overall survival ($p < 0.01$), univariate Cox regression was applied. We repeated the median PC1 method for patient classification and survival analysis using the Cox-refined predictive 6-gene geneset derived the fractionated classifier (Figures 7B - K). Using the expression profiles of the complete classifiers as well as the Cox-filtered geneset, k-means clustering (k=2) was used to partition datasets into 2 groups for which survival curves were compared as previously stated (Figure S7).

Supplementary Tables:

Supplementary Table 1. Logistic Regression Analysis of Unirradiated K-ras^{LA1} Mice for Gender and Strain Effects on Various Endpoints.

Phenotype	Independent Variable	Odds Ratio (95% CI)
Invasive Carcinoma	Gender	2.67 (0.63,11.23)
	Strain	4.00 (0.95, 16.92)
Bronchial Extension	Gender	1.25 (0.42, 3.73)
	Strain	1.09 (0.37, 3.23)
Pneumonia	Gender	1.09 (0.40, 2.98)
	Strain	0.71 (0.26, 1.94)
Lymphoma	Gender	2.24 (0.52, 9.66)
	Strain	1.22 (0.31, 4.73)
Leukemia	Gender	2.32 (0.41, 13.02)
	Strain	8.73 (0.98, 77.63)
Myeloproliferative Disorder	Gender	1.28 (0.20, 8.32)
	Strain	N/A

Gender relative to males; Strain relative to 129S2.

Supplementary Table 2. Logistic Regression Analysis for Gender and Strain Effects on Various Endpoints Controlling for Experiment.

Phenotype	Independent Variable	Odds Ratio (95% CI)
Invasive Carcinoma	Gender	0.85 (0.57, 1.27)
	Strain	1.90 (1.27, 2.85)
Bronchial Extension	Gender	0.95 (0.67, 1.36)
	Strain	0.73 (0.51, 1.05)
Pneumonia	Gender	1.32 (0.93, 1.87)
	Strain	0.35 (0.24, 0.50)
Lymphoma	Gender	2.23 (1.28, 3.88)
	Strain	1.94 (1.13, 3.32)
Leukemia	Gender	1.28 (0.68, 2.38)
	Strain	4.18 (2.08, 8.42)
Myeloproliferative Disorder	Gender	1.37 (0.60, 3.14)
	Strain	7.69 (2.57, 23.02)

Relative to unirradiated K-ras^{LA1} mice; Gender relative to males; Strain relative to 129S2

Supplementary Table 3. Logistic Regression Analysis of Radiation Effects on the Incidence of Invasive Adenocarcinoma Controlling for Gender and Strain.

Experimental Group	Independent Variable	Odds Ratio (95% CI)
X- 1.0 x 1	Gender	1.19 (0.45, 3.19)
	Strain	1.24 (0.46, 3.34)
X- 0.2 x 5	Gender	1.19 (0.44, 3.19)
	Strain	1.50 (0.55, 4.07)
X- 0.4 x 5	Gender	3.00 (1.13, 7.99)
	Strain	3.09 (1.15, 8.30)
Fe- 0.1 Gy x 1	Gender	2.61 (1.10, 6.19)
	Strain	2.63 (1.10, 6.29)
Fe- 0.2 Gy x 1	Gender	0.96 (0.37, 2.47)
	Strain	0.98 (0.38, 2.54)
Fe- 1.0 Gy x 1	Gender	1.29 (0.55, 3.05)
	Strain	1.37 (0.58, 3.25)
Fe- 0.2 Gy x 5	Gender	2.47 (1.05, 5.80)
	Strain	2.62 (1.11, 6.20)
Fe- 0.1 Gy x 5	Gender	1.51 (0.57, 3.99)
	Strain	1.56 (0.59, 4.15)
Fe- (0.1 Gy x 5) x 2	Gender	1.89 (0.72, 4.95)
	Strain	1.75 (0.67, 4.63)

Relative to unirradiated K-ras^{LA1} mice; Gender relative to males; Strain relative to 129S2

Supplementary Table 4. Multivariate Cox Analysis for Gender and Strain Effects on Overall Survival Controlling for Experiment.

Phenotype	Independent Variable	Hazard Ratio (95% CI)
Overall Survival	Gender	1.17 (0.99, 1.39)
	Strain	0.75 (0.63, 0.90

Relative to unirradiated K-ras^{LA1} mice; Gender relative to males; Strain relative to 129S2

Supplementary Table 5. Multivariate Cox Analysis of Radiation Effects on Overall Survival Controlling for Gender and Strain Effects.

.

Experimental Group	Independent Variable	Hazard Ratio (95% CI)
X- 1.0 x 1	Gender	1.73 (1.13, 2.64)
	Strain	1.68 (1.10, 2.57)
X- 0.2 x 5	Gender	1.56 (1.02, 2.40)
	Strain	1.41 (0.92, 2.17)
X- 0.4 x 5	Gender	3.00 (1.87, 4.81)
	Strain	3.09 (1.92, 4.95)
Fe- 0.1 Gy x 1	Gender	2.07 (1.40, 3.04)
	Strain	2.12 (1.44, 3.12)
Fe- 0.2 Gy x 1	Gender	2.97 (2.01, 4.38)
	Strain	2.99 (2.03, 4.41)
Fe- 1.0 Gy x 1	Gender	3.39 (2.34, 4.91)
	Strain	3.46 (2.39, 5.01)
Fe- 0.2 Gy x 5	Gender	2.99 (2.03, 4.40)
	Strain	2.95 (2.00, 4.35)
Fe- 0.1 Gy x 5	Gender	2.09 (1.40, 3.12)
	Strain	2.24 (1.49, 3.34)
Fe- (0.1 Gy x 5) x 2	Gender	5.19 (3.35, 8.04)
	Strain	5.51 (3.59, 8.56)

Relative to unirradiated K-ras^{LA1} mice; Gender relative to males; Strain relative to 129S2

Supplementary Table 6. IPA Network Annotations Associated with Corresponding Gene Lists.

Gene List	Number of Networks	Network Number	Network Functions	Number of ANOVA Genes Included (% Total)
ANOVA	10	1	DNA Replication, Recombination, and Repair; Gene Expression; Infectious Disease	54 (8.5%)
		2	Gene Expression; Cell Cycle; DNA Replication, Recombination, and Repair	50 (7.9%)
		3	Cellular Movement; Immune Cell Trafficking; Gastrointestinal Disease	46 (7.3%)
		4	Developmental Disorder; Cell-To-Cell Signaling and Interaction; Tissue Development	42 (6.6%)
		5	Humoral Immune Response; Protein Synthesis; Cellular Compromise	42 (6.6%)
		6	Infectious Disease; Cell Cycle; Organismal Development	40 (6.3%)
		7	Cancer; Hematological Disease; Organismal Injury and Abnormalities	39 (6.2%)
		8	Post-Translational Modification; Cell Death and Survival; Tumor Morphology	38 (6.0%)
		9	Cell Death and Survival; Cancer; Neurological Disease	34 (5.4%)
		10	Cell Cycle; DNA Replication, Recombination, and Repair; Cell-To-Cell Signaling and Interaction	33 (5.2%)
Fractionated Classifier	3	1	Infectious Disease; DNA Replication, Recombination, and Repair; Gene Expression	26 (57.8%)
		2	Small Molecule Biochemistry; Organismal Injury and Abnormalities; Renal Damage	18 (40%)
		3	Cell Morphology; Cellular Assembly and Organization; Cellular Function and Maintenance	1 (2.2%)
6-gene Fractionated Classifier	1	1	Hematological Disease; Metabolic Disease; Cellular Compromise	6 (100%)

Supplementary References.

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Figure S1

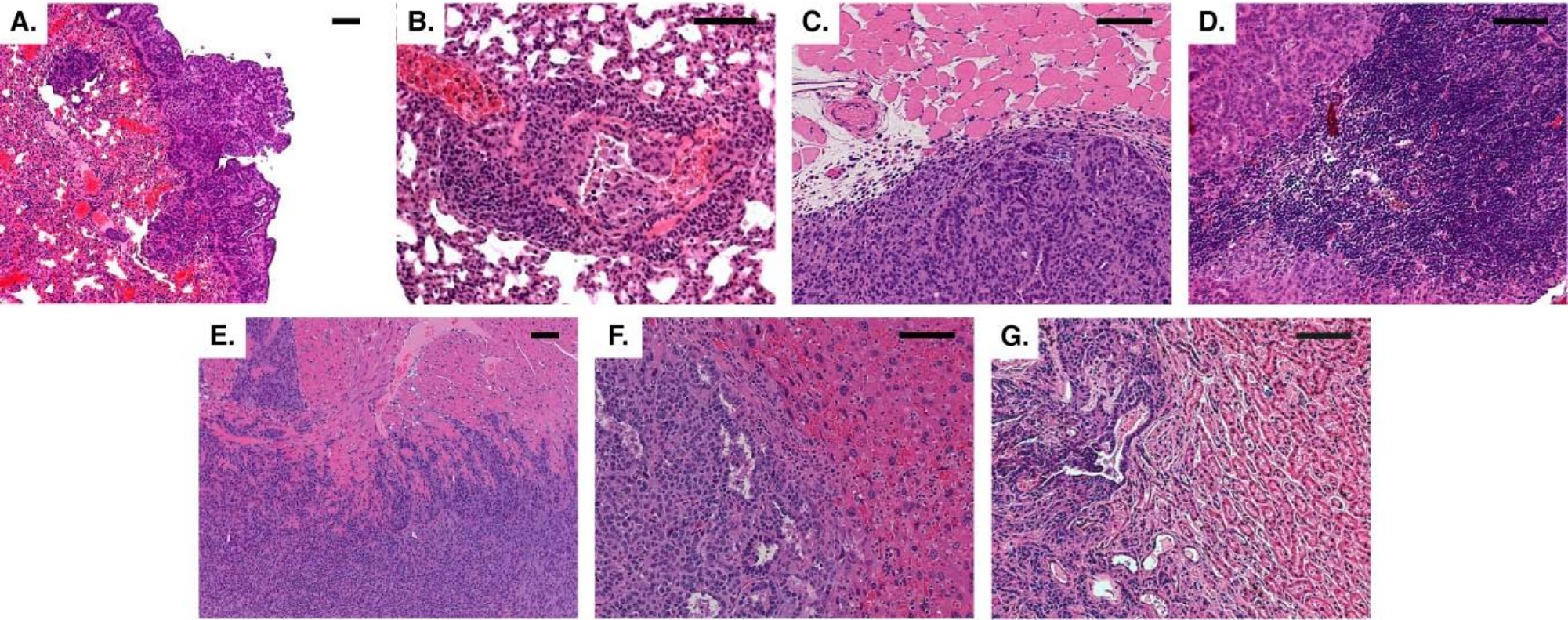


Figure S2

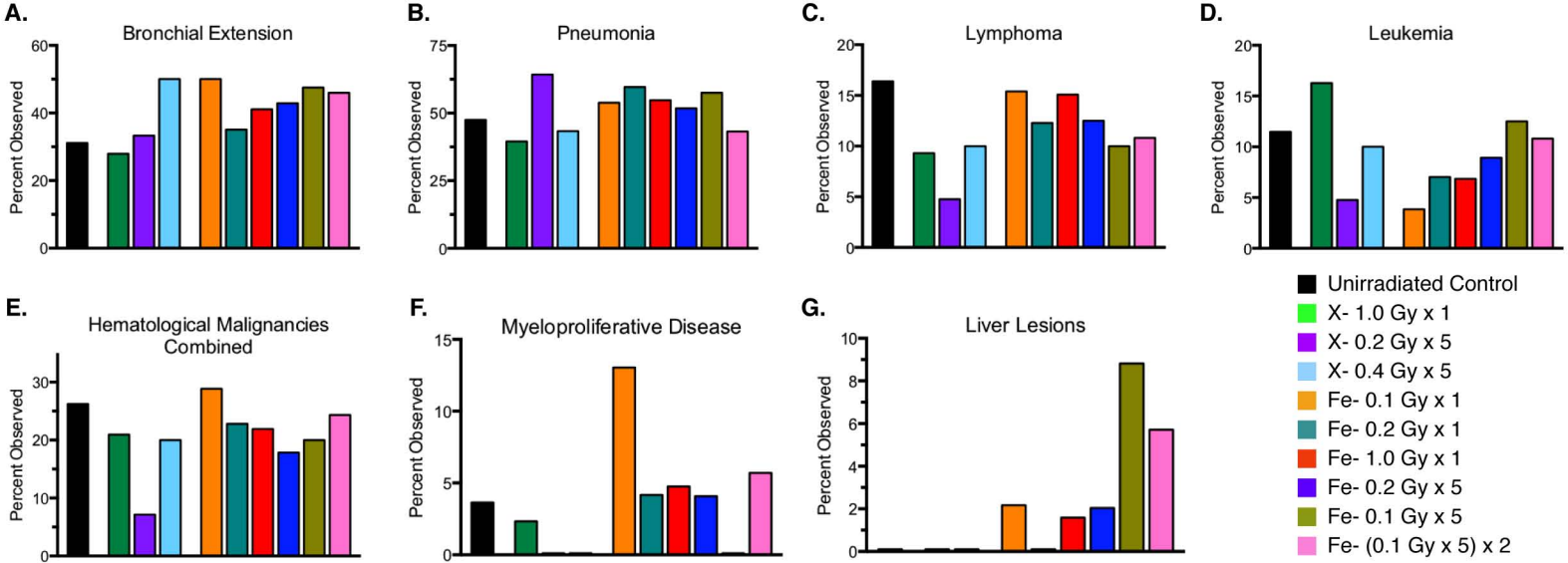

A.
Bronchial Extension
Percent Observed
60
40
20
0
B.
Pneumonia
Percent Observed
75
50
25
0
C.
Lymphoma
Percent Observed
20
15
10
5
0
D.
Leukemia
Percent Observed
20
15
10
5
0
E.
Hematological Malignancies Combined
Percent Observed
30
20
10
0
F.
Myeloproliferative Disease
Percent Observed
15
10
5
0
G.
Liver Lesions
Percent Observed
10
8
6
4
2
0
Unirradiated Control
X- 1.0 Gy x 1
X- 0.2 Gy x 5
X- 0.4 Gy x 5
Fe- 0.1 Gy x 1
Fe- 0.2 Gy x 1
Fe- 1.0 Gy x 1
Fe- 0.2 Gy x 5
Fe- 0.1 Gy x 5
Fe- (0.1 Gy x 5) x 2

Figure S3

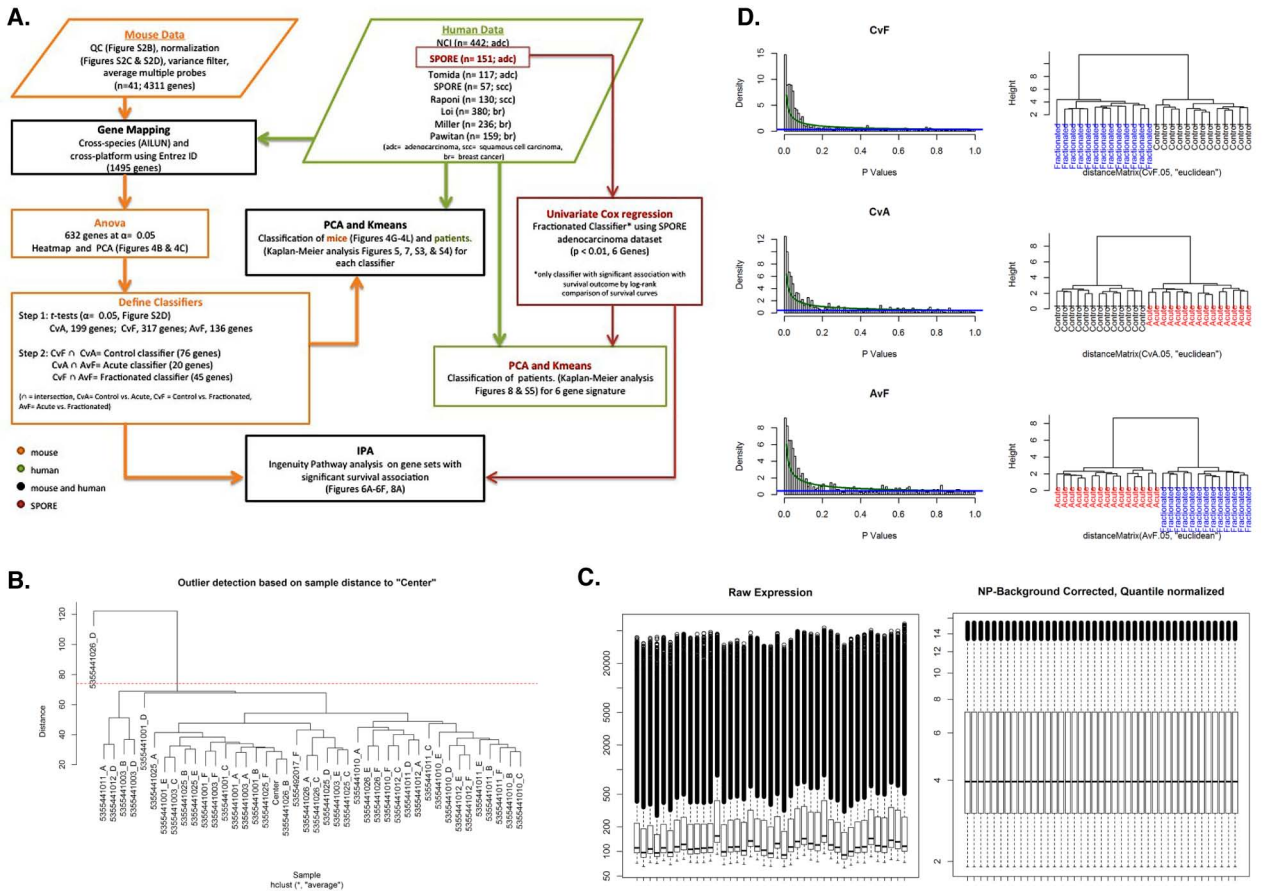
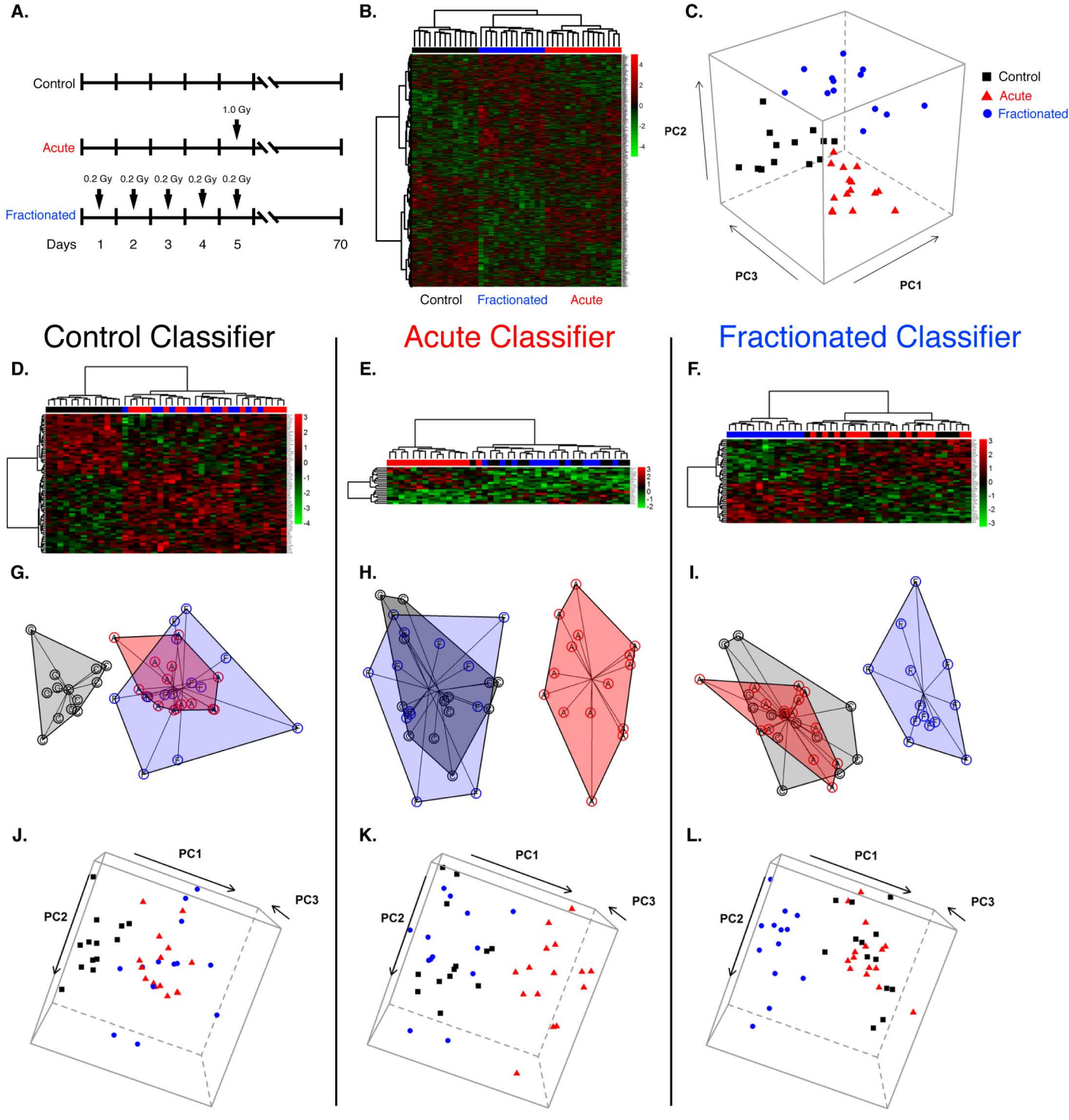

A.
Mouse Data
QC (Figure S2B), normalization
(Figures S2C & S2D), variance filter,
average multiple probes
(n=41; 4311 genes)
Human Data
NCI (n= 442; adc)
SPORE (n= 151; adc)
Tomida (n= 117; adc)
SPORE (n= 57; scc)
Raponi (n= 130; scc)
Loi (n= 380; br)
Miller (n= 236; br)
Pawitan (n= 159; br)
(adc= adenocarcinoma, scc= squamous cell carcinoma,
br= breast cancer)
Gene Mapping
Cross-species (AILUN) and
cross-platform using Entrez ID
(1495 genes)
Anova
632 genes at α= 0.05
Heatmap and PCA (Figures 4B & 4C)
PCA and Kmeans
Classification of mice (Figures 4G-4L) and patients.
(Kaplan-Meier analysis Figures 5, 7, S3, & S4) for
each classifier
Univariate Cox regression
Fractionated Classifier* using SPORE
adenocarcinoma dataset
(p < 0.01, 6 Genes)
*only classifier with significant association with
survival outcome by log-rank
comparison of survival curves
Define Classifiers
Step 1: t-tests (α= 0.05, Figure S2D)
CvA, 199 genes; CvF, 317 genes; AvF, 136 genes
Step 2: CvF ∩ CvA= Control classifier (76 genes)
CvA ∩ AvF= Acute classifier (20 genes)
CvF ∩ AvF= Fractionated classifier (45 genes)
(∩ = intersection, CvA= Control vs. Acute, CvF = Control vs. Fractionated,
AvF= Acute vs. Fractionated)
PCA and Kmeans
Classification of patients. (Kaplan-Meier analysis
Figures 8 & S5) for 6 gene signature
mouse
human
mouse and human
SPORE
IPA
Ingenuity Pathway analysis on gene sets with
significant survival association
(Figures 6A-6F, 8A)
D.
CvF
CvA
AvF
Density
P Values
Height
distanceMatrix(CvF.05, "euclidean")
distanceMatrix(CvA.05, "euclidean")
distanceMatrix(AvF.05, "euclidean")
B.
Outlier detection based on sample distance to "Center"
Distance
Sample
hclust (*, "average")
C.
Raw Expression
NP-Background Corrected, Quantile normalized

Figure S4


A.
Control
1.0 Gy
Acute
0.2 Gy 0.2 Gy 0.2 Gy 0.2 Gy 0.2 Gy
Fractionated
Days 1 2 3 4 5 70
B.
Control Fractionated Acute
C.
Control
Acute
Fractionated
PC2
PC3
PC1
Control Classifier
Acute Classifier
Fractionated Classifier
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E.
F.
G.
H.
I.
J.
K.
L.
PC1
PC2
PC3

Figure S5

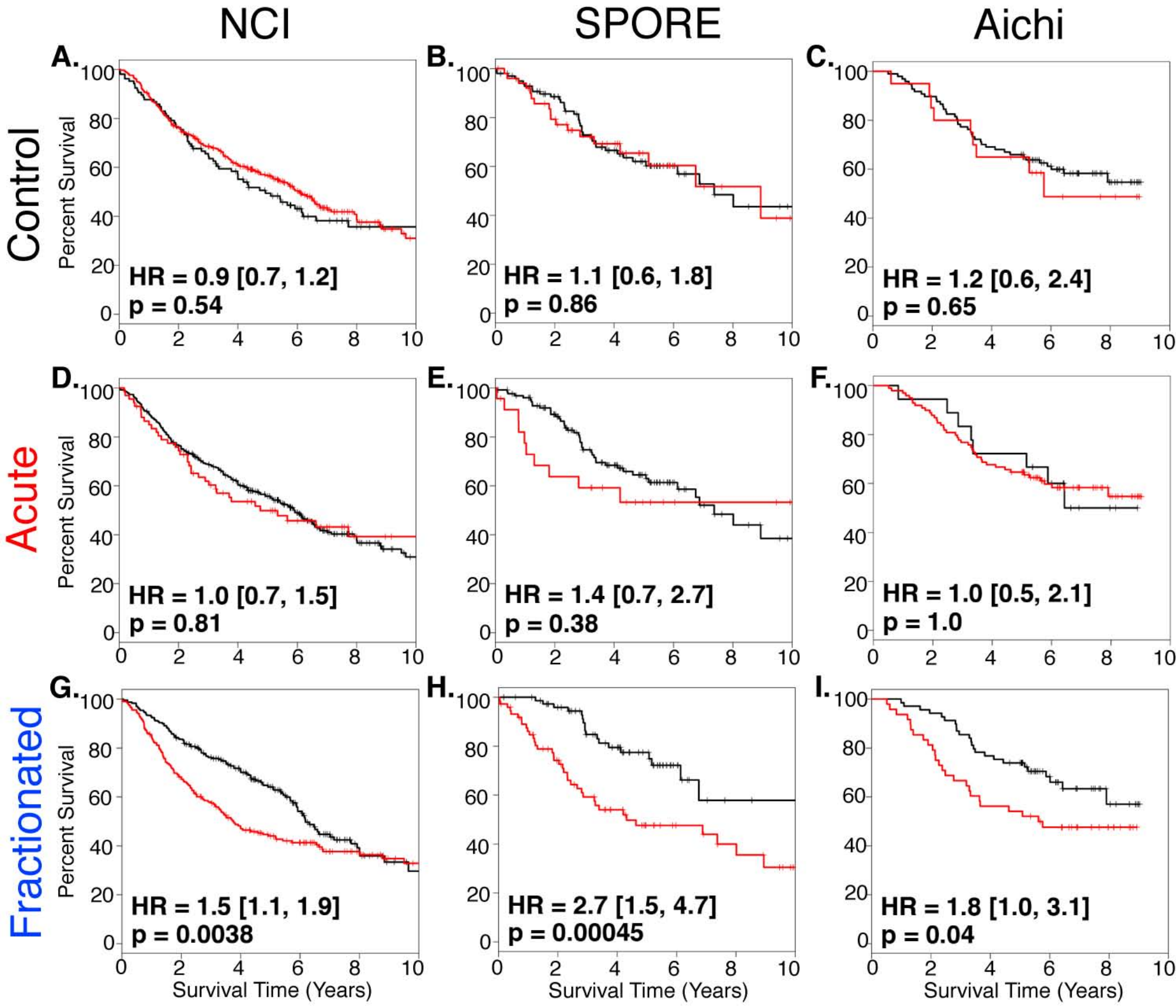

NCI
SPORE
Aichi
Control
Acute
Fractionated
A.
B.
C.
D.
E.
F.
G.
H.
I.
Percent Survival
Survival Time (Years)
HR = 0.9 [0.7, 1.2]
p = 0.54
HR = 1.1 [0.6, 1.8]
p = 0.86
HR = 1.2 [0.6, 2.4]
p = 0.65
HR = 1.0 [0.7, 1.5]
p = 0.81
HR = 1.4 [0.7, 2.7]
p = 0.38
HR = 1.0 [0.5, 2.1]
p = 1.0
HR = 1.5 [1.1, 1.9]
p = 0.0038
HR = 2.7 [1.5, 4.7]
p = 0.00045
HR = 1.8 [1.0, 3.1]
p = 0.04

Figure S6

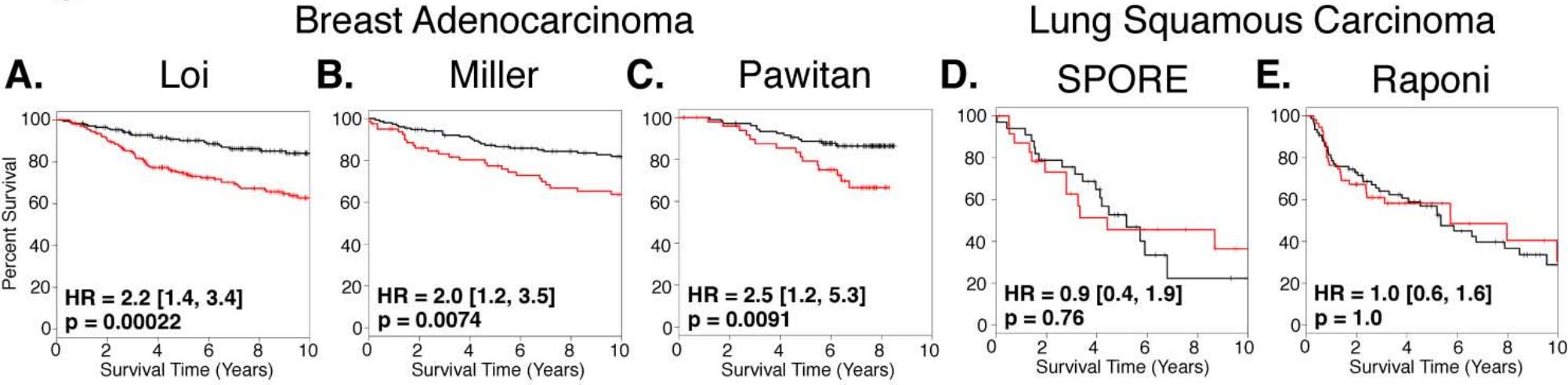
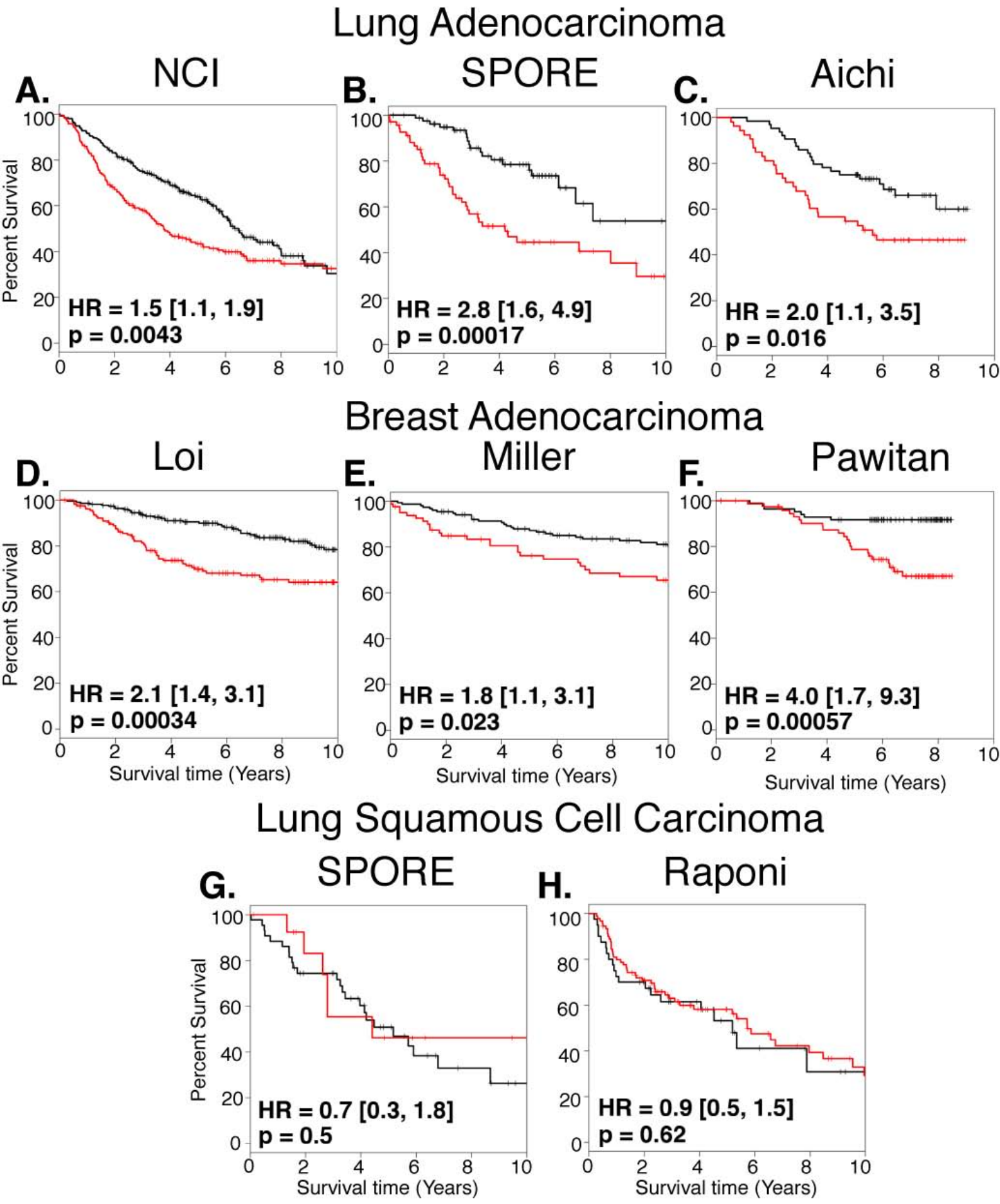

Breast Adenocarcinoma
Lung Squamous Carcinoma
A.
Loi
B.
Miller
C.
Pawitan
D.
SPORE
E.
Raponi
Percent Survival
Survival Time (Years)
HR = 2.2 [1.4, 3.4]
p = 0.00022
HR = 2.0 [1.2, 3.5]
p = 0.0074
HR = 2.5 [1.2, 5.3]
p = 0.0091
HR = 0.9 [0.4, 1.9]
p = 0.76
HR = 1.0 [0.6, 1.6]
p = 1.0

Figure S7


Lung Adenocarcinoma
A.
NCI
HR = 1.5 [1.1, 1.9]
p = 0.0043
B.
SPORE
HR = 2.8 [1.6, 4.9]
p = 0.00017
C.
Aichi
HR = 2.0 [1.1, 3.5]
p = 0.016
Breast Adenocarcinoma
D.
Loi
HR = 2.1 [1.4, 3.1]
p = 0.00034
E.
Miller
HR = 1.8 [1.1, 3.1]
p = 0.023
F.
Pawitan
HR = 4.0 [1.7, 9.3]
p = 0.00057
Lung Squamous Cell Carcinoma
G.
SPORE
HR = 0.7 [0.3, 1.8]
p = 0.5
H.
Raponi
HR = 0.9 [0.5, 1.5]
p = 0.62
Percent Survival
Survival time (Years)