

Supplemental Appendix

1. Supplemental Table 1 (2-3)
2. Supplemental Figures 1-4 (4-9)

Milène Crispin^{1,8}, Justin M. Ko^{1,8}, Brittany G. Craiglow^{2,3},

Shufeng Li¹, Gautam Shankar¹

Jennifer R. Urban⁴, James C. Chen^{5,6}, Jane E. Cerise⁵, Ali Jabbari⁵

Mårten CG Winge¹, M. Peter Marinkovich¹

Angela M. Christiano^{5,7}

Anthony E. Oro^{1,9}, Brett A. King^{2,9}

¹Program in Epithelial Biology and Department of Dermatology, Stanford University School of Medicine, Stanford, CA 94305; ²Departments of Dermatology, ³Pediatrics, and ⁴Internal Medicine, Yale University School of Medicine, New Haven, CT 06520; ⁵Department of Dermatology and ⁶Department of Systems Biology, ⁷Department of Genetics & Development, Columbia University, New York, New York 10032

⁸These authors contributed equally

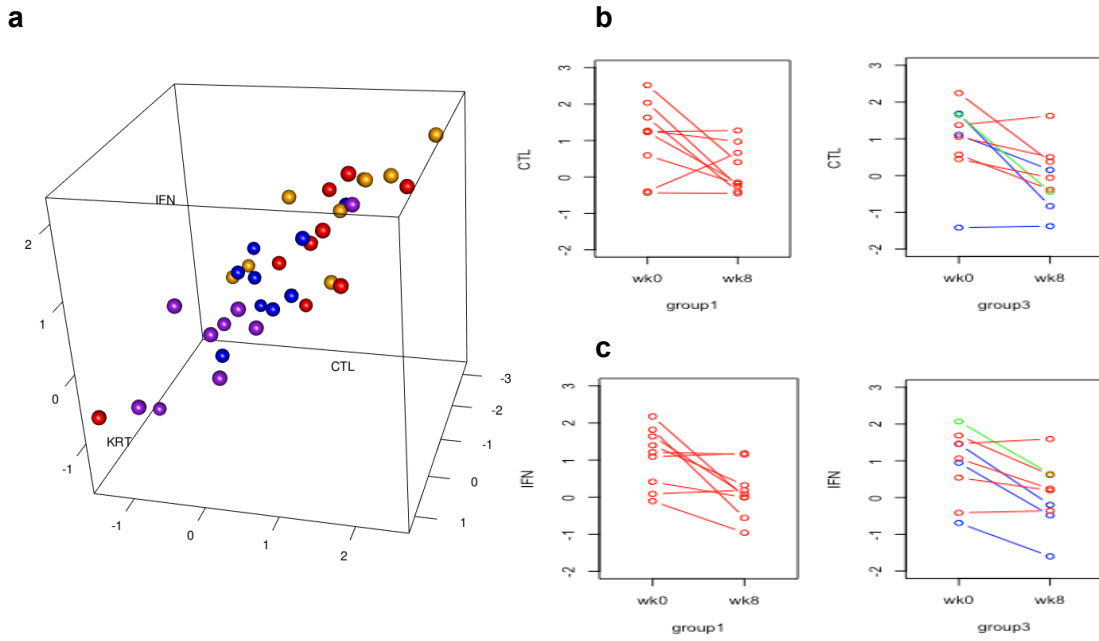
⁹co-senior authors

Supplemental Table 1. This gene list represents the top 50 statistically significant genes that are most differentially expressed between slow (SR) and likely non-responders (NR).

Gene	ttest(s.n)	log(fc)
SLC7A14	0.002767832	3.523309021
MIOX	0.015596102	3.191259203
DIRC1	0.004402072	3.058898071
SLCO1A2	0.01963604	2.94139846
ANKRD20A4	0.039423338	2.88185743
PDC	0.011076336	2.753828344
OTC	0.038039608	2.680887437
ZNF280A	0.02930004	2.632885846
BCYRN1	0.037133214	2.614524478
GDPD4	0.004362721	2.578516756
FMR1-AS1	0.032126901	2.542406675
MIR4326	0.037907732	2.537403617
FAM55B	0.034753999	2.428320795
HIST1H3J	0.02383998	2.221549811
HCRTR1	0.007586759	2.168706377
CFHR3	0.033157002	2.120530826
MIR590	0.00191407	2.043752204
PSORS1C3	0.010229834	2.029357634
FABP5P3	0.030070292	1.96029711
XIRP1	0.005415287	1.886416152
GPR31	0.041739374	1.863002684
ASB5	0.00353221	1.858190055
RAET1K	0.024034222	1.840665554
SNORD9	0.03600298	1.807327907
POTEKP	0.043495986	1.800695457

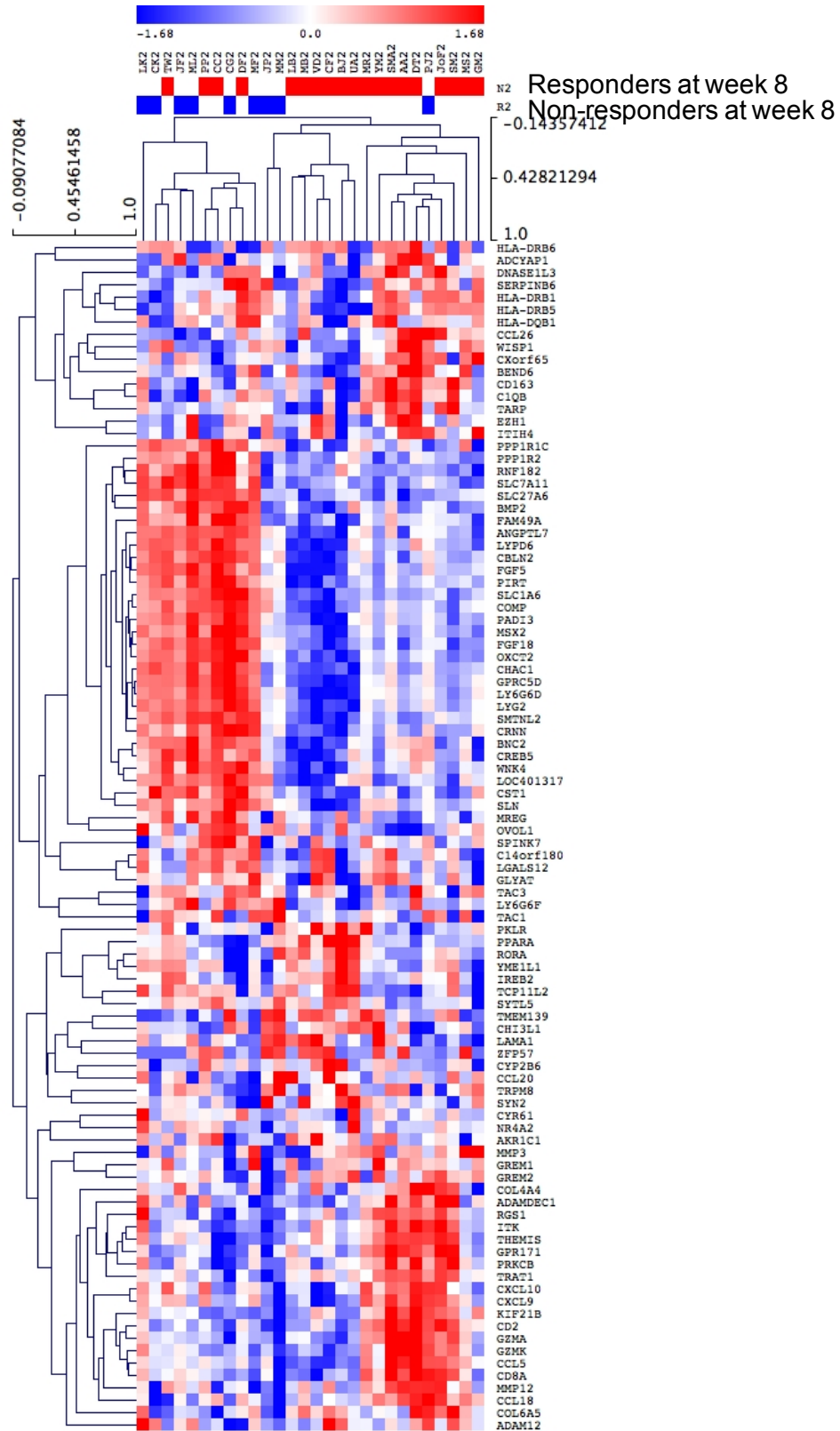
HOXA11-AS1	0.005042483	1.78186747
HLA2	0.014048475	1.760739028
LINC00402	0.048743482	1.754801301
ARMCX3-AS1	0.043888376	1.737035566
SFTPA2	0.046059014	1.719020263
FLJ45139	0.037083091	1.715862546
FLJ38723	0.016558928	1.702035568
MIR1302-3	0.039082597	1.67754393
ABCG8	0.031811604	1.582217348
GAL3ST3	0.027780041	1.468000011
OR10AD1	0.041227113	1.424409801
PCDHA1	0.022831717	1.422256826
OR4D11	0.040630715	-1.56992102
PCDHA13	0.046153857	-1.672451755
HIST1H1B	0.048226688	-1.680579141
HIST2H2AB	0.025019708	-1.686828477
SYT14L	0.011682506	-1.714422245
FLJ45832	0.036863506	-1.731170151
FAM58BP	0.004432895	-2.252232834
RSPH10B2	0.049633961	-2.324666211
IGLL1	0.048819027	-2.429162596
SEMG2	0.006326554	-3.147040553
IZUMO2	0.032228509	-3.221385982
H2BFM	0.028766749	-3.34025549
NMUR2	0.044236358	-3.656580098

Supplemental Figure 1: We collected scalp biopsies from 28 patients and performed transcriptomal analysis using RNA sequencing. (a) A 3D plot of ALADIN CTL, IFN and KRT scores shows that groups 1 and 3 cluster at baseline and cluster at week 8 (red = group 3 at baseline, orange = group 1 at baseline, purple = group 3 at week 8, blue = group 1 at week 8) (b) A plot of CTL score at baseline and week 8 for group 1 and group 3 (red=AU/AT, green=Ophiasis, blue=AA) and (c) plot of IFN score at baseline and week 8 for group 1 and group 3 (red = AU/AT, green = Ophiasis, blue = AA) changes in CTL scores and in IFN scores between baseline and week 8 were not significantly different in group 1 compared with group 3.



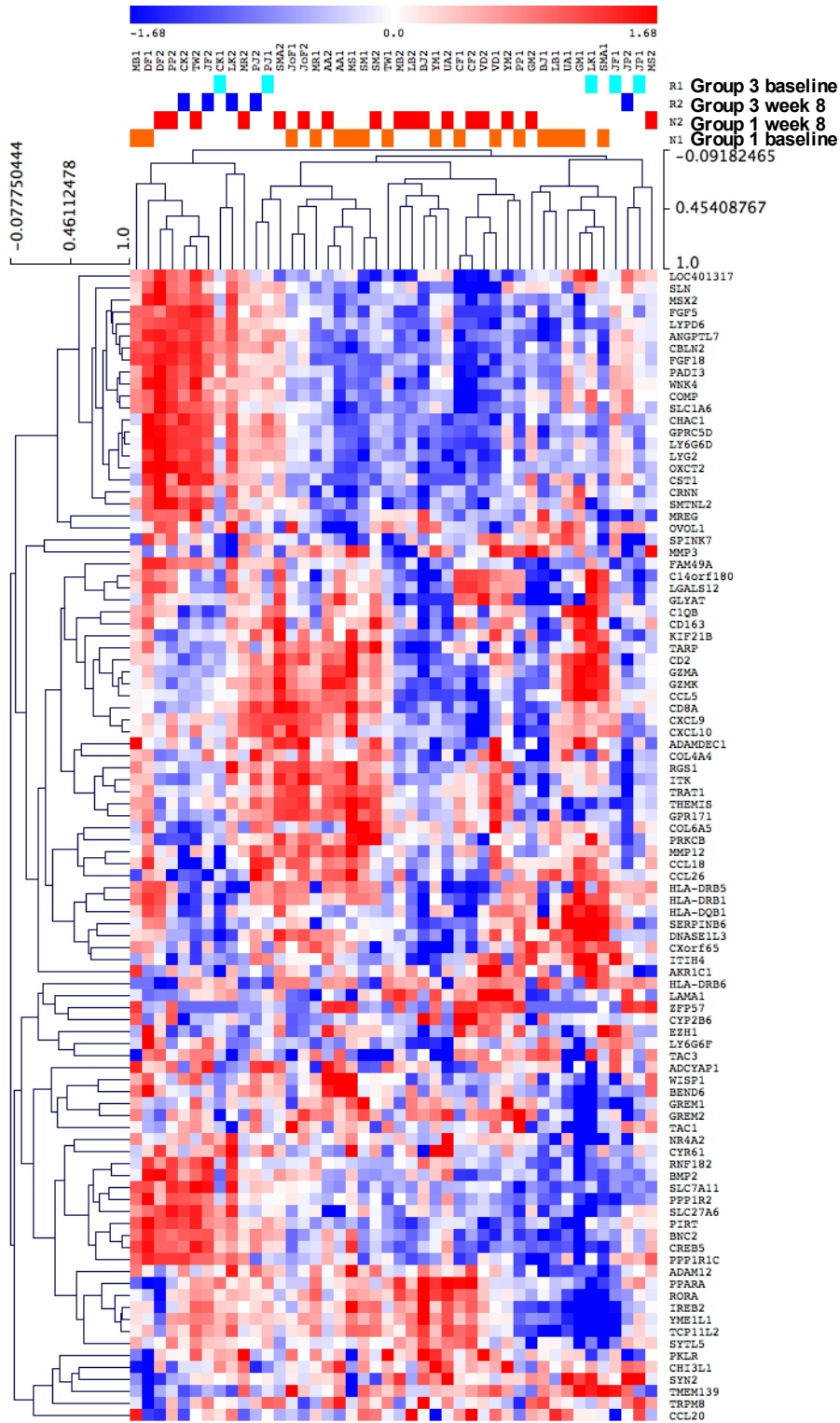
Supplemental Figure 2: Unsupervised hierarchical clustering of all biosamples taken at week 8, naïve to response success, was performed using previously published AA gene expression signatures. The clustering shows statistically significant separation of response correlated with SALT>50 scores ($p<0.05$). Secondary clustering was observed particularly in the non-responder group, suggesting the possibility of distinct “subtypes” of non-responder patients.

Supplemental Figure 2



Supplemental Figure 3: (a) A repeat of the unsupervised hierarchical clustering on published AA gene signatures including biosamples across both timepoints reveals a time-dependent co-clustering of patients. Specifically, patients designated as true responders in group 3 showed the most significant molecular divergence between initial (teal) and week 8 (blue) time points, represented as samples falling in disparate clusters based. The patients designated as nonresponders again displayed two distinct molecular subpopulations. (b) A principal component (PC) analysis of this data reveals that the non-responder molecular subtypes can be represented as patients whose change in molecular signature over time are not concordant with those observed in the responders (true non-responders), and those that are concordant, but seemingly delayed (slow responders). These molecular subtypes were subsequently defined and used for naïve biomarker development for predicting drug response.

Supplemental Figure 3



Supplemental Figure 4: The mean time until the start of scalp hair shedding during the follow up period was 10.8 weeks, with the median 8.5 weeks

