

## Supplementary Discussion

### Identification of rs4687102 as a dynamic element during differentiation

The disease-linked rs4687102 SNV breaks an ATF1/2/4 motif near the TP63 gene (**Supplementary Fig 5a-b**). rs4687102 risk and protective alleles were differentially active in driving transcription in MPRA (**Supplementary Fig 5c**). To examine potential ATF4 binding to rs4687102, CUT&RUN of ATF4 in a heterozygous individual was performed, revealing that the risk allele bound ATF4 at 68% of the level as the protective allele (**Supplementary Fig. 5d, inset**,  $P=0.06$ , two-sided binomial). Other studies in other tissues have found CEBPG, CEBPB, and ATF2/3/4 bound at this SNV, which could also be affected TFs<sup>1</sup>. These data propose a potential model in which rs4687102 SNV-mediated differences in the binding of ATF4 (or potentially CEBPB/G) to an enhancer dysregulates *TP63* expression in a manner that may predispose to the emergence of the altered epidermal homeostasis observed in lesional psoriatic tissue and suggest a rationale for additional studies of disease-linked SNV impacts on TF DNA binding.

## Supplementary Figure Legends

**Supplementary Fig. 1. Sankey diagram of polygenic skin disease incidence.** Incidence numbers reflect rates among U.S. persons, all ages, both sexes, 2021 GDB data<sup>2</sup> (with a few exceptions, see Methods). A rate of 1 per 100 person-years corresponds to one diagnosis per 100 people per year.

**Supplementary Fig. 2. Flow chart of the high throughput experiments in this work.** Blue boxes represent experiments, and green boxes represent analytical results. CUT&RUN candidates were determined from the union set of motifs enriched in MPRA, CRISPR screen hits, and TFs established in skin in the literature.

**Supplementary Fig. 3. Correlations in disease risk genes for polygenic skin disease. a** LDSC genetic correlation of 9 skin disease GWAS. Genetic correlations that surpassed significance thresholds following Benjamini–Hochberg (FDR) correction are labeled as follows:  $FDR < 0.05 \sim *$ ,  $0.01 \sim **$ ,  $0.001 \sim ***$ ,  $0.0001 \sim ****$ . **b** Overlap between diseases among the alleles used for MPRA. Inflammatory skin disease is marked with an asterisk because it included other inflammatory diseases listed separately, explaining the high overlap with psoriasis. **c** LDSC heritability enrichment of primary epithelial cell and GM12878 ATAC peaks for 9 skin disease studies. Enrichments surpassing both unadjusted (yellow) and FDR-adjusted (red) p-value thresholds ( $<0.1$ ) are highlighted. **d** LDSC heritability enrichment of immune cell ATAC peaks for 2 atopic dermatitis studies.

**Supplementary Fig. 4. MPRA quality metrics and overview. a**, The location of skin disease SNVs in human chromosomes highlights the epidermal differentiation complex and the HLA locus. **b**, Number of variants by disease. **c**, The number of unique reads per MPRA dataset shows no large difference between samples. **d**, The count frequency per barcode for 82,555 barcodes is not overly skewed.  $\mu$ , mean count frequency. **e**, Nearly all fragments had all 10 barcodes observed in the plasmid DNA of the MPRA library. **f**, Nearly all fragments had 9-10 out of 10 barcodes observed in the final RNA library. A similarly high recovery was observed for the CRISPR knock-out MPRA libraries. **g**, Day 3 and 6 correlate better in MPRA data than

with Day 0. **h**, Replicate correlation in MPRA was robust. **i**, The number of active fragments in each condition relative to random controls does not vary greatly. “Safe” denotes cells treated with a safe targeting control guide, while SNAI2/IRF6/NRF1 denote cells treated with guides for the indicated TF. **j**, Enrichment of fragment activity by chromVAR category shows that elements from active TSS are enriched for increased transcriptional activity (color: 2-sided t-test P value). **k**, Enrichment of changes in transcriptional activity across the differentiation time-course by chromVAR category identifies TSS elements as more likely to be dynamic across differentiation, and to be most active at Day 3. **l**, Sample correlation in CRISPR KO-MPRA libraries show IRF6 knock-out Day 3 cells have an undifferentiated phenotype. “Safe” denotes cells treated with a safe targeting control guide.

**Supplementary Fig. 5. rs4687102 is a differentially active allele linked to TP63.** **a**, rs4687102 alters an ATF motif. **b**, rs4687102 resides in an enhancer with increased H3K27ac and looping during differentiation, and is bound by ATF4. The SNV is H3K27ac HiChIP-looped to the promoter of the TP63 gene. **c**, rs4687102 is differentially active in MPRA. **d**, ATF4 binds to the risk allele in CUT&RUN data (*insert*). ASB, allele specific binding. RPM, reads per million.

**Supplementary Fig. 6. SNVs in MPRA that differ in activity by allele.** **a**, Enrichment of differential risk/protective activity by chromVAR category suggests a depletion of differential activity from “strong transcription” regions. **b**, Top outlier daSNVs with the most extreme allelic effects. **c**, Motif activity by JASPAR motif category. “g” denotes Hedge’s g (effect size). P values, two-sided Mann-Whitney for the motif being associated with a change in transcriptional activity. **d**, The atopic dermatitis daSNV rs72696969 is connected via looping to the promoters of FLG and FLG2. **e**, The risk “A” allele of rs72696969 has lower transcriptional activity in MPRA. **f**, HDR edits of primary keratinocytes results in reduced FLG expression for the risk-associated rs72696969 “A” allele. PA2G4 and ERBB3 are nearby control genes. The panel on the right is Sanger sequencing traces of the input and edited populations. Expression was evaluated in progenitor cells. Whiskers, 95% CI. **g**, The rs2349075 and rs2540334 loci, which are eQTLs for CASP8 and are associated with skin cancer. Dotted lines indicate the position of indicated variants. **h**, IRF6 has reduced binding to the risk allele of rs2540334 by microscale thermophoresis. **i**, Differential activity in MPRA of rs2540334 shows reduced activity for the risk variant. **j**, The risk allele of rs2349075 has decreased transcriptional activity in MPRA. **k**, CUT&RUN of JUN and JUNB show decreased binding to the risk allele of rs2349075 in a heterozygous individual. ASB, allele-specific binding event. **l**, JUN, but not the control TF YY1, shows decreased binding to the risk allele of rs2349075 when expressed from the PROBER vector and assayed by proximity biotinylation<sup>3</sup>. In PROBER, a promiscuous biotin ligase “sprayer” is tethered to a vector bearing the protective or risk alleles, then biotinylated proteins are purified and assayed by immunoblot. **m**, Edits to the endogenous rs2349075 locus in primary keratinocytes from a heterozygous individual shows decreased CASP8 expression for the risk allele. CFLAR is a nearby control gene. The panel on the right is Sanger sequencing traces of the input and edited populations. Whiskers, 95% CI.

**Supplementary Fig. 7. CRISPR-flow.** **a**, The correlation of SSC-A and KRT10 staining in CRISPR-flow agrees with prior results that cells enlarge and become more granular across differentiation. **b**, The number of UMIs by sample in CRISPR-flow. **c**, Essential gene guides are depleted from cells. UMIs per million UMIs were summed across all samples, then the plasmid DNA library (“pDNA”) UMI-per-million count is subtracted to normalize to the input

distribution. Essential genes are as defined by DepMap. **d**, Guides targeting DepMap “selective” genes (e.g., genes that reduce growth) and keratinocyte-specific factors were depleted by growth and a subset of TFs were enriched by growth. **e**, Combining high/low enrichment statistics into a single pseudo-P value for an effect leads to a depletion of un-expressed genes (see methods). P values for enrichment or depletion from the KRT10 high or low samples were combined into a pseudo-P value and the effect of different cutoffs (top panel) was evaluated for the depletion of un-expressed genes (middle panel, fraction of the expected number of unexpressed genes included, bottom panel, significance of the depletion of unexpressed genes by Fisher’s exact test). **f**, Calculation of an FDR rate for pseudo-P cutoffs based on the depletion of un-expressed genes as a function of prior belief in the true positive (TP) number. **g**, Diagram of how effects on growth can result in effects on the distribution of guides between progenitor and differentiated cells. **h**, The number of UMIs per gene as a histogram for genes in the given categories. **i**, PCA of guide enrichments reveal a primary axis of promoting/repressing differentiation, and a secondary axis correlated with whether the TF is more essential under differentiation or progenitor growth conditions.

**Supplementary Fig. 8. Overview of perturb-seq experiments.** **a**, Keratinocytes were seeded for differentiation and harvested at the indicated timepoint for perturb-seq. “# usable cells” is the cells used for statistical analysis of effects on the pseudotime distribution (see Methods). **b**, RNA velocity analysis of Day 6 differentiated keratinocytes shows an RNA-velocity derived trajectory matching the pseudotime curve. This UMAP differs due to it being calculated by a different program (scanpy) based on a smaller subset of cells. **c**, Transcription factors are successfully depleted in target cells. The pink distribution represents the distribution of Z scores for expression of the target TF RNA in cells with guides against the target TF. Only TFs in the top half of expression are included, due to the limited sensitivity of single cell RNA-seq. **d-e**, Guide effects on differentiation in perturb-seq as determined by Mann-Whitney tests for altered pseudotime trajectory at day 3 and day 6. **f**, Correlation of differentiation effects between split-pool and 10X-based sequencing.

**Supplementary Fig. 9. Gene set enrichments in homeostasis TFs.** **a**, Enrichment of the 123 homeostasis TFs for TF PPIs, followed by transcription factor networks **b** and co-expression **c**.

**Supplementary Fig. 10. Perturb-seq and inflammation.** **a**, Expression of three keratinocyte inflammation factors in Day 3 UMAPs in 10x data. **b**, Expression of pro-inflammatory IL1 family cytokines IL1A, IL1B and IL18 in Day 3 UMAPs. **c**, Expression of IL1 cytokines across differentiation either in cells with any guide (solid lines) or with ATF4 or HIF1A knock-outs (dashed lines). **d**, RNA velocity and expression analysis of basal marker ITGA6, differentiation marker KRTDAP, IL1 cytokines (IL1A/B, IL18) and the IL1 receptor IL1R2 in Day 6 differentiated keratinocytes. A positive (green) velocity value represents induction, and negative (red) repression. Pseudotime increases left-to-right. **e**, Correlation of IL1 cytokine regulation and differentiation effects for TF knock-outs reveals only a slight negative correlation after regressing out pseudotime effects. **f**, Inflammation effects for TFs controlling for effects on differentiation. **g**, TFs with significant effects on inflammation,  $FDR < 0.2$ . **h**, ATF4 and TFAP2A bind proximal to IL1A/B. ATF4 binding is reduced 2-fold over differentiation, consistent with a loss of activation at the later time points.

**Supplementary Fig. 11 Novel TF validation.** **a**, Target TFs were successfully depleted by siRNAs, as determined by DESeq2 from bulk RNA-seq. **b**, GO analysis of differential genes in bulk RNA-seq, compared to expressed genes. **c**, Number of CUT&RUN peaks FDR<0.01. **d**, CUT&RUN enriched motifs of the novel epidermal TFs and SP1 match known motifs for factors with established motifs (NRF1, SP1), or general sequence preference (CXXC1). The right inset shows the fraction of peaks with the given motif (“Peaks”) or the fraction of length-matched random sequence controls (“Random”) with the given motif, as reported by DREME. **e-f**, Changes in motif location categories across differentiation and their significance (t-test, 2-sided). **g**, Binding at the YWHAZ locus. **h**, TSS peaks and RNA expression changes on siRNA knock-down were correlated for CXXC1 and SP1. The increased significance for the overlap of induced genes with binding suggests an activating effect on balance.

**Supplementary Fig. 12 Homeostasis TF binding.** **a**, Number of FDR<0.05 MACS2 peaks per condition. **b**, Number of CUT&RUN samples per condition. **c**, Mean linear correlation coefficients between replicates for CUT&RUN samples, based on the maximum read depth observed in a random subset of the union peak set. **d-e**, PCA plot of peak heights in CUT&RUN place similar proteins together. The union peak set is determined by a random 200 peak subset of the top 500 peaks for each condition, then those peaks merged across all conditions.

**Supplementary Fig 13 Co-binding analysis.** **a**, fraction of peaks that overlap between the indicated TFs. The overlap is counted as a fraction of the top 2,000 peaks for the given column. Peaks were downsampled to the number of peaks in the smallest peak set (916) so that the denominator was the same for (A & B)/A and (A & B)/B. All except two datasets had >2,000 peaks. **b**, Absolute (non-differential) enrichment of TF motifs in CUT&RUN peaks by Centrimo. Adjusted p values as calculated by Centrimo with default parameters. **c**, Differential (Day 4 vs Day 0) Centrimo analysis<sup>4</sup> of TF motif enrichment in CUT&RUN peaks. Differential enrichment was run for each direction of change (Day 4 vs Day 0 and Day 0 vs Day 4), and the largest absolute value of the log Fisher adjusted p-value for differential enrichment was plotted. If the motif was more enriched in progenitors, the log p value was made negative.

**Supplementary Fig 14 Homeostasis TF peak analysis.** **a**, MACS2 peak significance for specific keratinocyte differentiation genes and progenitor markers for the indicated TFs. **b**, Enrichment in peak locations vs random expressed genes for skin homeostasis genes (skin genes defined by GO). **c**, Enrichment of peaks in skin genes (*left*) and monogenic skin disease genes (*right*), for the indicated region. “Proteins separate” is the average across TFs (P value, two-sided t-test). Proteins combined is the combination of peaks across all TFs, followed by empirical P value determination (one-sided). Whiskers, 95% CI. **d**, As panel **b**, but for monogenic skin disease genes.

**Supplementary Fig 15 Promoters are buffered against allele-specific binding.** **a** Mean  $|\log_2|$  in reads at ASBs as a function of genomic location. P values, two-sided t-tests. Box plot, 25-75% data range; center line, median; whiskers, 1.5 IQR; outliers not plotted. Overlay, mean and 95% CI. **b** A lack of read depth dependence suggests background reads do not create the buffering effect. Left axis and green dots, ratio of  $|\log_2(\text{allele1}/\text{allele2})|$  values (buffering); right axis and black/red dots, mean read numbers in the indicated bin. **c**, ANOVA coefficients for SNV location, regressing  $|\log_2(\text{allele1}/\text{allele2})|$  values on TF, SNV location and read depth. P



values, two-sided t-tests for coefficients  $\neq$  zero. **d**, Kernel density estimate plot of the population frequency of an allele vs the binding ratio (in log2 reads) of the second allele (allele 1 is picked randomly between reference/alternate). Binding correlates with allele frequency with very low correlation ( $R=0.05$ ). Red lines,  $\pm \log_2(0.3)$ ; the region inside the red lines is set to zero for easier visualization. **e** Motif clustering at bound heterozygous sites. Motifs were considered in a cluster if their start positions were 2-50 nucleotides apart. P values for this panel and the following are two-sided Mann-Whitney U tests. This is a boxenplot, depicting the median as center line, 50% of the data in the center two boxes, and each successive set of thinner boxes containing half of the remaining data; values clipped to 10,  $\log_2(N) - 3$  box levels drawn. Overlay, mean and 95% CI. P values for this panel and the following are two-sided Mann-Whitney U tests. **f** Information content at SNV modified bases. P values were  $P > 0.1$  except TFAP2A. Overlay, mean and 95% CI. Box plot parameters are as in panel (a). **g** Fraction of the 141 bp region around bound heterozygous SNVs that are covered by a cognate motif. Box plot, 25-75% data range; center line, median; whiskers, 10-90% data range. Overlay, mean and 95% CI. **h** Fraction of bound heterozygous sites with the SNV in a cognate motif. Whiskers, 95% CI. **i** Fraction of bound heterozygous sites with a modified cognate motif. Whiskers, 95% CI.

**Supplementary Fig. 16 ASBs and TF occupancy.** **a** “HOT” regions have reduced ASB fold changes. “HOT” regions denote regions sensitive to DNase I in epithelial keratinocytes (generally ~10 kbp) with a high number of bound TFs, as defined in by the “wgEncodeUWDukeDnaseNHEK” dataset. Non-HOT regions were DNase I sensitive but without the high number of bound TFs. “Non-DNase regions” were regions not listed in the NHEK DNase sensitivity dataset. Peaks were subset to those with at least 50 reads over the SNV. P values, two-sided Mann-Whitney U. This is a boxenplot, which depicts the median as the center line, and 50% of the data in the center two boxes, with each successive set of thinner boxes containing half of the remaining data. The overlay shows the mean and 95% CI. Box levels were  $\log_2(N) - 3$  for N datapoints. **b** ASB fold changes in promoter SNVs for TFs with at least 50 bound heterozygous sites in promoters correlated with the number of TFs in this dataset bound to the same promoter. That is, the greater the number of different TFs at the promoter, the more equal the number of reads mapping to each allele in a SNV. P values, Spearman rank correlation.

**Supplementary Fig. 17 ASB enrichment near homeostasis genes.** **a**, Putative ASBs are enriched near skin homeostasis genes and monogenic epithelial disease genes. “Proteins separate” is the average across TFs (P value, two-sided t-test, for the average TF’s ASBs being unrelated to the indicated gene group). Proteins combined is the combination of ASBs across all TFs, followed by comparing overlap vs random expressed gene selection and empirical P value determination (one-sided). Whiskers, 95% CI. **b**, Putative ASBs are enriched near monogenic epithelial disease genes. P value is determined from comparing overlap vs random gene selection.

**Supplementary Fig. 18 ASB features** **a**, For skin disease associated SNVs, there is a positive correlation in SP/KLF TF CUT&RUN reads for risk vs protective alleles with transcriptional activity from the same alleles. **b**, Upset plot of  $FDR < 0.01$  daSNV “validation” through eQTL (GTEx), or binding/ASB by a homeostasis TF (this work). Three SNVs were also subject to genomic edits. **c**, Sanger sequencing traces of the rs4704864 input and edited populations.

## Supplementary Methods

### Reagents

| Reagent           | Company      | Cat. #         |
|-------------------|--------------|----------------|
| <b>Antibodies</b> |              |                |
| anti-HA           | CST          | #3724          |
| anti-H3K27me3     | Active Motif | #39055         |
| anti-V5           | CST          | #13202         |
| anti-ATF4         | ProteinTech  | 10835-1-AP     |
| anti-SP1          | ProteinTech  | 21962-1-AP     |
| anti-CXXC1        | ProteinTech  | 27963-1-AP     |
| anti-SP3          | ProteinTech  | 26584-1-AP     |
| anti-KLF4         | Sigma        | HPA002926      |
| anti-KLF4         | Bio-Techne   | AF3640         |
| anti-c-JUN        | Abcam        | ab32137        |
| anti-CREB1        | Abcam        | ab32096        |
| anti-YY1          | Abcam        | ab109228       |
| anti-SNAI2        | CST          | #9585          |
| anti-TFAP2A       | SCBT         | sc-12726/25343 |
| anti-AHR          | SCBT         | sc-133088      |
| anti-TEAD1        | Abcam        | ab133533       |
| anti-JUNB         | SCBT         | sc-8051        |
| anti-OVOL1        | ProteinTech  | 14082-1-AP     |
| anti-TADA2B       | ProteinTech  | 67439-1-Ig     |
| anti-SOX9         | ProteinTech  | 17367-1-AP     |
| anti-GRHL1        | ProteinTech  | 17644-1-AP     |
| anti-P63          | CST          | #4892          |
| anti-KLF5         | ProteinTech  | 21017-1-AP     |
| anti-RUNX1        | ProteinTech  | 25315-1-AP     |
| anti-TEAD3        | CST          | 13224          |
| anti-DLX3         | ProteinTech  | 13261-3-AP     |

| <b>Reagent</b>                         | <b>Company</b>                         | <b>Cat. #</b>   |
|----------------------------------------|----------------------------------------|-----------------|
| anti-ETV3                              | Aviva                                  | ARP85957        |
| anti-KHDRBS1                           | Proteintech                            | 10222-1-AP      |
| anti-HOPX                              | ProteinTech                            | 11419-1-AP      |
| IgG                                    | CST                                    | #2729           |
| IgG                                    | SCBT                                   | sc-2025         |
| anti-KRT10                             | Novus Bio                              | NBP2-47825AF647 |
| <b>Cell Lines</b>                      |                                        |                 |
| Lenti-X 293T                           | Takara Bio                             | #632180         |
| Human primary normal skin cells        | Stanford University School of Medicine | GDS             |
| <b>Reagents and Materials</b>          |                                        |                 |
| anti-FLAG M2 affinity gel              | Millipore                              | A2220           |
| Amicon 3k cutoff columns               | Millipore                              | UFC500396       |
| Stellar Competent Cells                | Takara Bio                             | #636763         |
| DMEM                                   | Gibco                                  | #11995-065      |
| Keratinocyte-SFM                       | Gibco                                  | #17005-042      |
| Medium 154                             | Gibco                                  | #M-154-500      |
| rEGF                                   | Gibco                                  | #10450-013      |
| BPE                                    | Gibco                                  | 13028-014       |
| Pen/Strep                              | Life Technologies                      | 15140-122       |
| Lipofectamine 3000                     | Invitrogen                             | L3000015        |
| Lipofectamine RNAiMax                  | Invitrogen                             | 13778150        |
| Lenti-X concentrator                   | Takara Bio                             | 631231          |
| Monolith His-Tag Labeling Kit          | NanoTemper                             | MO-L018         |
| Monolith NT.115 Capillaries            | NanoTemper                             | MO-K022         |
| RNeasy Plus Kit                        | QIAGEN                                 | 74136           |
| Dynabeads mRNA DIRECT purification kit | ThermoFisher                           | 61011           |
| SuperScript IV                         | ThermoFisher                           | 18090050        |
| AMPure XP beads                        | Beckman Coulter                        | A63880          |
| SYBR green                             | ThermoFisher                           | S7563           |

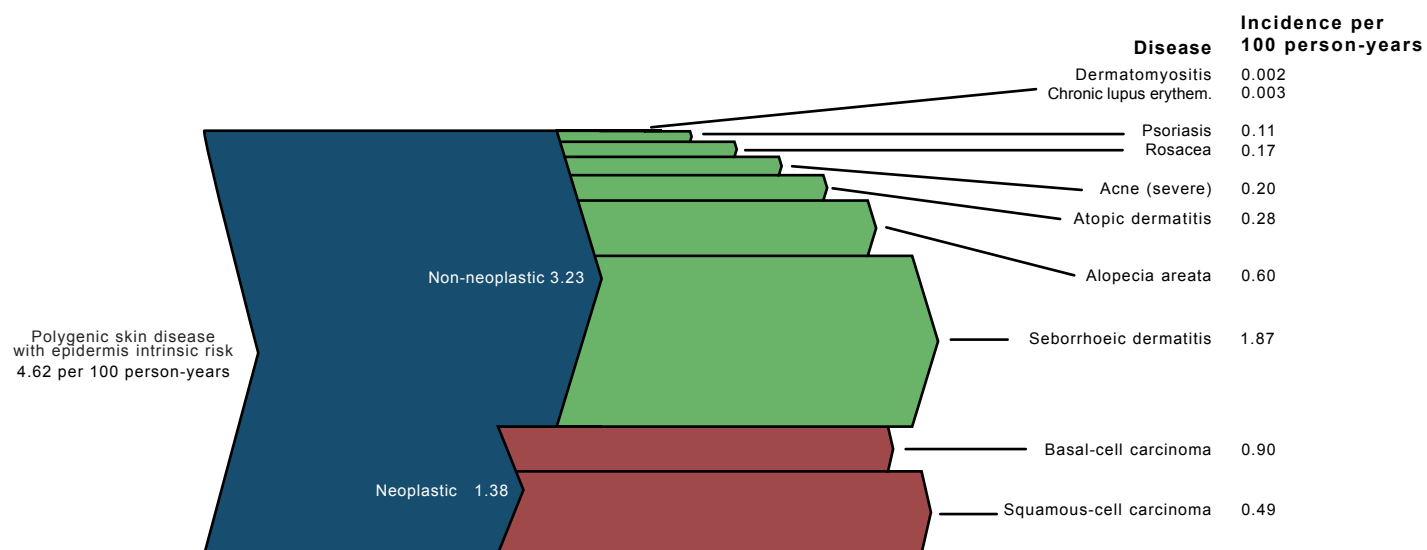
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| Cell Staining Buffer                                  | Biolegend     | #420201      |
| Fix/Perm buffer                                       | Biolegend     | #426803      |
| Perm/Wash buffer                                      | Biolegend     | #426803      |
| RNAse A                                               | Qiagen        | #19101       |
| Proteinase K                                          | ThermoFisher  | AM2546       |
| InstantBlue Protein Stain                             | Abcam         | ab119211     |
| Zymo PCR cleanup kit                                  | Zymo          | D4013/#D4029 |
| QuantSeq 3' mRNA-Seq V2 Library Prep Kit FWD with UDI | Lexogen       | 191.96       |
| UMI Second Strand Synthesis Module for QuantSeq FWD   | Lexogen       | 018.96       |
| Protein G dynabeads                                   | Invitrogen    | 10004D       |
| anti-HA magnetic beads                                | ThermoFisher  | #88837       |
| CUTANA™ ChIC/CUT&RUN Kit                              | EpiCypher     | 14-1048      |
| Concanavalin A beads                                  | EpiCypher     | 21-1401      |
| pAG-MNase                                             | EpiCypher     | 15-1016      |
| Zymo DNA Clean and Concentrator                       | Zymo Research | D4014        |
| NEBNext Ultra II DNA library prep kit                 | NEB           | E7103S       |
| <b>Plasmids</b>                                       |               |              |
| pLEX-FHH-Empty Vector-IRES-Puro                       | Addgene       | #120568      |
| pLEX-FHH-ZNF217                                       | Addgene       | #25648       |
| pLEX-FHH-NRF1                                         | Addgene       | #212207      |

## References

1. Boyle, A. P. *et al.* Annotation of functional variation in personal genomes using RegulomeDB. *Genome Res* **22**, 1790–1797 (2012).
2. Global Burden of Disease Collaborative Network. Global Burden of Disease Study 2021 (GBD 2021). *Institute for Health Metrics and Evaluation (IHME)* (2024).
3. Mondal, S. *et al.* PROBER identifies proteins associated with programmable sequence-specific DNA in living cells. *Nat Methods* **19**, 959–968 (2022).
4. Lesluyes, T., Johnson, J., Machanick, P. & Bailey, T. L. Differential motif enrichment analysis of paired ChIP-seq experiments. *BMC Genomics* **15**, 752 (2014).

Figure S1

Sankey diagram of skin disease incidence per 100 person-years



**Figure S2**

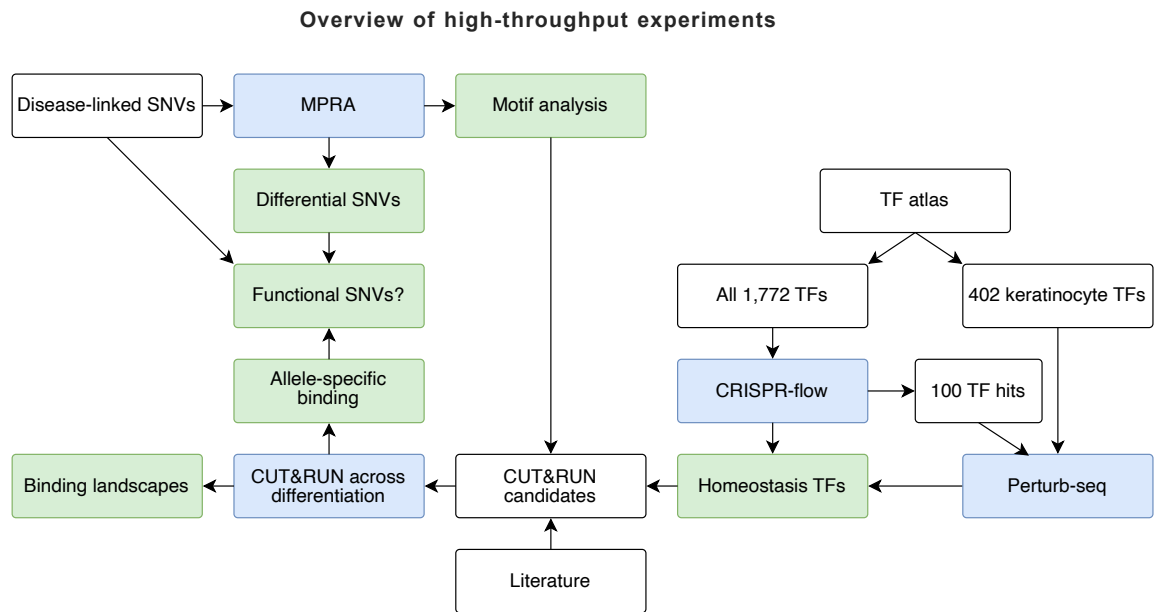


Figure S3

## LD analysis

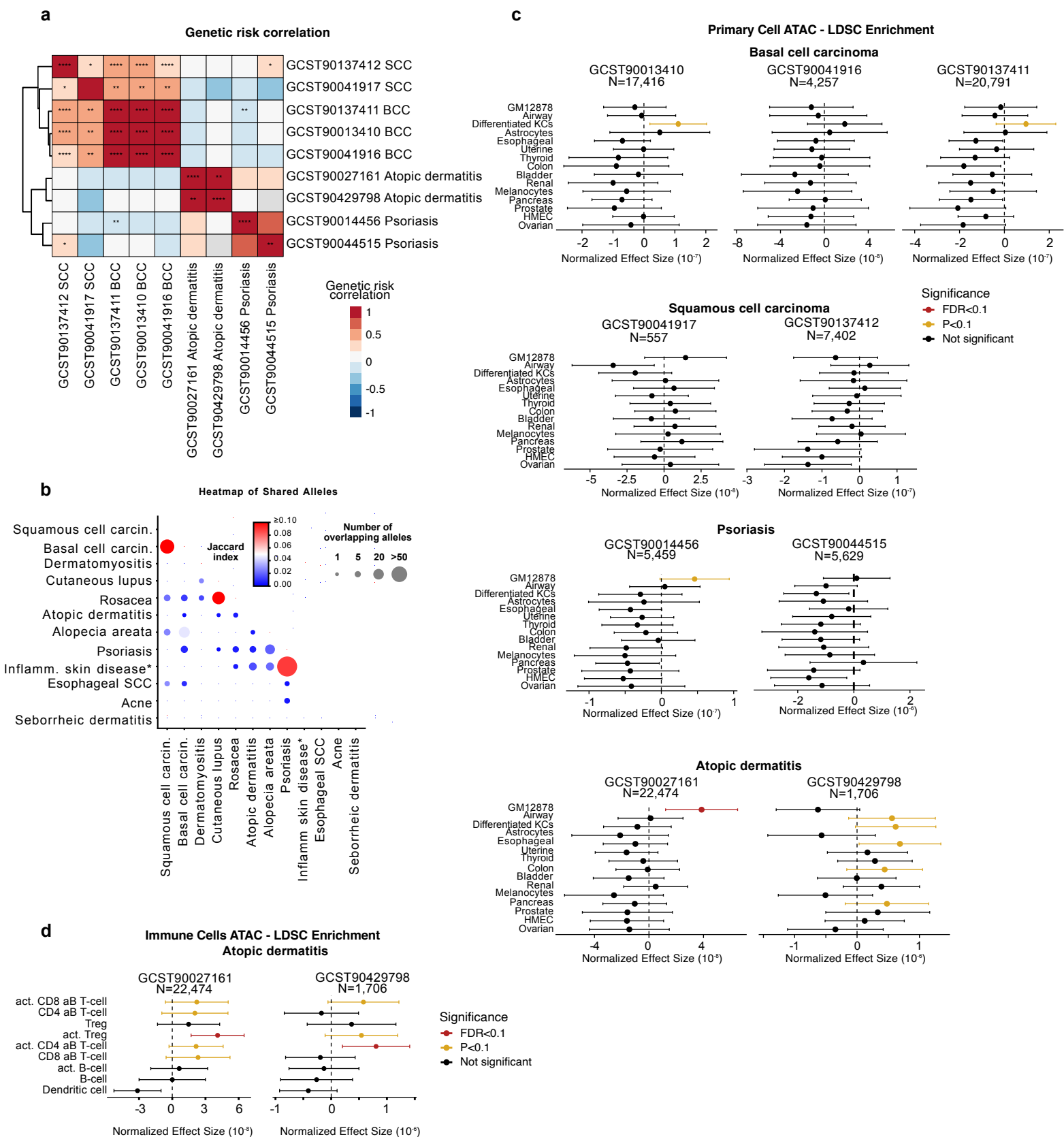
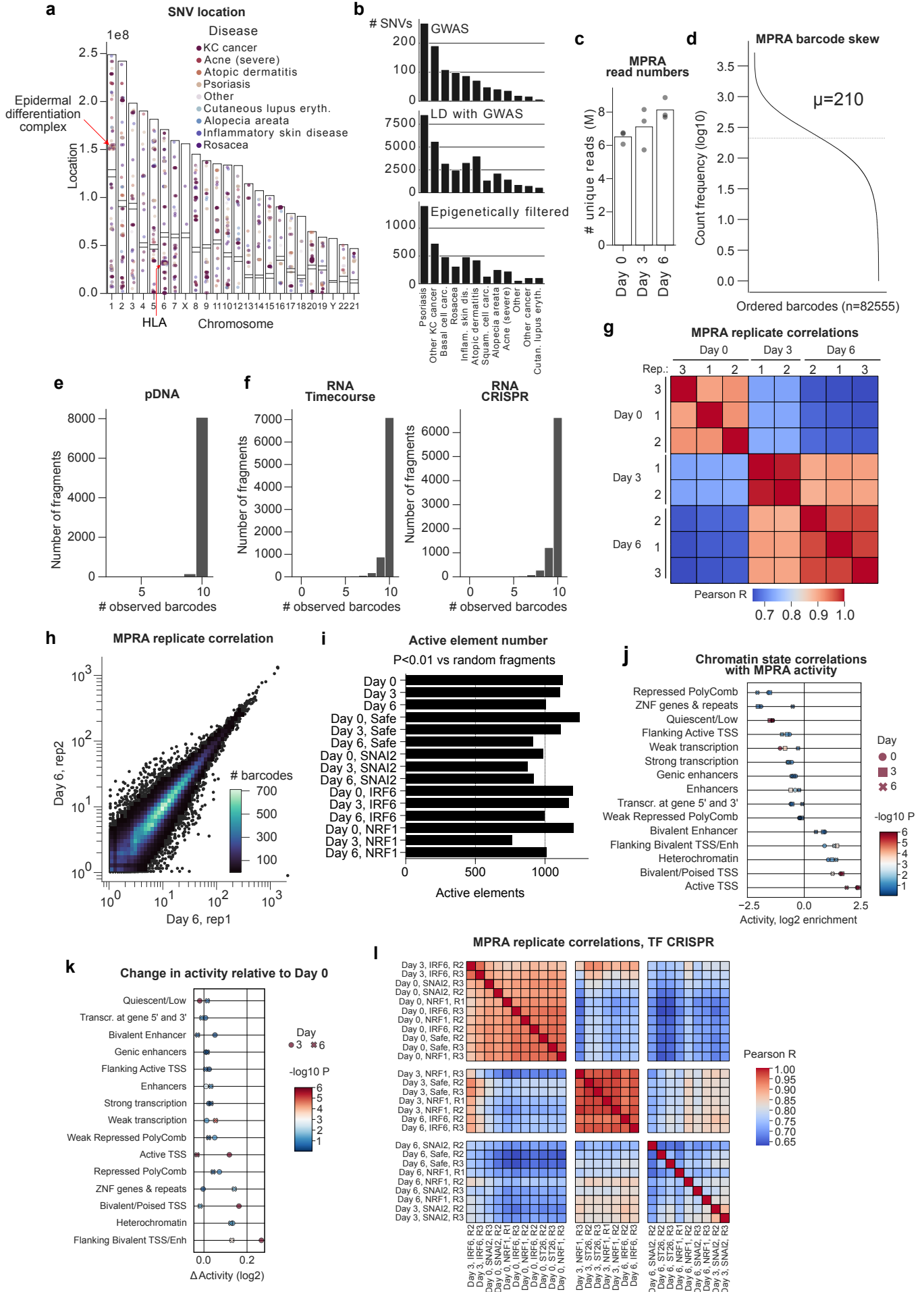


Figure S4

## Skin disease linked SNVs MPRA overview and data quality





**Figure S5**

**rs4687102 is a differential SNV linked to psoriasis and TP63**

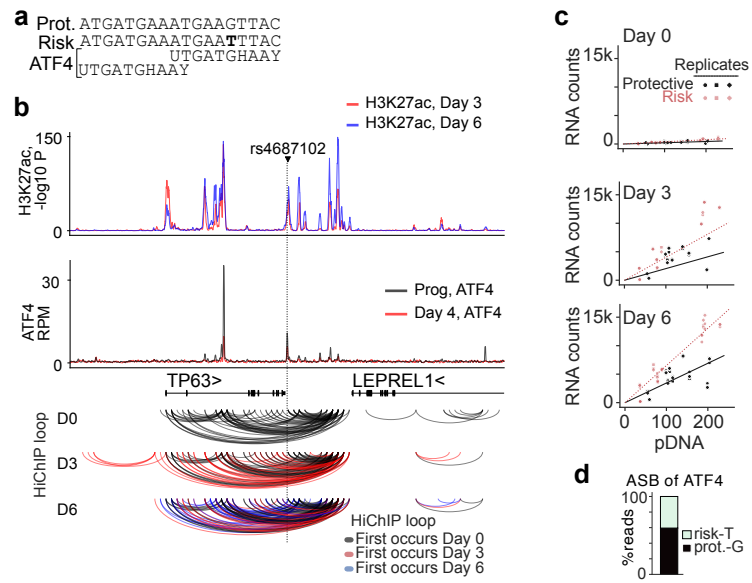


Figure S6

## Differentially active skin disease SNVs

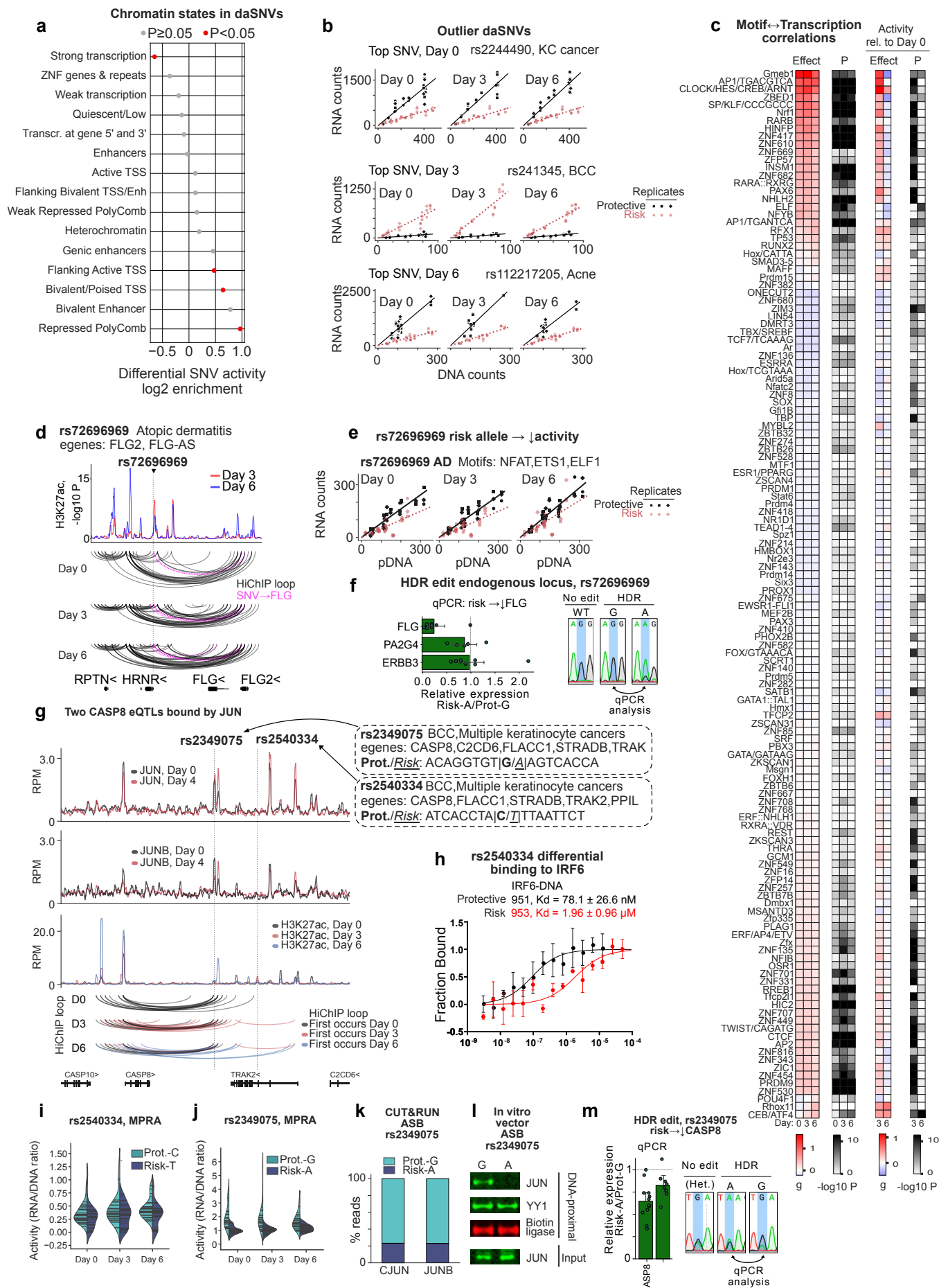


Figure S7

## CRISPR-FLOW of all transcription factors in keratinocytes

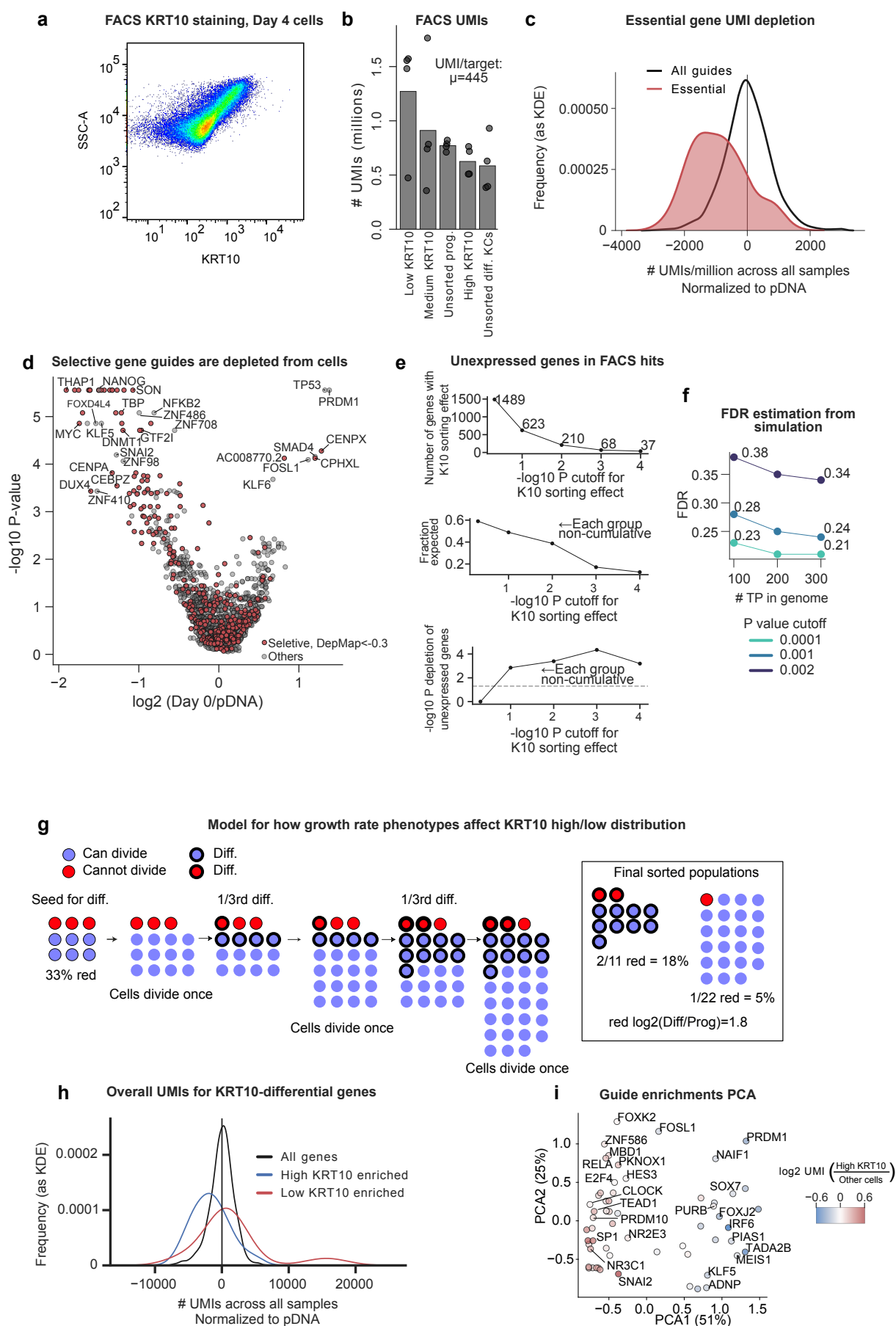


Figure S8

Overview of perturb-seq experiments

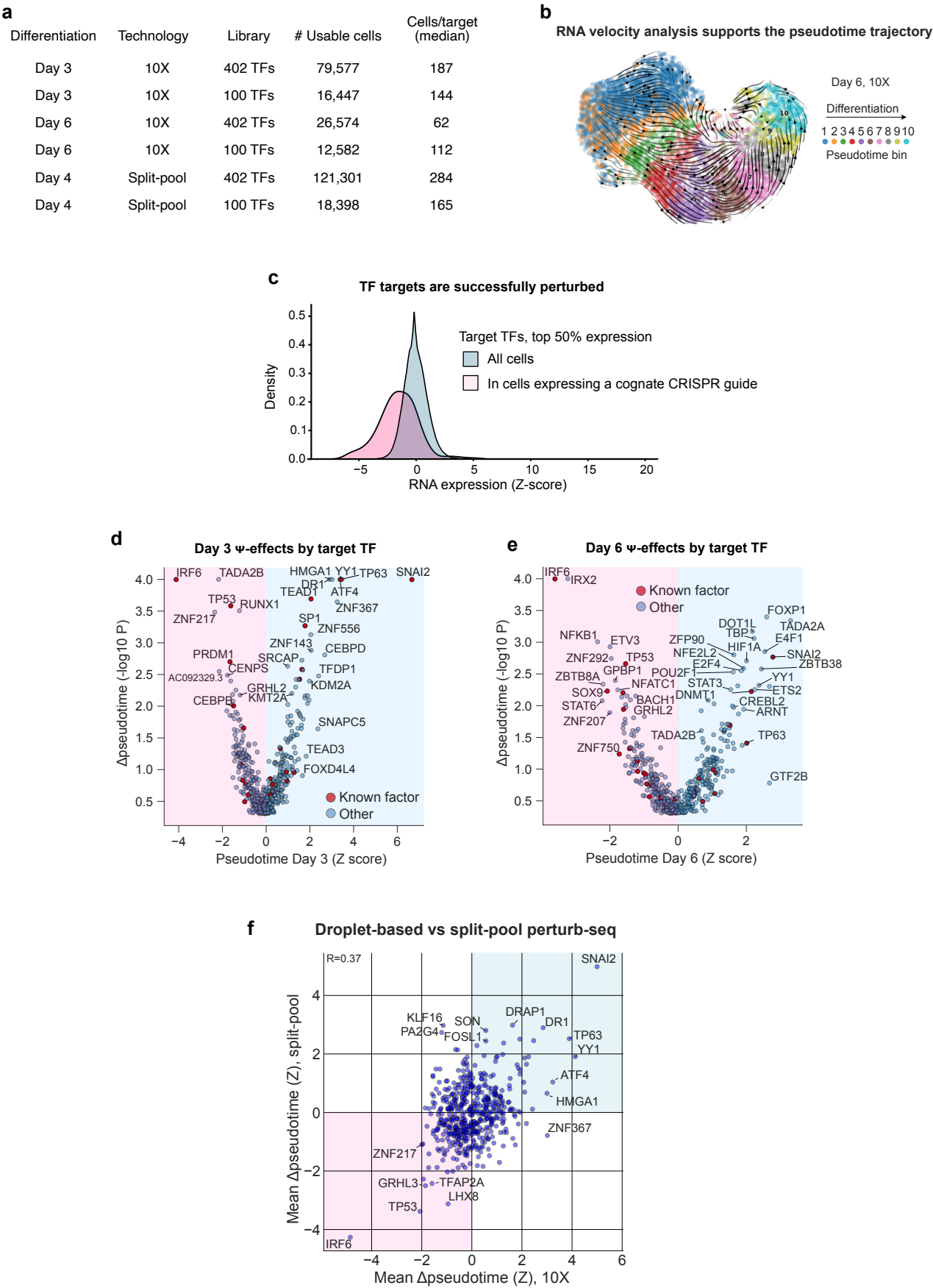


Figure S9

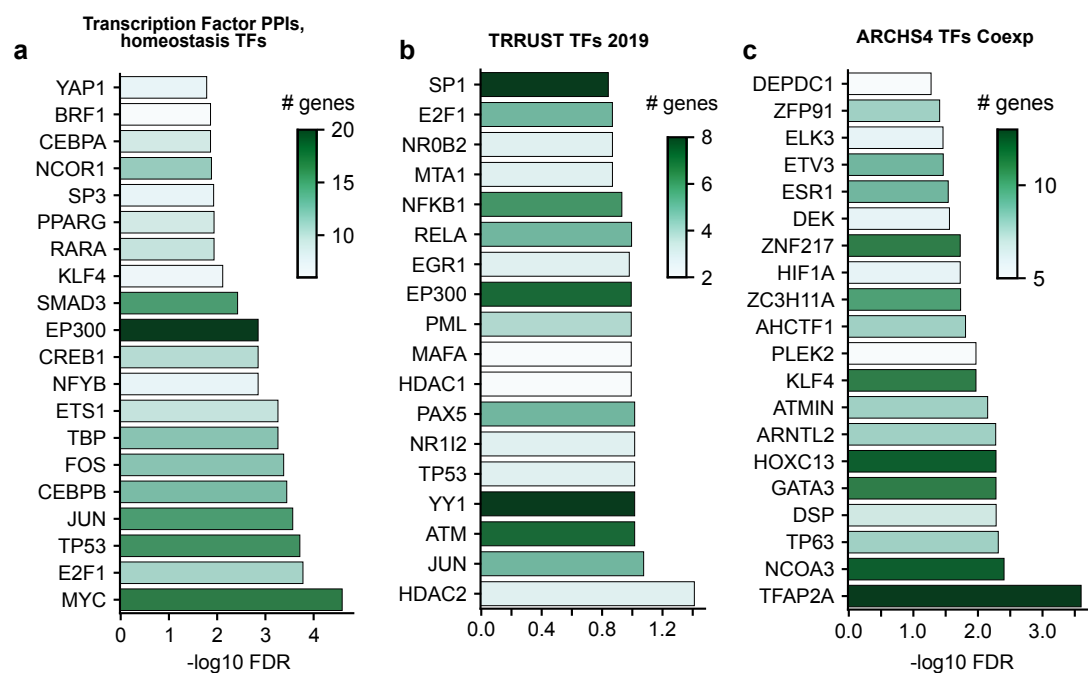
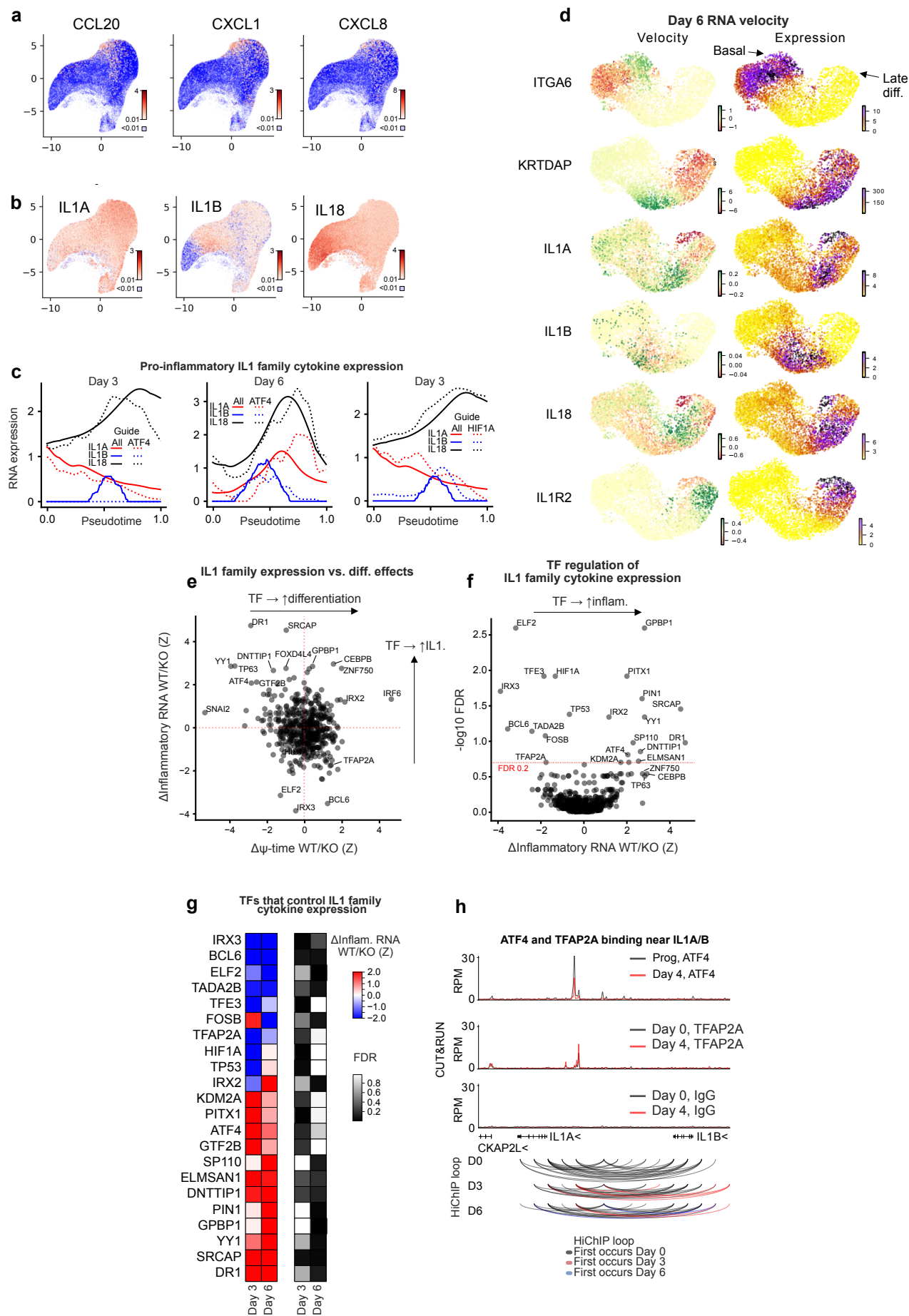
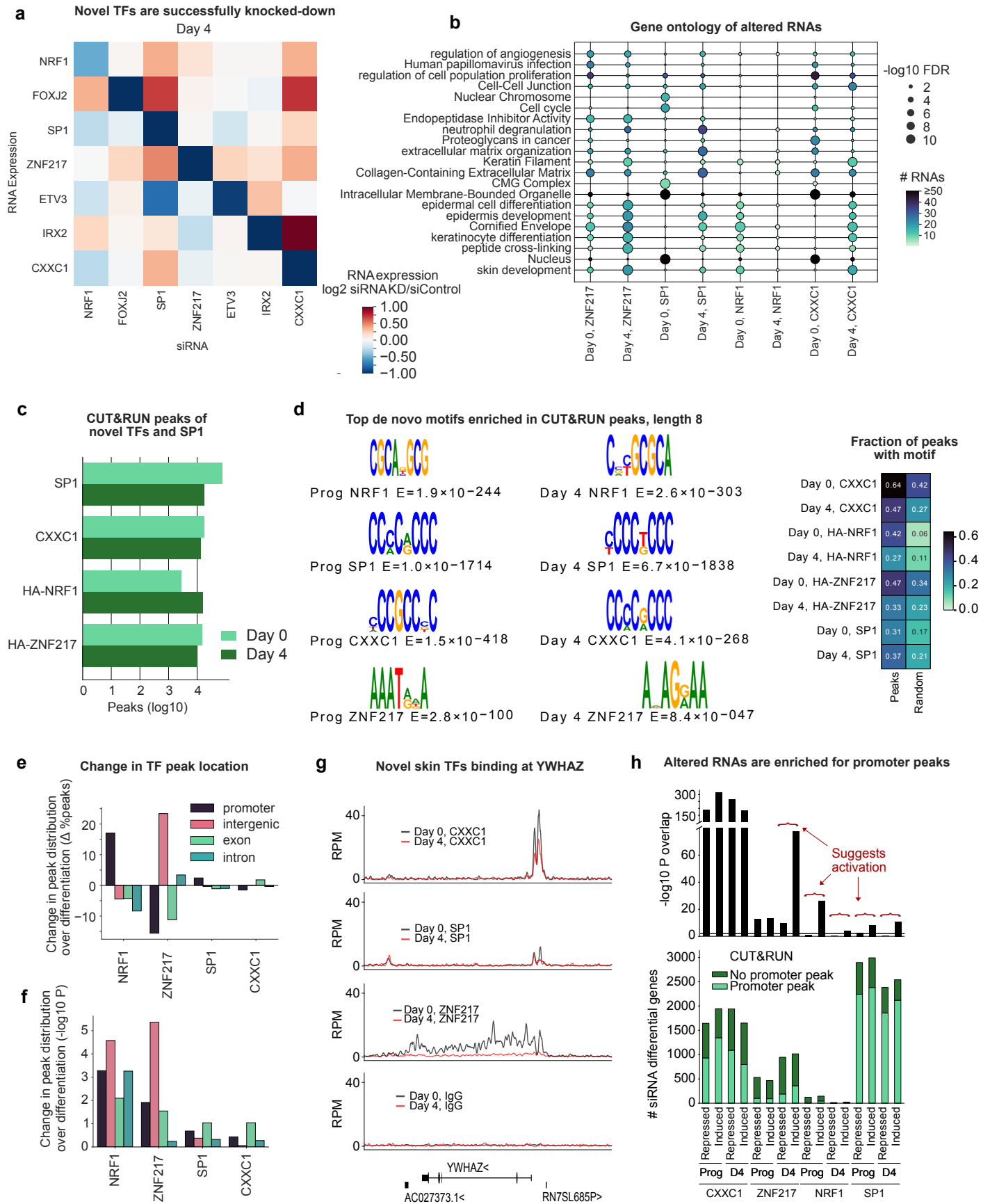


Figure S10





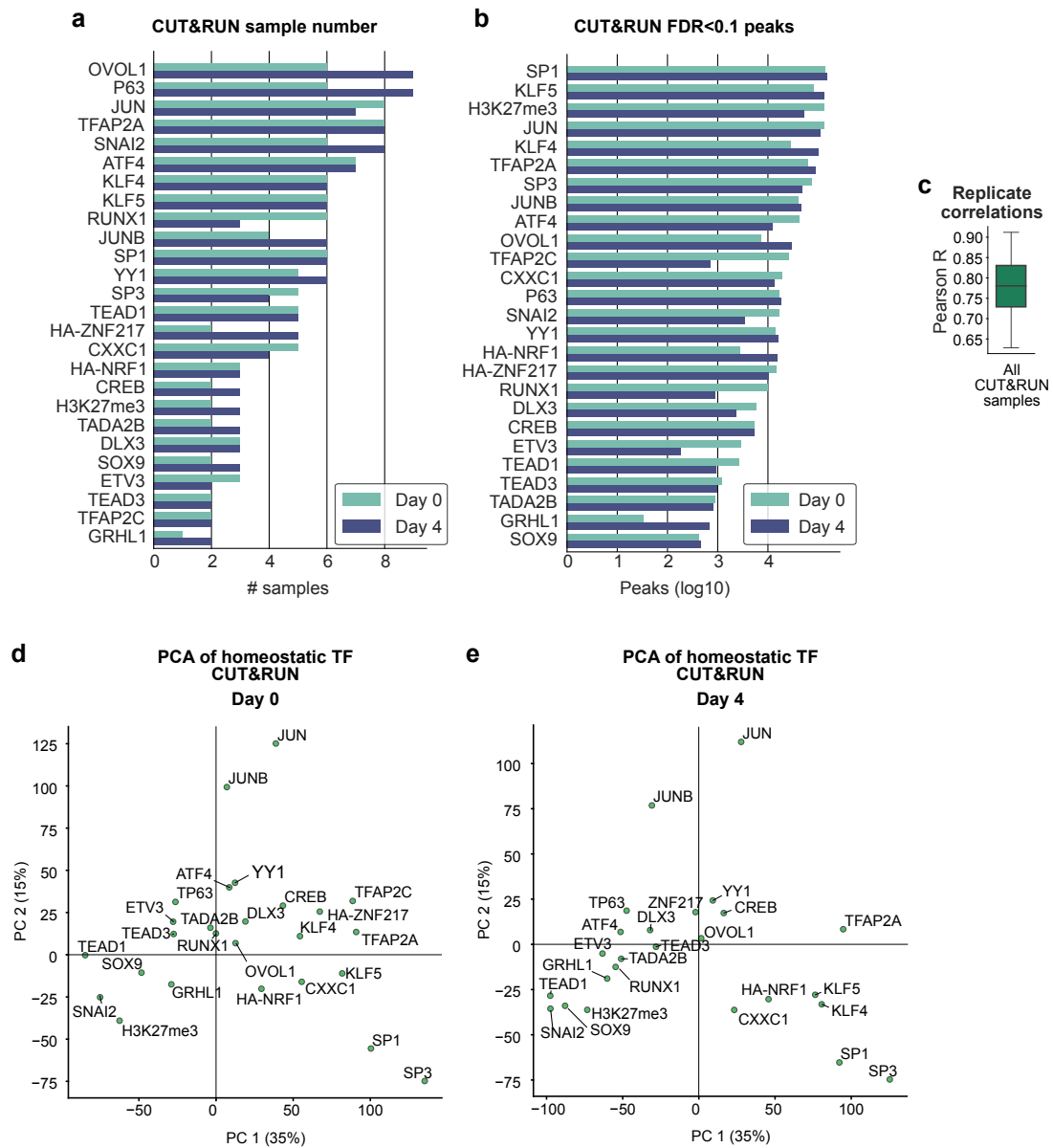




Figure S13

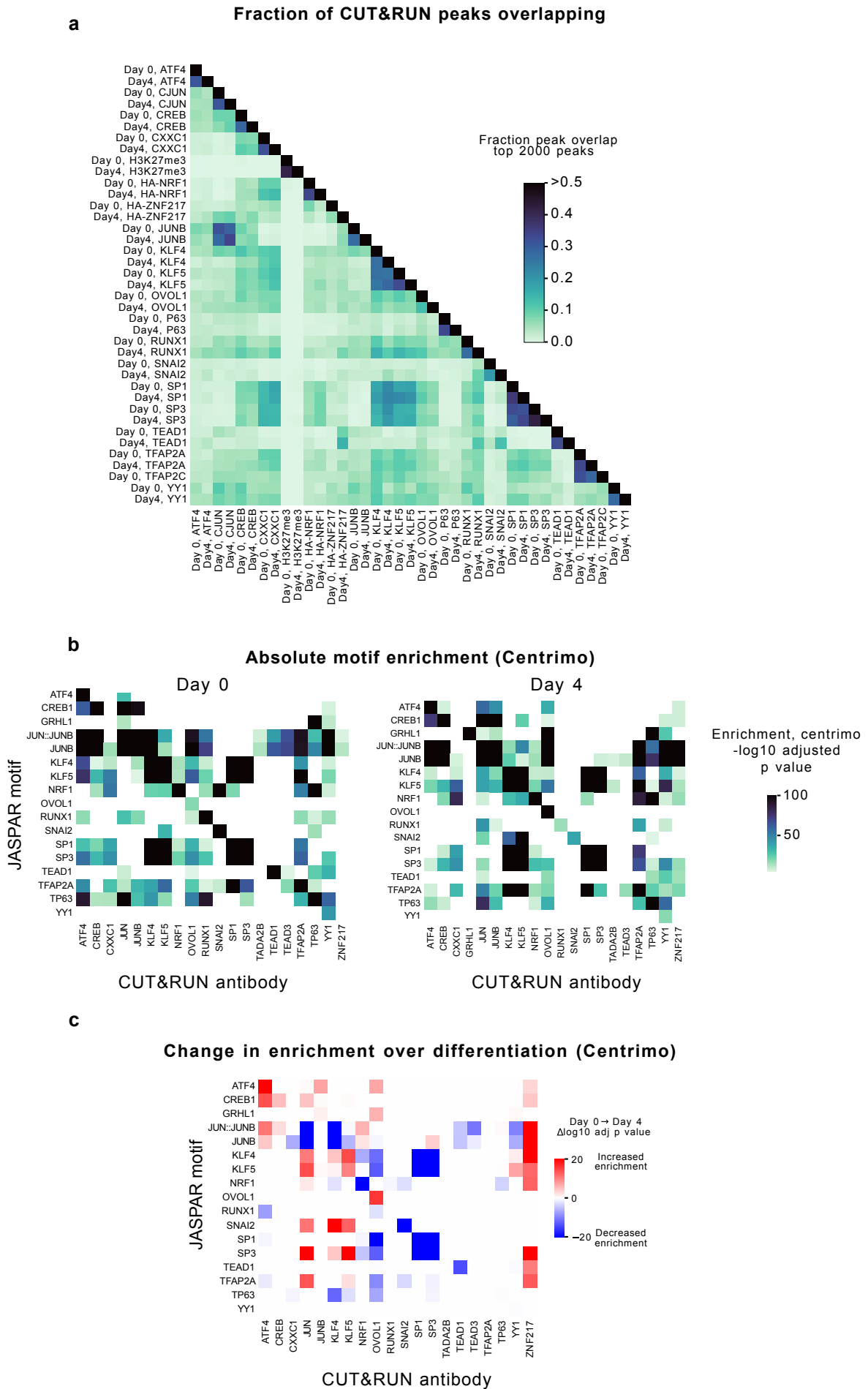


Figure S14

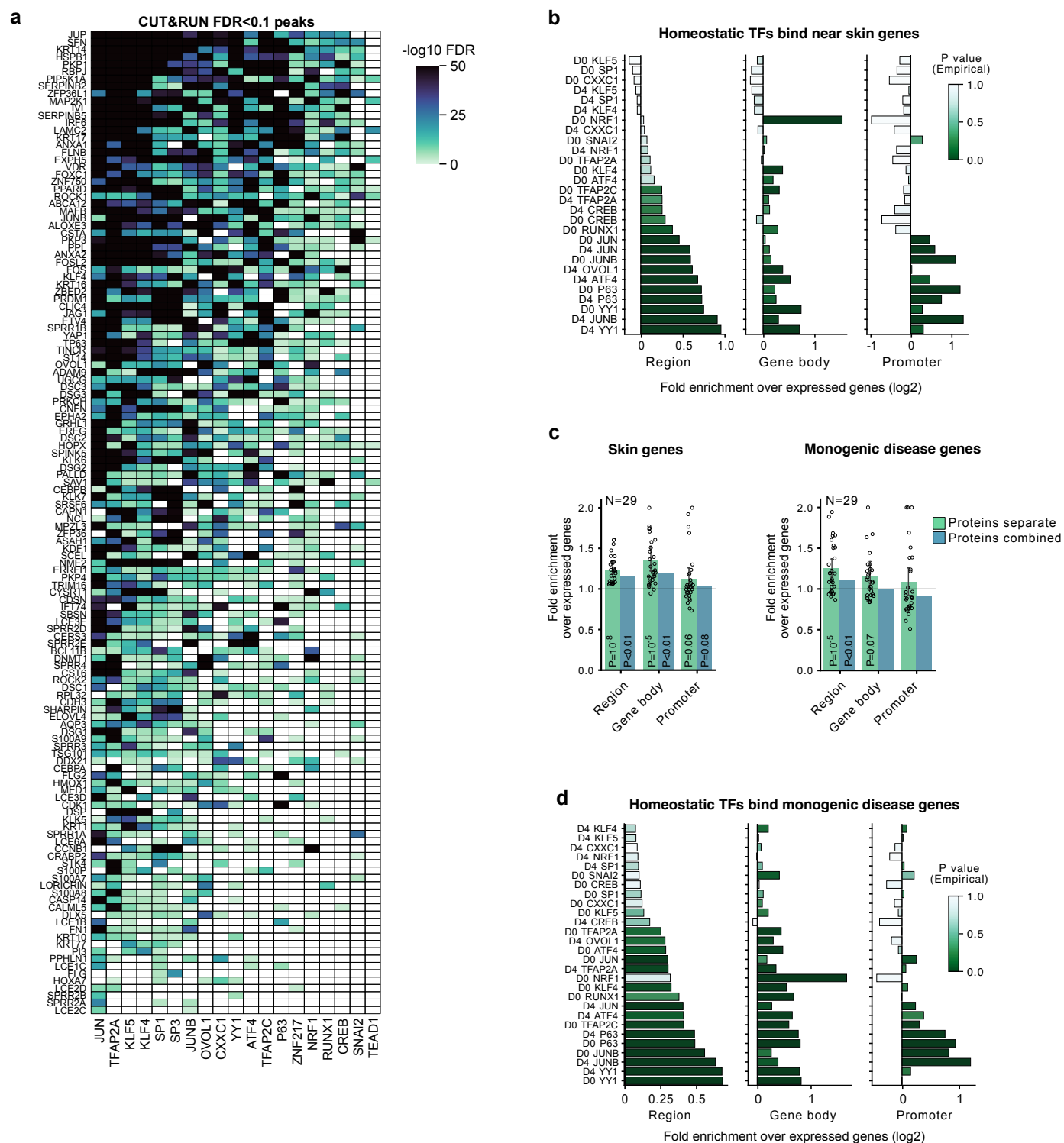


Figure S15

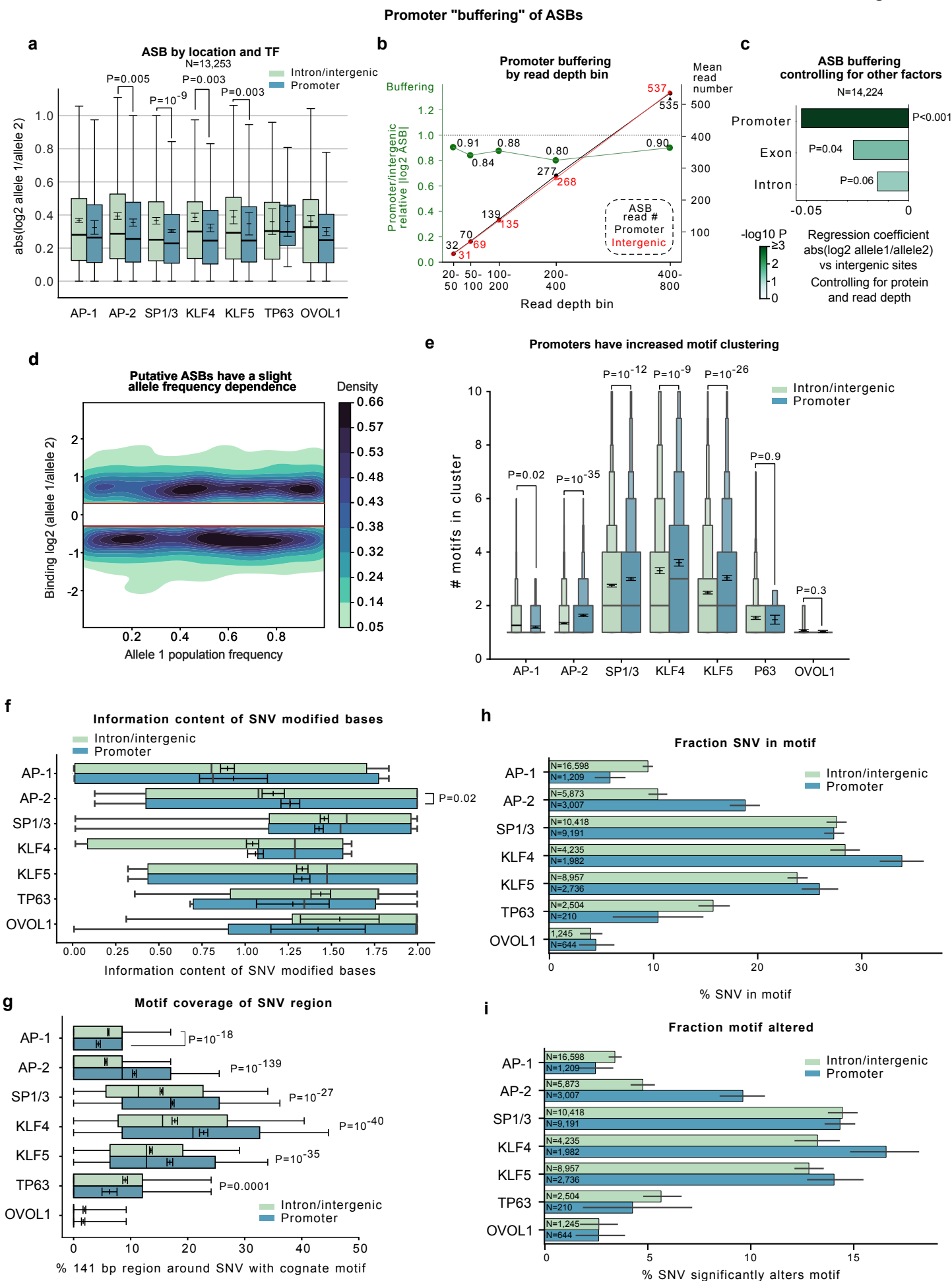


Figure S16

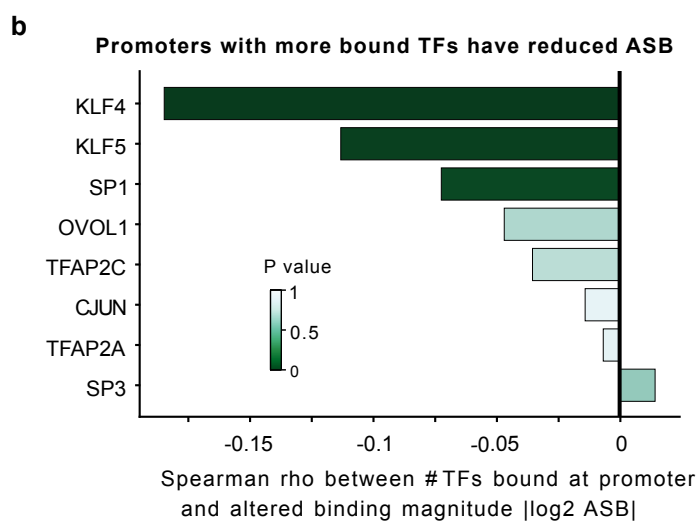
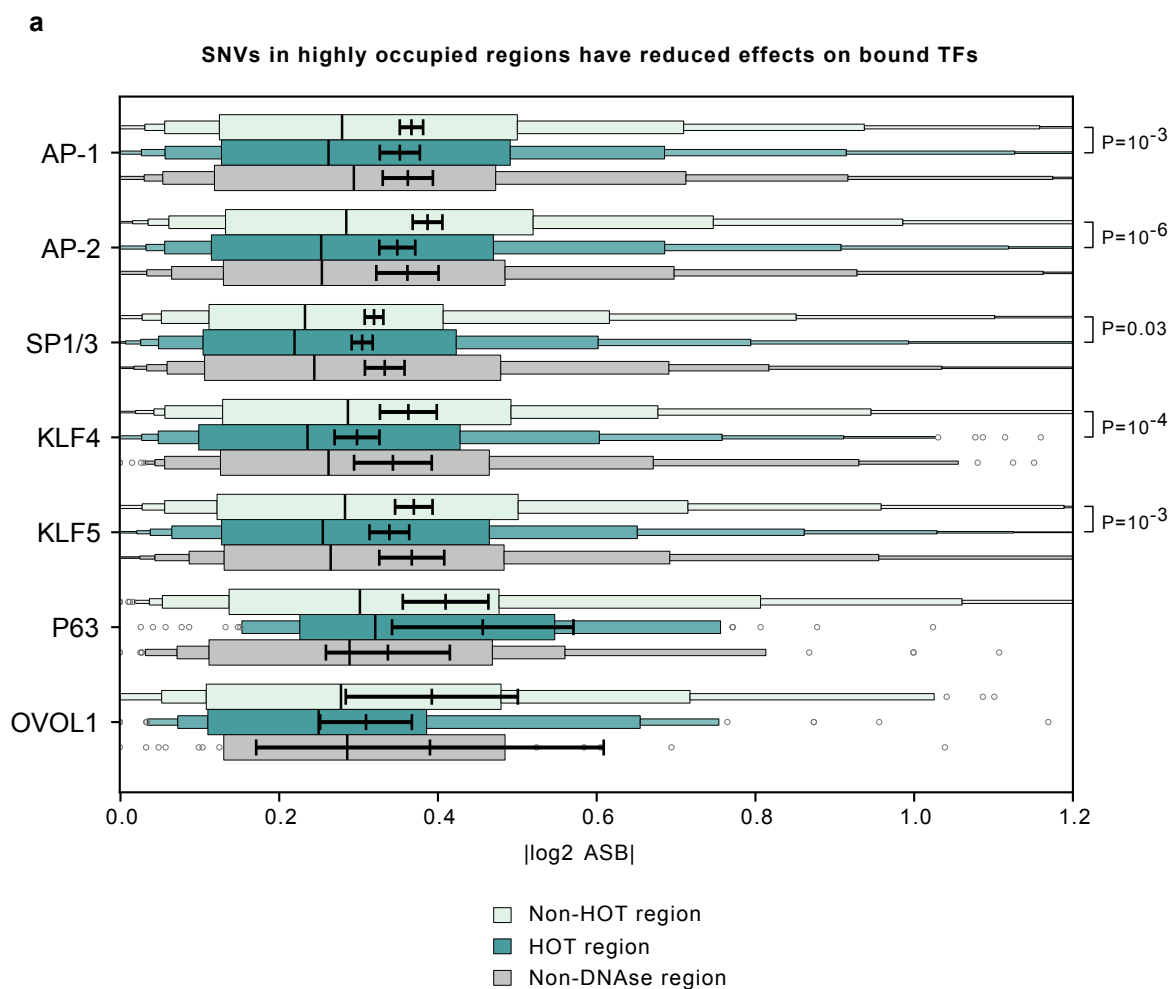


Figure S17

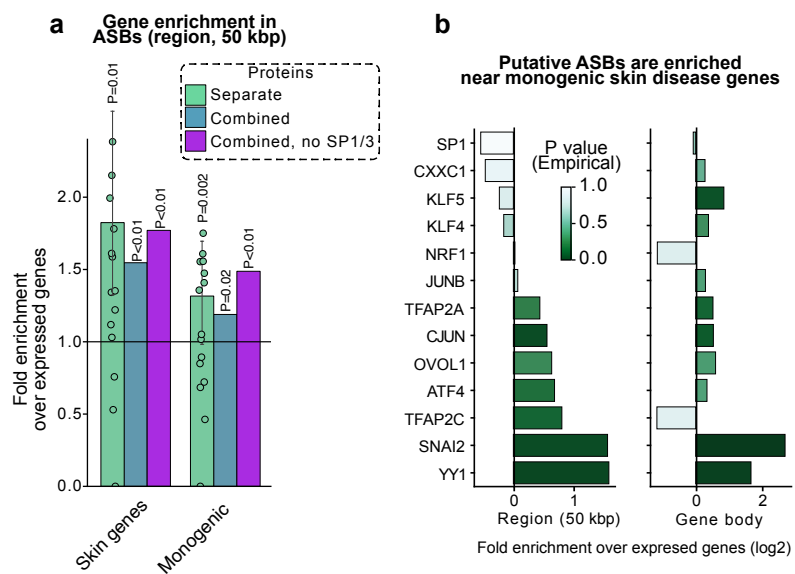


Figure S18

