Supplementary Information of
"Assessment of Polygenic Architecture and Risk Prediction based on Common Variants Across Fourteen Cancers"

Zhang et al.

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1. SUPPLEMENTARY TABLES

Supplementary Table 1. Sample sizes for summary-level GWAS data used for analysis of 14 cancers.

Group[a]	Cancer site	Number of SNPs analyzed after filtering in the model	Number of cases	Number of controls	Chip imputation Information
1	CLL[b1]	1,068,036	3,100	7,667	Infor score >0.3
	Esophageal[c2]	1,068,728	3,914	6,718	Info score >0.4
	Testicular[3]	1,066,591	3,558	13,970	Info score >0.3
	Oropharyngeal[4]	1,067,193	6,034	6,585	Call rate >98%
	Pancreas[5]	915,805	8,638	12,217	Info >0.3
2	Renal[6]	1,067,952	10,784	20,407	Info >0.3
	Glioma[7]	1,067,960	12,488	18,169	Info >0.4
	Melanoma[8]	1,052,042	12,874	23,203	Quality R^2>0.95
	Colorectal[9]	1,058,067	17,050	19,529	Call rate >95%, R^2 >0.7
	Endometrial[10]	1,068,132	12,906	108,979	Info score>0.4
	Ovarian[11]	1,068,810	22,406	40,951	Call rate >95%
3	Lung[12]	1,009,906	29,266	56,450	R^2>0.3, info >0.4
	Prostate[13]	806,185	79,148	61,106	R^2>0.8
	Breast[14]	1,067,502	108,067	88,386	Info score >0.3

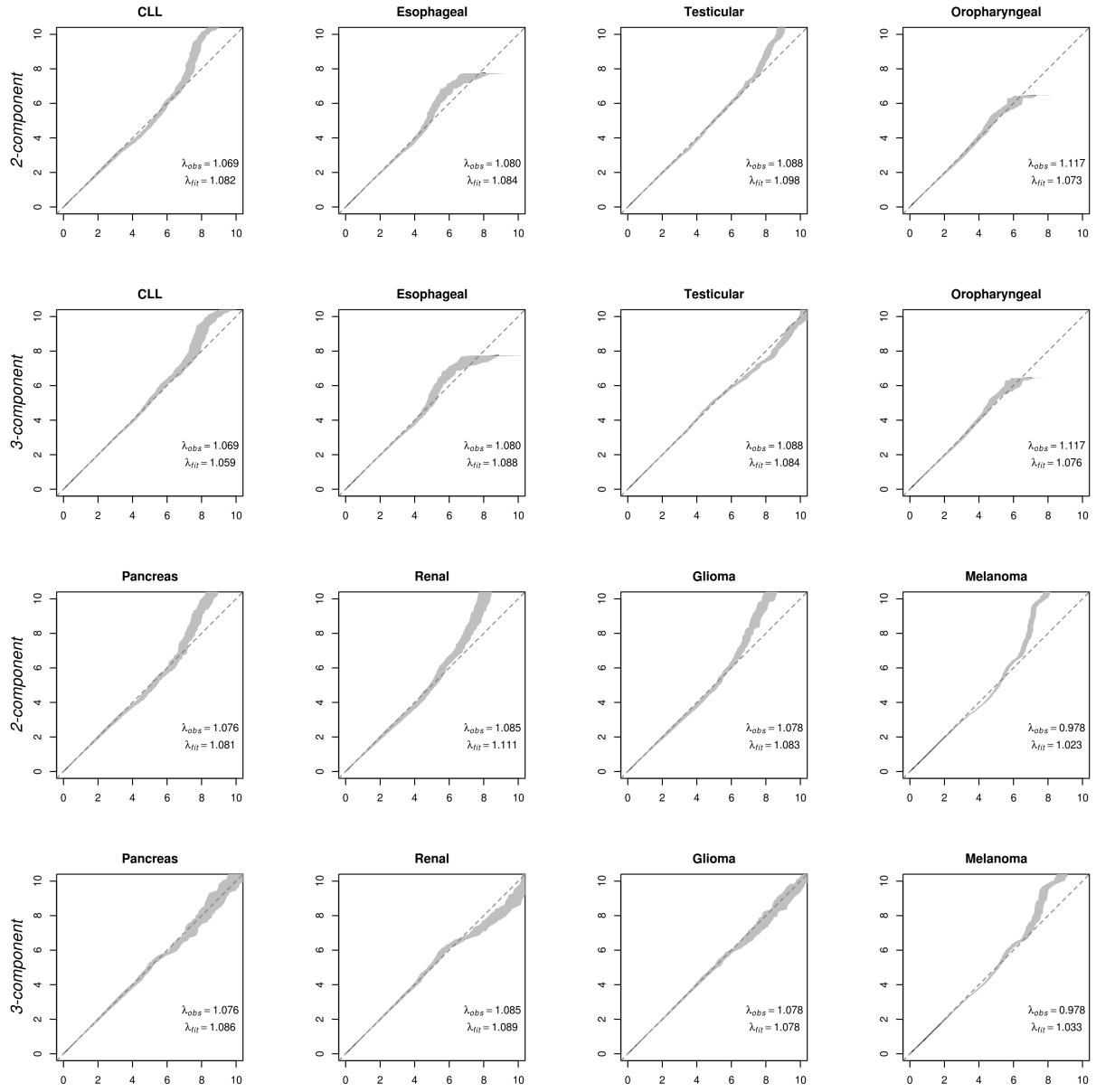
[a]Group 1, group 2, and group 3 include <10K, 10-25K, and >25K cases in the analysis, respectively. [b]CLL = chronic lymphocytic leukemia.
[c]Includes Barrett's esophagus and esophageal adenocarcinoma cases.

Supplementary Table 2. Number of independent genome-wide significant SNPs ($p< 5 \times 10^{-8}$) and associated heritability explained in the current datasets and those projected based on the estimated effect-size distribution using sample sizes of the current datasets. The number of independent SNPs reaching genome-wide significance in current studies were based on LD-clumping with an r^2-threhold of 0.1 and 1 MB window size. The heritability explained (h^2_{e}) associated with these SNPs were calculated using the formula $h^2_{\mathrm{e}} = \sum_i (\hat{\beta}_i^2 - \tau_i^2)$ where $\hat{\beta}_i$ is the estimate of log-odds-ratio (in standardized scale) and τ_i is the corresponding standard error for the i-th SNP.

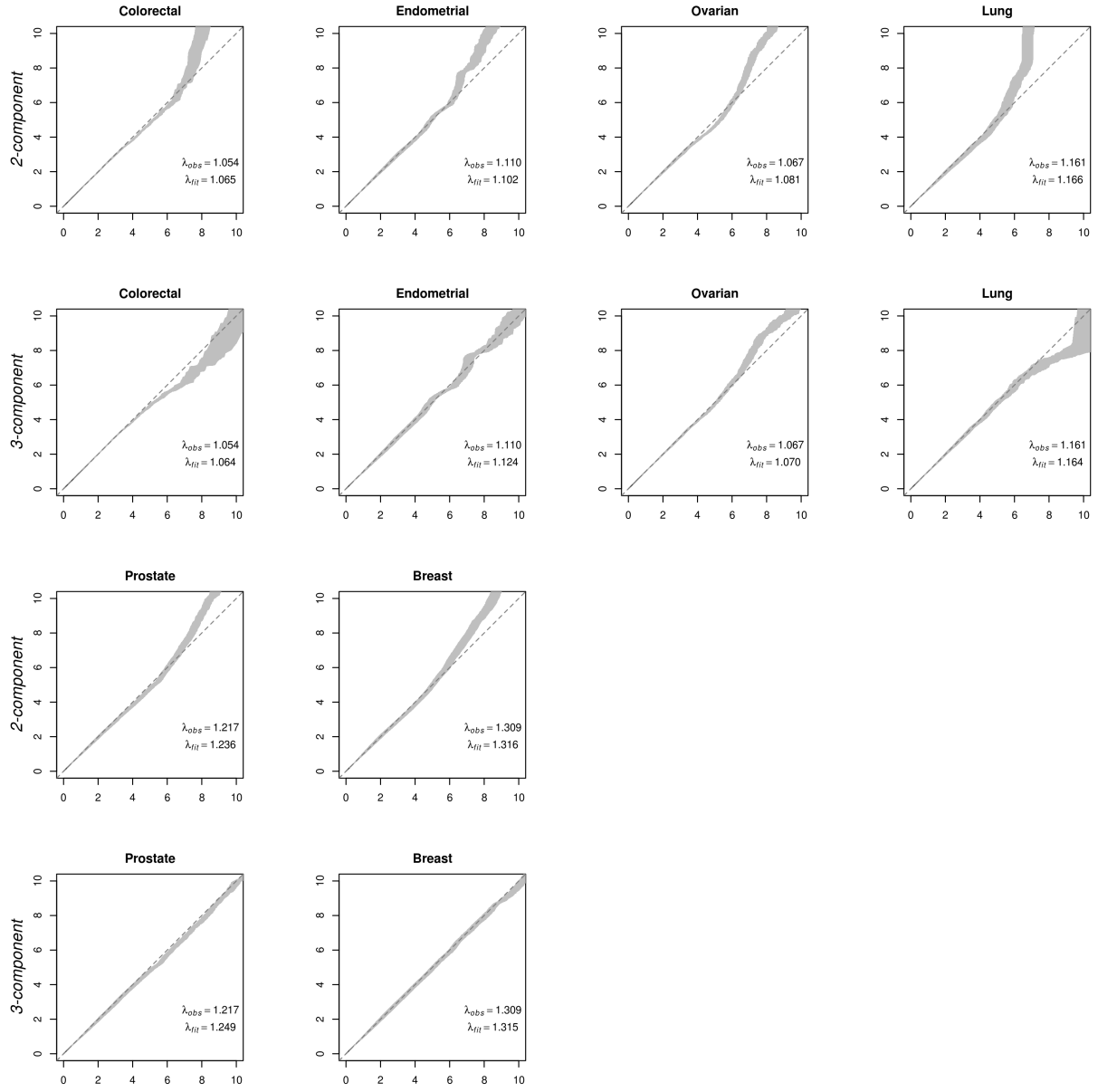
	Number of genome-wide significant SNPs observed	Observed h^2_{e} of the genome-wide significant SNPs	GENESIS projections of number of genome-wide significant SNPs (95%CI)	GENESIS projection of h^2_{e} of the genome-wide significant SNPs (95%CI)
CLL[a]	20	0.559	16 (8, 29)	0.53 (0.31, 0.83)
Esophageal	1	0.012	0 (0, 5)	0.00 (0.00, 0.03)
Testicular	38	0.908	44 (28, 79)	1.10 (0.81, 1.59)
Oropharyngeal	0	0.000	0 (NA)	0.00 (NA)
Pancreas	11	0.128	11 (5, 25)	0.13 (0.07, 0.24)
Renal	16	0.116	12 (5, 28)	0.10 (0.05, 0.17)
Glioma	33	0.488	22 (13, 35)	0.44 (0.33, 0.55)
Melanoma	26	0.265	28 (16, 48)	0.27 (0.20, 0.36)
Colorectal	8	0.039	8 (3, 19)	0.03 (0.01, 0.08)
Endometrial	13	0.047	12 (5, 34)	0.05 (0.03, 0.12)
Ovarian	12	0.056	16 (7, 34)	0.07 (0.04, 0.12)
Lung	15	0.066	8 (3, 18)	0.06 (0.04, 0.08)
Prostate	144	0.392	127 (102, 169)	0.37 (0.33, 0.41)
Breast	169	0.278	149 (120, 192)	0.27 (0.24, 0.29)

[a]CLL = chronic lymphocytic leukemia.

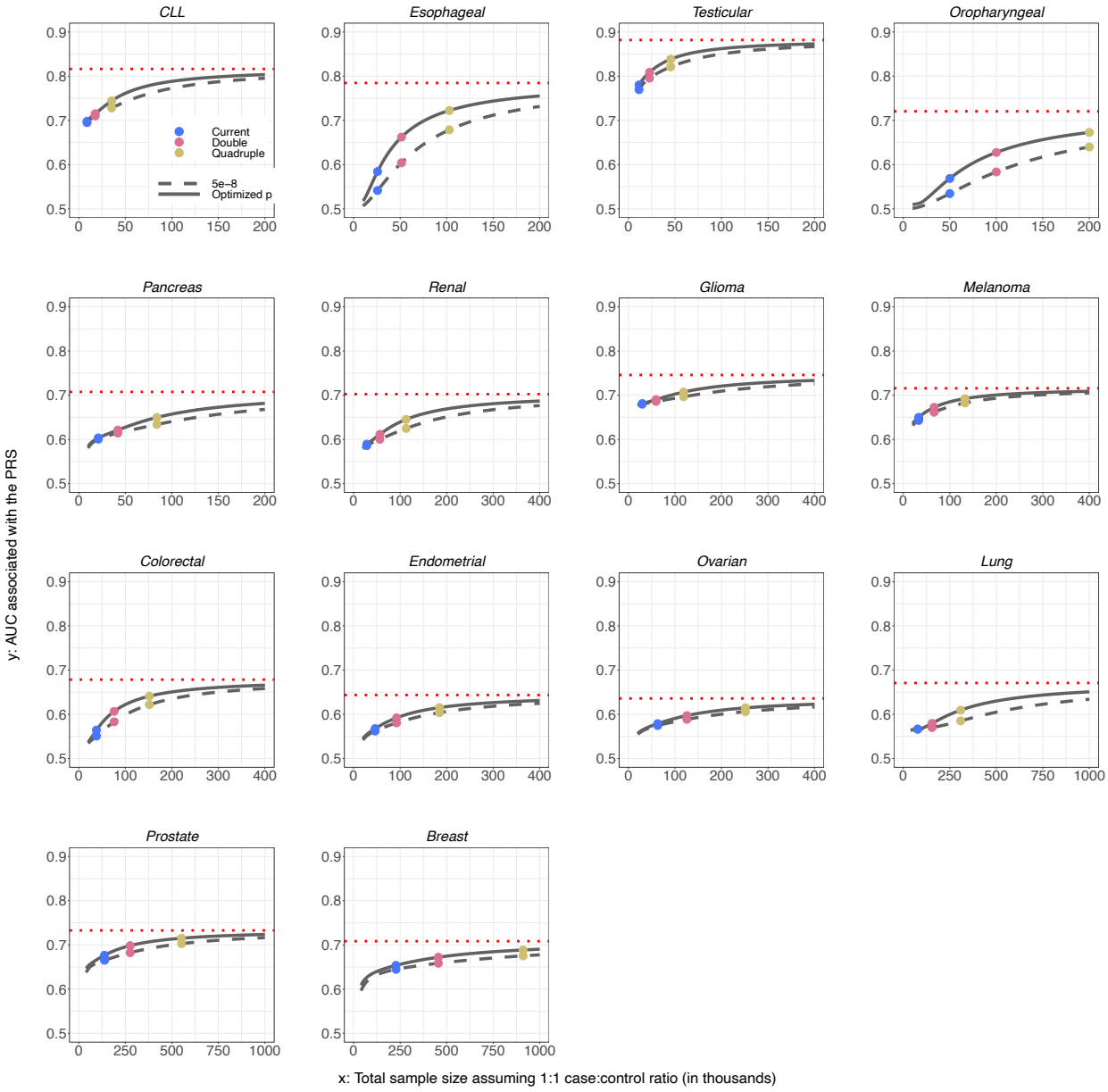
2. SUPPLEMENTARY FIGURES



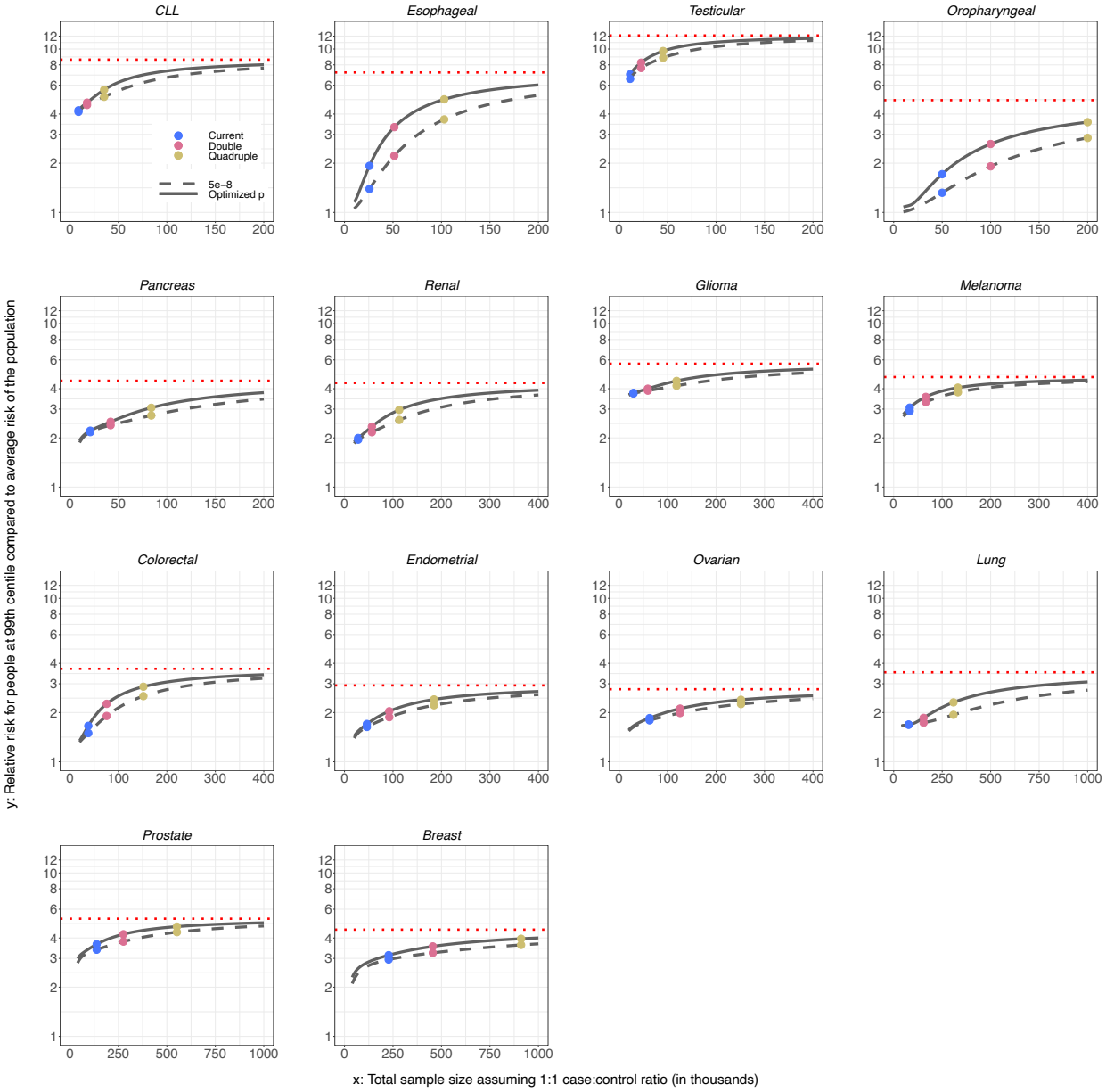
Supplementary Figure 1. Q-Q plots comparing observed distributions of GWAS summary statistics against those expected under the fitted GENESIS models across 8 different cancers. X-axis represents expected $-\log_{10}$(*p*-value), and y-axis represent observed $-\log_{10}$(*p*-value). Plots in upper and lower panels are generated under the two- and three-component models, respectively. The two-component model assumes the non-null effect sizes to follow a single normal distribution. The three-component model assumes the non-null effect sizes to follow a mixture of two normal distributions with two distinct variance components. Shaded regions mark 80% point-wise confidence intervals derived from 100 simulations. λ_{obs} is the genomic control factor in the observed summary-level GWAS data; λ_{fit} is the mean genomic control factor in simulated data over 100 replications. The ranges for all plots are restricted to be *p*-value < 10^{-10}. CLL = chronic lymphocytic leukemia.



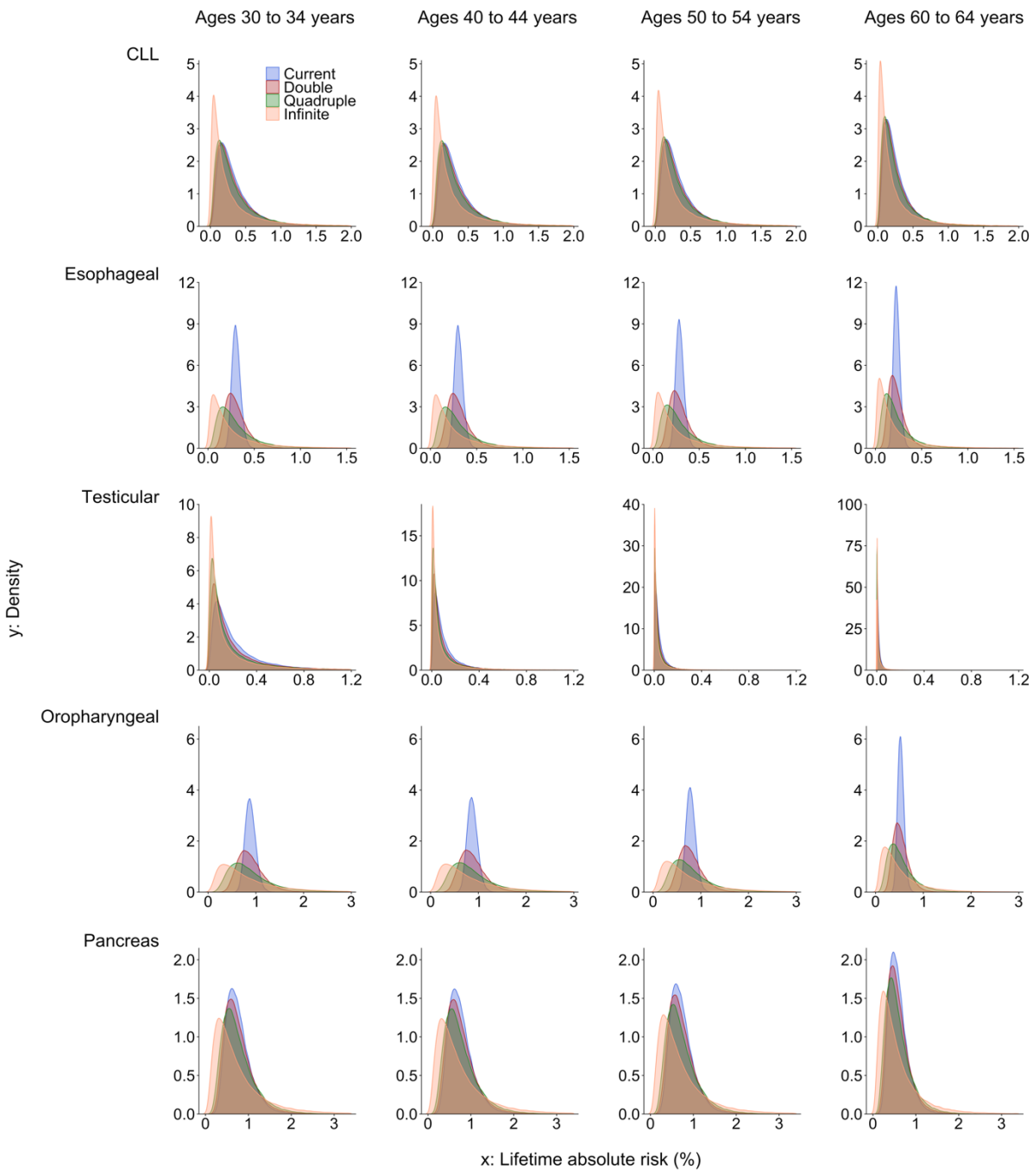
Supplementary Figure 2. Q-Q plots comparing observed distributions of GWAS summary statistics against those expected under the fitted GENESIS models across 6 different cancers. X-axis represents expected $-\log_{10}$(*p*-value), and y-axis represent observed $-\log_{10}$(*p*-value). Plots in upper and lower panels are generated under the two- and three-component models, respectively. The two-component model assumes the non-null effect sizes to follow a single normal distribution. The three-component model assumes the non-null effect sizes to follow a mixture of two normal distributions with two distinct variance components. Shaded regions mark 80% point-wise confidence intervals derived from 100 simulations. λ_{obs} is the genomic control factor in the observed summary-level GWAS data; λ_{fit} is the mean genomic control factor in simulated data over 100 replications. The ranges for all plots are restricted to be *p*-value < 10^{-10}.



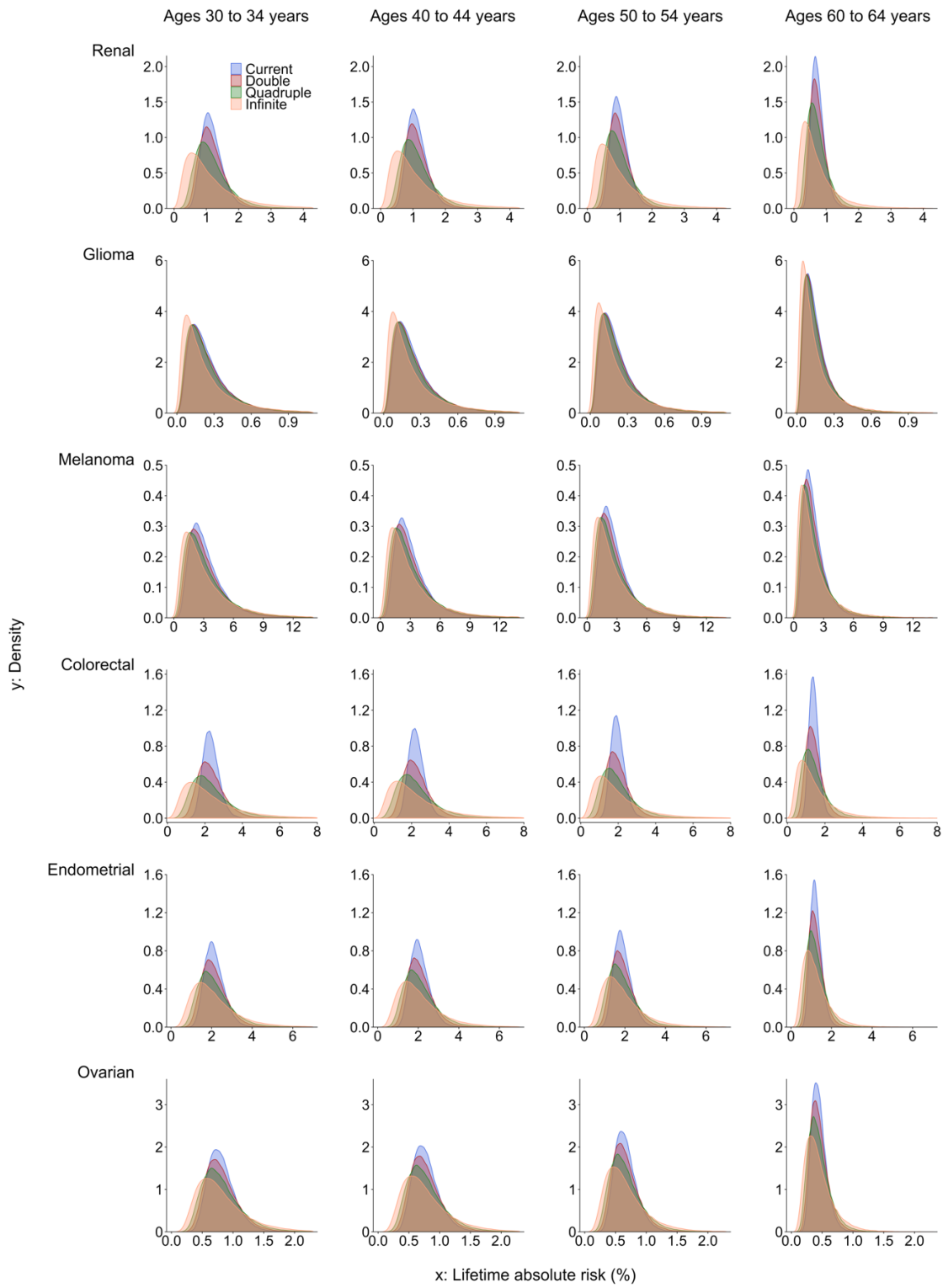
Supplementary Figure 3. Projections of area under the curve (AUC) characterizing predictive performance of PRS as sample size for GWAS increases. Results are shown for PRS including SNPs at the optimized *p*-value threshold (solid curve) and at genome-wide significance ($p<5 \times 10^{-8}$) level (dashed curve). The dotted horizontal red line indicates the maximum AUC achievable according to the estimate of GWAS heritability. Colored dots correspond to sample size for largest published GWAS and those for doubled and quadruped sizes. For oropharyngeal cancer, the projections at the "current sample size" are based on a sample size of 25K cases and 25K controls. For breast and esophageal cancer, the projections at the "current sample size" are based on the current largest GWAS sample sizes: 123K cases and 106K controls, and 10K cases and 17K controls, respectively. For all other cancer sites, the projections at the "current sample size" are based on the GWAS sample sizes in **Supplementary Table 1.** CLL = chronic lymphocytic leukemia.



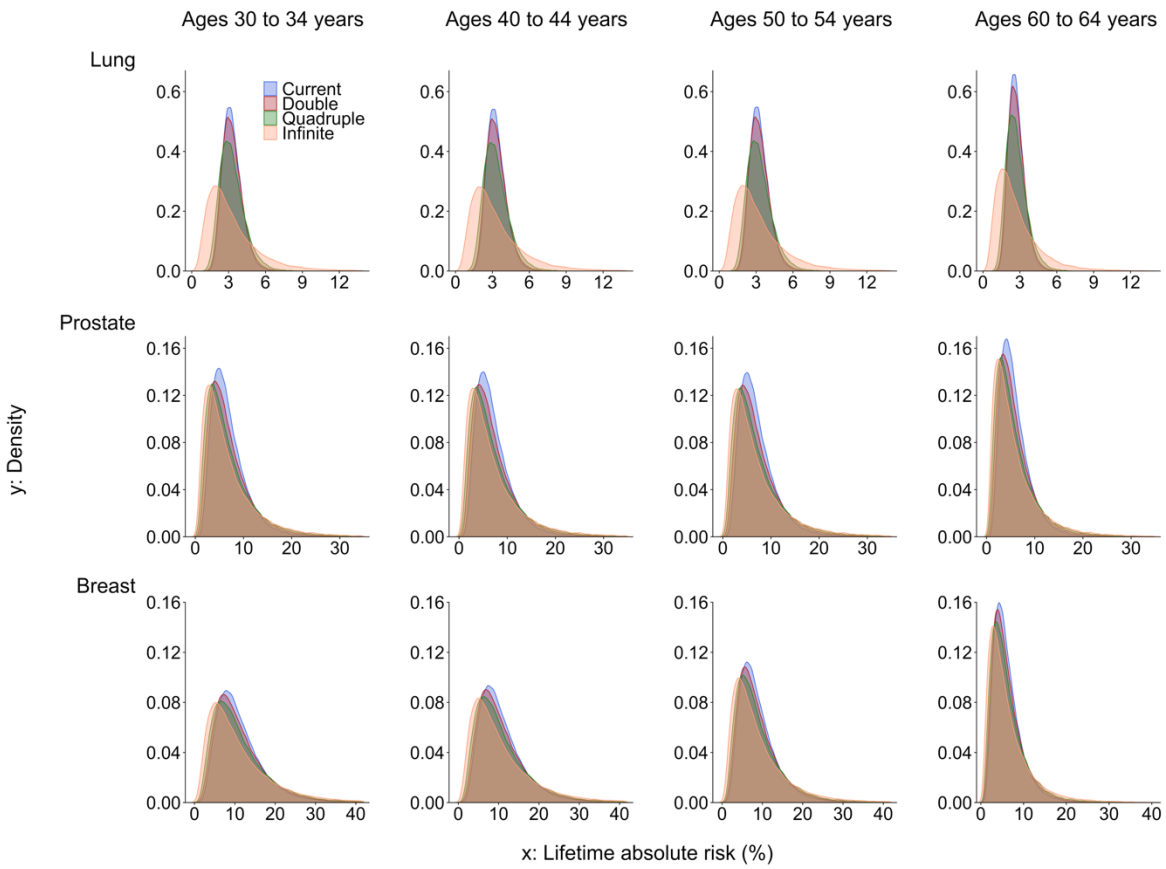
Supplementary Figure 4. Projections of relative risks for individuals at or higher than 99th percentile of PRS distribution (compared to average risk) as sample size for GWAS increases. Results are shown for PRS including SNPs at the optimized *p*-value threshold (solid curve) and at genome-wide significance ($p<5 \times 10^{-8}$) level (dashed curve). The dotted horizontal red line indicates the maximum relative risk achievable according to estimate of GWAS heritability. Colored dots correspond to sample size for largest published GWAS and those for doubled and quadruped sizes. Y-axis is presented in log10 scale.
For oropharyngeal cancer, the projections at the "current sample size" are based on a sample size of 25K cases and 25K controls. For breast and esophageal cancer, the projections at the "current sample size" are based on the current largest GWAS sample sizes: 123K cases and 106K controls, and 10K cases and 17K controls, respectively. For all other cancer sites, the projections at the "current sample size" are based on the GWAS sample sizes in **Supplementary Table 1.** CLL = chronic lymphocytic leukemia.



Supplementary Figure 5. Projected distribution of age-stratified residual lifetime risk (up to age 75) in US Non-Hispanic Whites according to variation of polygenic risk scores (group 1 cancers). Colored shades correspond to sample size for largest published GWAS and those for doubled, quadruped and infinite sizes. For oropharyngeal cancer, the projections at the "current sample size" are based on a sample size of 25K cases and 25K controls. For esophageal cancer, the projections at the "current sample size" are based on the current largest GWAS sample sizes: 10K cases and 17K controls. For all other cancer sites, the projections at the "current sample size" are based on the GWAS sample sizes in **Supplementary Table 1.** CLL = chronic lymphocytic leukemia.



Supplementary Figure 6. Projected distribution of age-stratified residual lifetime risk (up to age 75) in US Non-Hispanic Whites according to variation of polygenic risk scores (group 2 cancers). Colored shades correspond to sample size for largest published GWAS and those for doubled, quadruped and infinite sizes. For all cancer sites, the projections at the "current sample size" are based on the GWAS sample sizes in **Supplementary Table 1.**



Supplementary Figure 7. Projected distribution of age-stratified residual lifetime risk (up to age 75) in US Non-Hispanic Whites according to variation of polygenic risk scores (group 3 cancers). Colored shades correspond to sample size for largest published GWAS and those for doubled, quadruped and infinite sizes. For breast cancer, the projections at the "current sample size" are based on the current largest GWAS sample sizes: 123K cases and 106K controls. For all other cancer sites, the projections at the "current sample size" are based on the GWAS sample sizes in **Supplementary Table 1.**

3. SUPPLEMENTARY NOTE

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