ARID1A influences HDAC1/BRD4 activity, intrinsic proliferative capacity and breast cancer treatment response

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Using genome-wide clustered regularly interspaced short palindromic repeats (CRISPR) screens to understand endocrine drug resistance, we discovered ARID1A and other SWI/SNF complex components as the factors most critically required for response to two classes of estrogen receptor-alpha (ER) antagonists. In this context, SWI/SNF-specific gene deletion resulted in drug resistance. Unexpectedly, ARID1A was also the top candidate in regard to response to the bromodomain and extraterminal domain inhibitor JQ1, but in the opposite direction, with loss of ARID1A sensitizing breast cancer cells to bromodomain and extraterminal domain inhibition. We show that ARID1A is a repressor that binds chromatin at ER cis-regulatory elements. However, ARID1A elicits repressive activity in an enhancer-specific, but forkhead box A1-dependent and active, ER-independent manner. Deletion of ARID1A resulted in loss of histone deacetylase 1 binding, increased histone 4 lysine acetylation and subsequent BRD4-driven transcription and growth. ARID1A mutations are more frequent in treatment-resistant disease, and our findings provide mechanistic insight into this process while revealing rational treatment strategies for these patients.

Three-quarters of breast cancers are driven by ER1, which uses a slew of associated proteins to access compacted chromatin (including forkhead box A1 (FOXA1) and GATA-binding protein-3 (GATA3))2,3. Drugs that target the ER pathway are effective treatments for a majority of women with ER2 disease4, but a substantial fraction of women will present with de novo or acquired drug resistance. Mechanisms of resistance are varied and include changes in co-factor levels, growth factor-activated transcription and mutations in ER and associated transcription factors and co-factors4.

Substantial effort has been invested in identifying associated protein complexes that influence ER transcriptional activity5,6. A role for the ATP-dependent chromatin remodeling complex/switch mating type/sucrose non-fermenting chromatin remodeling complex (SWI/SNF) has been linked with nuclear receptor function7,8, in which this complex modulates chromatin accessibility. There are three ATPase SWI/SNF complexes—BAF, P-BAF and a recently identified non-canonical BAF (ncBAF)—and the BRG1 and BRM subunits are common among the three complexes. However, there are proteins specific to BAF (ARID1A, ARID1B, DPFI2/3, SS18), P-BAF complex (ARID2, Polybromo (PBRM1), BRD7) and ncBAF (BRD9, GLTSCR1, GLTSCR1)9,10. Previous work has shown a physical association between the SWI/SNF component BRG1 and ER and a requirement for BRG1 for ER-mediated transcriptional activity11,12. The recruitment of SWI/SNF to the ER complex is mediated by shared co-factors13, and BRG1 occupancy at ER regulatory elements coincides with increased localized histone acetylation14. On a locus-specific level, BRG1 can bind to ER regulatory elements independently of ER15, suggesting that the SWI/SNF complex might contribute to chromatin preparation before ER recruitment.

The SWI/SNF complex is important for chromatin regulation and gene expression16; it is mutated in ~20% of all human cancers17 and has been linked with the transcriptional activity of numerous nuclear receptors18,19,20. Wild-type (WT) ARID1A expression is associated with better clinical outcome in ER+ breast cancer patients18 and, importantly, ARID1A-inactivating mutations are enriched in treatment-resistant tumors and metastases (in total, 12% of cases)21. In addition, ARID1A inactivation has been implicated as a tumor-promoting event in ER+ breast cancer22.

To systematically identify genes involved in treatment response in breast cancer, we employed global clustered regularly interspaced short palindromic repeats (CRISPR) screening approaches, coupled with three different treatment modalities, revealing a role for the SWI/SNF complex as a critical determinant of treatment response.

Results
CRISPR screening reveals ARID1A as a gene involved in treatment response. We employed a CRISPR screening approach,
which encompassed guide RNAs targeting a total of 18,009 human genes. We established Cas9-expressing MCF-7 breast cancer cells (Supplementary Fig. 1), which were infected and grown for 20 d. All cell line experiments were conducted in asynchronous cells grown in estrogen-rich media. Three biological cell cultures of independent viral infections with CRISPR vectors were performed as described in Methods. Analysis of depleted gRNAs at different post-infection time points revealed known ER interactors, including Cyclin D1 (CCND1), FOXA1 and GATA3 (Fig. 1a, Supplementary Fig. 2 and Supplementary Table 1), albeit with different essentiality kinetics (Fig. 1a,b). In addition, a number of gRNAs were enriched representing tumor suppressors or growth inhibitors (Fig. 1c). As expected, growth-promoting genes required for cellular viability showed greater gRNA depletion with longer infection (Fig. 1d). After 9 d of infection, we subsequently treated cells for a total of 26 d with the selective estrogen receptor modulator 4-hydroxytamoxifen (tamoxifen) or the selective estrogen receptor degrader fulvestrant (ICI 182780). We also used the tool compound JQ1, which targets bromodomains and extraterminal domain (BET)-containing proteins since bromodomain-containing protein-4 (BRD4) is postulated to be a therapeutic target in ER+ breast cancers and BET inhibitors are currently being explored in clinical trials. Three independent infections were performed, and the data were integrated as described in Methods (complete data given in Supplementary Table 2). When specifically assessing genes required for treatment response, we found that the fulvestrant and tamoxifen CRISPR screens looked largely similar (Fig. 1e). Despite the distinct mechanisms of growth suppression (fulvestrant degrades ER whereas tamoxifen-bound ER is recruited to chromatin as a repressive complex), 63.5% of the genes required for the antiproliferative effects of fulvestrant were also required for tamoxifen activity (Fig. 1e and Supplementary Fig. 2). Among the most significantly enriched genes was AT-rich interaction domain 1A (ARID1A), a component of the BAFCP-dependent chromatin remodeling complex. It is among the genes found most essential for both tamoxifen and fulvestrant activity, and depletion of ARID1A (that is, enrichment of gRNAs targeting ARID1A) resulted in drug resistance to both compounds. Unexpectedly, ARID1A was the highest-ranked gene in JQ1-treated cells (ranked 1 out of 18,009), but in the opposite direction; gRNAs were observed to be depleted in JQ1-treated conditions (Fig. 1f,g and Supplementary Fig. 2). Other BAF components, including ARID1B, SWI/SNF-related, matrix-associated, actin-dependent regulator of chromatin, Subfamily B, Member-1 (SMARCB1/BAF47/SNF5) and synovial sarcoma translocation, chromosome 18 (SS18), showed the same pattern (Fig. 1f,g and Extended Data Fig. 1), suggesting that the BAF complex is required for ER-targeted drugs to work but, when it is lost, cells are sensitized to BET inhibitors. The dependence on ARID1A for growth arrest mediated by ER-targeted agents was validated in MCF-7 and ZR-75-1 cells using ARID1A small interfering RNA (Extended Data Fig. 1, Source Data Fig. 1 and Supplementary Fig. 3).

**Genomic characterization of ARID1A function.** We subsequently assessed the potential genomic interplay between ARID1A and ER. We performed three independent biological replicates of chromatin immunoprecipitation sequencing (ChIP-seq) for ARID1A in MCF-7 and ZR-75-1 cells, and peaks were called using MACS v2 (ref. 23), resulting in 21,226 ARID1A peaks in MCF-7 and 56,966 in ZR-75-1. ARID1A binding sites were found commonly to co-occur at ER and FOXA1 binding events (Fig. 1h and Extended Data Fig. 2), and global analysis revealed that >78% of all ARID1A binding events were shared with ER, FOXA1, or both, in MCF-7 (Fig. 1i), implying a functional connection between ARID1A and regulatory elements occupied by the ER/FOXA1 complex. Interestingly, ARID1A overlapped more with FOXA1 (78% of ARID1A binding sites was co-bound by FOXA1) than with ER (66%) in ZR-75-1 cells (Extended Data Fig. 2). We assessed whether ARID1A binding to ER-bound enhancers was dependent on ER by hormone-depriving cells, by treating with vehicle (ethanol) or estrogen for 6 h and conducting ChIP-seq. ARID1A was able to bind to ER/FOXA1 binding events before ligand-induced ER recruitment (Fig. 1j and Extended Data Fig. 2). These findings suggest that ARID1A is not a classic ER-associated co-factor and can bind to regulatory elements independent of active ER, potentially in a repressive manner.

To validate the CRISPR screen, we specifically deleted ARID1A from MCF-7 cells, thus resulting in two separate ARID1A knockout clones (clones 11 and 14). ARID1A deletion was confirmed by Sanger- and amplicon-based next generation sequencing and immunoblotting (Fig. 2a, Supplementary Fig. 4 and Source Data Fig. 2), and potential off-target effects were assessed. In vitro growth of these clones and the WT control (WT clone 219) validated the CRISPR screening results, showing that both clones had increased intrinsic proliferation and were resistant to tamoxifen but showed sensitivity to JQ1 (Fig. 2b and Supplementary Fig. 5) and two additional clinically relevant BET inhibitors, OTX015 (from OncoEthix/Merck) and IBET762 (from GlaxoSmithKline) (Supplementary Fig. 4).

We established xenograft tumors from either the WT or the two ARID1A knockout clones in the presence of estrogen pellets to maintain ER+ tumor growth, and subsequently treated cells with either vehicle or 4-hydroxytamoxifen. Tumor growth at day 25 was increased in both ARID1A knockout clones in the presence of 4-hydroxytamoxifen compared to WT (Supplementary Fig. 5, which includes details of statistical tests), validating that ARID1A is required for anti-estrogen efficacy. However, the greatest difference in growth rate was seen in ARID1A WT versus knockout contexts (Fig. 2c and Supplementary Fig. 5), and we postulated that the diminution of tamoxifen efficacy in ARID1A-null tumors may simply be due to an increased overall intrinsic proliferative potential.

**ARID1A regulates ER target genes and is part of the ER complex.** To explore the mechanistic role of ARID1A in drug response, RNA sequencing (RNA-seq) was conducted using four biological cell culture samples of the WT or ARID1A knockout lines treated with vehicle, fulvestrant, 4-hydroxytamoxifen or BETi (JQ1). Gene-expression analysis of the ARID1A knockout clones and controls revealed several findings. The control lines looked similar, regardless of whether they were parental cells or WT clones (Supplementary Fig. 8). Whereas fulvestrant and tamoxifen showed similar gene repression patterns, JQ1 treatment resulted in a substantially different gene-expression profile (Fig. 2d and Supplementary Fig. 8). In the ARID1A knockout clones, JQ1 treatment showed a more consistent expression pattern than that in WT cells, whereas the majority of genes repressed by fulvestrant/tamoxifen were either upregulated or unchanged in the ARID1A knockout cells (Fig. 2d and Supplementary Fig. 6). In total, 86% of the fulvestrant- and 85% of the tamoxifen-repressed genes were no longer significantly repressed in the ARID1A knockout cells, and a cluster of them (highlighted in Fig. 2d) were significantly downregulated by JQ1 treatment, to the same degree as in WT cells. ARID1A deletion, therefore, resulted in induction of fulvestrant/tamoxifen-repressed genes even in the absence of an ER antagonist, implying ARID1A-mediated basal repression of the ER target genes. We generated a gene signature from the RNA-seq data and could show in the Molecular Taxonomy of Breast Cancer International Consortium (METABRIC) cohort of ER+ breast cancer patients that ARID1A-repressed genes, under both vehicle and anti-estrogen conditions (those that were upregulated in the ARID1A knockout cell lines), were associated with poor clinical outcome when upregulated in patients (Fig. 2e and Supplementary Fig. 6; the latter includes details of the statistical tests), again supporting the notion that ARID1A can repress genes linked with clinical outcome.
Fig. 1 | CRISPR screens reveal ARID1A and BAF components as essential genes for treatment response. a–c, log2 fold of gRNA counts changes as a function of time per gene (red lines) and on averages (black line), based on a sample size of n = 3 for three categories of genes; those showing rapid growth depletion (a), those showing longer-term growth depletion (b) and those showing increased proliferation (c). For each category, example genes are shown in red and ARID1A in blue. d, Heatmap representing log2 change of significant genes (n = 1,915) under nontreated conditions (days 3–20 (D3–20) of infection compared to uninfected gRNA pool). Rows were ordered according to hierarchical clustering. e, Heatmap representing log2 fold change of genes after 26 d of treatment with fulvestrant (Fulv, initiated with 300 nM and reduced gradually to 100 nM), 100 nM 4-hydroxytamoxifen (Tamox) or BETi (JQ1, 1 μM reduced to 250 nM) compared to DMSO treatment (DMSO control after D9 of infection). Rows were ordered according to hierarchical clustering. f, ARID1A and other BAF components were enriched, but in different directions depending on the specific drug treatment. Values show changes in gRNA counts from CRISPR screening. gRNA counts from CRISPR screening

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To understand the mechanism behind ARID1A regulation of innate proliferation, we used an unbiased proteomic approach, rapid immunoprecipitation mass spectrometry of endogenous interactions (RIME), combined with a label-free quantification method24, to identity interactors of ARID1A, BRG1 or ER from asynchronous MCF-7 cells, using an IgG pulldown as a negative control (information is provided in Supplementary Fig. 7). ARID1A and BRG1 purification revealed almost all the known BAF components, as well
as ER and, similarly, the ER RIME contained ARID1A and BRG1 in the complex (Fig. 3a, Extended Data Fig. 3 and Supplementary Tables 3 and 4). The other BAF complexes, P-BAF and ncBAF, were identified in BRG1 pulldown but not in ARID1A pulldown. BRG1 RIME identified all BAF, P-BAF and ncBAF components, validating that BRG1 is common to these complexes. It also showed enrichment of GLTSCR1/GLTSCR1L (BICRA/BICRL) subunits. BRG1 RIME revealed BET proteins as interactors (data not shown). We expanded on these observations by re-analyzing our recently published ER quantitative multiplexed RIME (qPLEX-RIME) data on five ER+ primary tumor samples from different patients. We discovered ARID1A and several SWI/SNF components, including BRG1, BRM, BAF57, BAF170 and BAF155, as physical interactors of ER, even in surgical tumor tissue (Fig. 3b and Extended Data Fig. 3). Importantly, we also observed an interaction between ER and BRD4, a target of BETi, in patient tumor material (Fig. 3b), verifying physical associations among endogenous ER, the SWI/SNF complex and BRD4 in vivo. We re-analyzed our previous proteomic data to identify proteins that interact with tamoxifen-bound ER. ARID1A, BRG1 and a number of additional SWI/SNF components were enriched with tamoxifen-ligated ER complex after treatment with 4-hydroxytamoxifen for 6h (Fig. 3c and Extended Data Fig. 3), confirming that SWI/SNF–ER complex formation is repressive.

To explore the putative functional connection between SWI/SNF and the ER complex, we conducted a series of ChIP-seq experiments to map binding sites for ARID1A and two SWI/SNF common proteins, BRG1 and SNF5 (BAF47), in estrogen-rich asynchronous MCF-7 cells treated with either control or 4-hydroxytamoxifen for 6h. Three independent biological replicates were conducted. Binding of all three proteins was increased globally following 4-hydroxytamoxifen treatment (Fig. 3d and Supplementary Fig. 11), supporting the hypothesis that they are involved in drug responsiveness. Both induced BRG1 and SNF5 sites overlapped with induced ARID1A sites, ER and FOXA1 (Fig. 3d and Supplementary Fig. 8). Unexpectedly, binding of these proteins was also increased following fulvestrant treatment (Extended Data Fig. 4). The fulvestrant-induced sites overlapped with both the tamoxifen-gained sites and estrogen-lost sites from Fig. 1j (Extended Data Fig. 4), implying that these are the consistent hormone-regulated SWI/SNF binding regions. Altogether, our findings suggest that during the recruitment of these factors, while they are able to associate with the ER complex, they can bind to chromatin in an ER-independent manner, in support of data showing basal repression of ER target genes by the BAF complex (Fig. 2d,e). FOXA1 recruits ARID1A to chromatin. Because fulvestrant and tamoxifen both increased BAF binding to chromatin, we speculated that the pioneer factor FOXA1 might modulate ARID1A and BRG1 recruitment to chromatin, as supported by data showing considerable overlap between ARID1A and FOXA1 binding (Fig. 1i and Extended Data Fig. 2). MCF-7 and ZR-75-1 cells were hormone-deprived and transfected with FOXA1 or control siRNA, and ChIP-seq of ARID1A or BRG1 was performed. Binding of both ARID1A and BRG1 was substantially reduced following FOXA1 silencing at enhancers, in both cell lines assessed (Fig. 4a–c, Extended Data Figs. 5 and 6 and Supplementary Figs. 9 and 10), suggesting a degree of dependence on the pioneer factor FOXA1 for SWI/SNF recruitment. Importantly, the FOXA1-dependent ARID1A binding sites were the same regions where tamoxifen induced ARID1A binding to the genome (Fig. 4f.g). To understand the importance of FOXA1 in regard to ARID1A-dependent genes, we identified the ER-bound cis-regulatory elements close to ARID1A-repressed genes (those upregulated in ARID1A knockout cells), which we had previously shown to correlate with clinical outcome (Fig. 2e). We observed a modest change in ARID1A and BRG1 recruitment at these sites with FOXA1 loss (Fig. 4h). These findings show that the key ARID1A binding events are mediated by FOXA1 rather than ER.

We sought to identify the molecular mechanism that dictates decreased drug responsiveness when SWI/SNF components are deleted (Fig. 1f and Extended Data Fig. 1). We performed an assay for transposase-accessible chromatin sequencing (ATAC-seq) on MCF-7 ARID1A knockout or WT control cells, to assess whether ARID1A is required for maintenance of chromatin accessibility. Four independent cell culture samplings were performed. We observed 233,862 accessible regions in the genome, of which 83% (n = 194,341) were not altered in ARID1A knockout cells. Only 0.7% of sites showed a gain in accessibility in ARID1A knockout cells, and 16.3% of sites (n = 38,002) had decreased accessibility in ARID1A knockout cells (Extended Data Fig. 7). Integrative analyses of the chromatin accessibility and gene-expression datasets showed that genes that are upregulated in ARID1A knockout cells are associated more with ATAC-seq gained sites, implicating ARID1A in basal repression of these targets via inhibition of chromatin accessibility (Extended Data Fig. 7). However, there was no significant difference in accessibility at the regions co-bound by ARID1A and ER (data not shown), suggesting that loss of ARID1A does not alter chromatin accessibility at the regulatory regions bound by these protein complexes.
Because previous work showed that inhibition of SWI/SNF subunit BRD9 results in a switch to P-BAF activity\textsuperscript{18}, we hypothesized that loss of ARID1A and BAF activity might result in a switch to a P-BAF-driven pathway. We therefore conducted ChIP-seq on ARID2 (a P-BAF-specific complex component) and BRG1 in WT or ARID1A knockout clonal cell lines, and showed that ARID2 binding was not appreciably changed by tamoxifen treatment and that there was substantially less ARID2 binding in both ARID1A knockout clones, regardless of hormonal treatment conditions (Extended Data Fig. 8). This is a possible consequence of the decreased overall BRG1 binding in ARID1A-deleted cells (Fig. 5a and Extended Data Fig. 9). As such, loss of...
Fig. 3 | The SWI/SNF complex interacts with ER and is recruited to chromatin following drug treatment. 

a. ER, ARID1A or BRG1 RIME was conducted with asynchronous MCF-7 cells. IgG was used as a negative control. ER, FOXA1 and HDAC1 were identified as interactors in the ARID1A and BRG1 pulldowns, and vice versa. Boxplots show the enrichment of selected known interactors in the pulldown samples compared to IgG controls. Pulldowns were performed in two biological cell culture samples, and label-free quantification was performed using the Minora algorithm. The log2 intensities are normalized so that the median of IgGs is zero. Center line shows the median. *n* = 2 independent biological cell culture samples.

b. Five ER+ progesterone receptor primary tumor samples were split for ER or IgG pulldowns and the enrichment of known co-factors in ER compared to IgGs is shown. Boxplots show the enrichment of selected known ER interactors in the ER RIME samples compared to IgG controls in human breast cancer tissues. The log2 values are normalized so that the median of IgGs is zero. Center line shows the median.

c. ER qPLEX-RIME was conducted with asynchronous MCF-7 cells treated with 100 nM 4-hydroxytamoxifen in a four-point time course (*n* = 6 independent biological samples per group). Specific BAF proteins are highlighted, and the enrichment of BAF components in the ER complex following 4-hydroxytamoxifen treatment is shown. Center line shows the median. 

d. ChIP-seq of ARID1A, BRG1 or SNF5 (SMARCB1/BAF47) in asynchronous MCF-7 cells treated with either vehicle (ethanol) or 100 nM 4-hydroxytamoxifen (*n* = 3 independent biological ChIP-seq samples). Heatmaps represent the 39,214 ARID1A binding events observed after 4-hydroxytamoxifen treatment. Also included are H3K27Ac, ER and FOXA1 binding signal intensities at these regions.
ARID1A does not result in recruitment of ARID2 and a switch to P-BAF dependency.

ARID1A contributes to HDAC1 recruitment and regulation of acetylation. To assess the mechanistic basis for the ARID1A-repressive function, we performed H3K27Ac ChIP-seq and found that it was not affected in ARID1A knockout versus WT cells (Extended Data Fig. 9). To identify other possibilities explaining sustained gene expression in the presence of ER-targeted drugs when ARID1A is suppressed, we explored our RIME data and found that the histone deacetylase protein HDAC1 was an ARID1A-interacting protein in untreated conditions (Fig. 3a).

Fig. 4 | FOXA1 promotes binding of ARID1A and BRG1 to a subset of potential enhancer elements. a.b. Hormone-deprived MCF-7 cells were transfected with control or FOXA1 siRNA and ChIP-seq was conducted for ARID1A (a) or BRG1 (b), n = 3 independent biological ChIP-seq samples. Plots are shown with the average intensity of binding (mean read counts per 20 x 10⁶ library depth) versus log2 fold change with FOXA1 siRNA compared to control siRNA. c.d. Heatmaps (c) and boxplots (d) shown on ARID1A-BRG1 constant (n = 65,563 sites) and ARID1A-BRG1 lost sites (n = 9,355 sites) defined by DiffBind following FOXA1 silencing in MCF-7 cells. ER and FOXA1 overlap is also shown in e. n = 3 independent biological cell culture samples. P-values (d) were calculated by Welch’s t-test, two-sided. For boxplots, center line shows median values with the box bounds corresponding to the first and third quartiles; the upper and lower whiskers extend to the highest and lowest values, respectively, no further than 1.5 x IQR. Further statistical details are given in Supplementary Table 5a. e. Scatterplot showing the association of decreased ARID1A and BRG1 binding following FOXA1 silencing. P-values were calculated by Pearson correlation coefficient (PCC), two-sided. f.g. Scatterplots showing the association of ARID1A (f) and BRG1 (g) binding following FOXA1 silencing at tamoxifen (tam)-induced ARID1A (f) and BRG1 (g) binding sites from Fig. 3d. P-values were calculated by PCC, two-sided. h. Boxplots illustrating the effect of siFOXA1 on ARID1A and BRG1 binding on ER binding sites (n = 2,746 sites) close to ARID1A-repressed genes under vehicle conditions. P-values were calculated by Welch’s t-test, two-sided. Window of 400 bp around center of the factor binding. Center line shows the median values, with box bounds corresponding to the first and third quartiles; the upper and lower whiskers extend to the highest and lowest values, respectively, no further than 1.5 x IQR. Further statistical details are given in Supplementary Table 5b.
Fig. 5 | Loss of ARID1A results in decreased BRG1 and HDAC1 recruitment and increased histone H4 acetylation. a–c. Quantitative signal from BRG1 (a), HDAC1 (b) and ER (c) ChIP-seq in ARID1A knockout cells (n = 3 independent biological cell culture samples per group). ChIP-seq was conducted in WT cells or the two ARID1A knockout clones, showing decreased binding of factors in the absence of ARID1A. Average plots shown on HDAC1 lost sites in ARID1A knockout cells. d,e. Scatterplots showing the association of decreased BRG1 and HDAC1 binding in ARID1A knockout clones 11 (d) and 14 (e) following treatment with 100 nM 4-hydroxytamoxifen. n = 3 independent biological cell culture samples. P values were calculated by PCC, two-sided. f. ER qPLEX-RIME was conducted in four ER+ PDX tumors, two of which had loss of ARID1A via mutation (MT1/2) and two were WT (WT1/2) for ARID1A. Heatmaps reveal decreased BAF and HDAC1 interactions with ER in ARID1A mutant tumors compared to WT tumors. g. We specifically identified ARID1A-repressed genes in proximity to ER-bound regulatory elements (n = 686 sites) that display, according to principal component analysis (PCA), >75% contribution to the variance in intensity of histone H4 acetylation. The data are shown as boxplots. ARID1A-dependent genes acquired H4 acetylation, especially H4K8Ac and H4K12Ac at adjacent enhancers, coincident with increased gene expression. P values were calculated by Welch's t-test, two-sided. Window of 2 kb around the center of the binding event. Further statistical details are provided in Supplementary Table 5c.
In addition, in our qPLEX-RIME data, HDAC1 recruitment to the ER complex was enriched following tamoxifen treatment during active gene repression. Furthermore, HDAC1 was one of the most statistically enriched interactors in ER+ primary tumor samples (Extended Data Fig. 3) compared to IgG controls. We therefore conducted HDAC1 ChIP-seq and found a substantial decrease...
in HDAC1 recruitment when ARID1A was specifically knocked out (Fig. 5b and Extended Data Fig. 9). Only modest changes in ER binding were observed on HDAC1 lost sites (Fig. 5c, Extended Data Fig. 9 and Supplementary Fig. 11). We also observed a modest decrease in global FOXA1 binding as determined by ChIP-seq (data not shown). However, this was explained by a parallel decrease in FOXA1 expression, suggesting that ARID1A does not directly modulate FOXA1 recruitment to cis-regulatory elements but moderately influences FOXA1 binding by affecting total levels of this pioneer factor. BRG1 and HDAC1 binding was decreased at the same genomic regions in both ARID1A knockout clones (Fig. 5d,e), suggesting that binding of both HDAC1 and BRG1 is dependent on ARID1A.

Additionally, we performed ER qPLEX-RIME on four ER+ patient-derived xenograft (PDX) tumors, including two that had ARID1A loss via mutation and two ARID1A WT control models (Extended Data Fig. 9 and Supplementary Fig. 12). We found a decrease in interactions between ER and HDAC1, BAFl170 and BAFl55 in the ARID1A mutant PDX models (Fig. 5f). As such, BRG1-associated SWI/SNF complex physically associates with HDAC1 in an ARID1A-dependent manner, and the transcriptional repression elicited by HDAC1 requires functional ARID1A.

Given that HDAC proteins can actively remove the acetylation marks that are read by BET proteins, we speculated that changes in HDAC activity might explain the increased sensitivity to BET inhibition in the absence of a functional SWI/SNF complex. The histone acetylation marks that are read by BET proteins involve histone-4 lysine residues, including H4K5Ac, H4K8Ac and H4K12Ac. We assessed for increases in these histone marks in our ARID1A knockout cells as a potential consequence of decreased HDAC1 recruitment. A distinct subset of histone H4 acetylated sites were increased under both untreated and tamoxifen-treated conditions in ARID1A knockout cells, with the most prominent change observed in H4K8Ac (Supplementary Fig. 11). To understand the function of H4Ac following ARID1A-dependent genes, we examined the adjacent ER-bound cis-regulatory elements on ARID1A target genes in ARID1A WT versus knockout cells. ARID1A was recruited to these enhancers in WT cells, and these sites showed substantial upregulation of histone H4 acetylation, particularly H4K8/12Ac, in both clones (Fig. 5g and Extended Data Fig. 10). Given the decreased HDAC1 recruitment, the increase in H4K8/12Ac in ARID1A-depleted cells and the increased responsiveness to BETi in ARID1A-deleted contexts (Fig. 1g), we hypothesized that depletion of ARID1A would result in enhanced BRD4 binding and activity. BRD4 ChIP-seq in WT and ARID1A knockout cells revealed a gain of 6,197 BRD4 binding sites in ARID1A-depleted cells, confirming a significant increase in BRD4 chromatin binding. Analyses of ER binding sites close to ARID1A target genes showed increased BRD4 binding under ARID1A loss under both treatment conditions (Fig. 6a,c). The same regions showed a gain of histone H4 acetylation and BRD4 and decreased HDAC1 binding in ARID1A-deleted cells (Fig. 6b and Supplementary Figs. 13 and 14). We integrated the gained BRD4 binding that was observed only in ARID1A-null cells with the fulvestrant/tamoxifen-repressed genes, and found a significant enrichment of BRD4 recruitment to those genes typically repressed by both ER antagonists (Supplementary Fig. 13). Mechanistically, our findings show that depletion of ARID1A results in decreased HDAC1 binding, a gain in histone 4 acetylation and coincident BRD4 recruitment to regulatory elements adjacent to genes normally repressed by ER-targeted drugs in WT contexts (Supplementary Fig. 14). This culminates in increased basal proliferation that occurs in a BET-dependent manner. In support of the intrinsic regulation of proliferation by ARID1A, we assessed breast cancer patients with ARID1A mutations compared to patients with WT ARID1A, revealing a poorer clinical outcome in women with ARID1A mutant tumors (Fig. 6d and Supplementary Fig. 14, with details of statistical tests). To explore the link between BET-driven growth in ARID1A-null contexts and to assess other treatment options for women with ARID1A mutations, we established a tumor explant from an ARID1A mutant PDX tumor that has a frameshift mutation leading to ARID1A loss (Supplementary Fig. 14). Tumor tissue was cultured ex vivo and treated with either vehicle or two different BETi for 48 h, and we observed significant antiproliferative effects by assessment of Ki67 expression, a surrogate marker for proliferation, following treatment (Fig. 6e,f), confirming the dependence on BET proteins in ARID1A mutant/deleted contexts similar to WT contexts.

Our study shows that the SWI/SNF complex is recruited to ER cis-regulatory elements before active ER binding, via the pioneer factor FOXA1. ARID1A exhibits transcriptional repression by recruiting HDAC1 and, when ARID1A is functionally inactivated, HDAC1 binding is diminished, resulting in a gain in enhancer-specific acetylation that is subsequently ‘read’ by the BET protein BRD4 (Fig. 6g and Supplementary Fig. 15).

Discussion

Our unbiased genetic screening approach has revealed a critical role for the SWI/SNF complex in estrogen receptor-targeted treatment efficacy. Loss of ARID1A had profound effects on the gene-expression program and growth phenotype, by affecting the chromatin environment. Tumor growth and clinical outcome were influenced by ARID1A status, independent of estrogen-bound ER activity, in support of previous work showing that BAF57 could be recruited to the ER-target gene promoter pS2 (TFF1) in an estrogen-independent manner. In contrast, glucocorticoid receptor was shown to recruit the BAF complex to the MMTV chromatin template, implying that the mode of BAF-chromatin occupancy is nuclear receptor-specific. Our findings suggest that, while ARID1A and SWI/SNF components can be recruited to ER cis-regulatory elements by ER antagonistic ligands, in particular tamoxifen, this complex can associate with these enhancer elements independently of nuclear-receptor activation. In this study, we identified that the pioneer factor FOXA1, which demarcates ER regulatory elements and binds chromatin independently of hormonal status, is responsible for recruiting the BAF complex to chromatin. FOXA1 can directly recruit the histone-modifying methyltransferase that deposits the histone modification that is the hallmark of enhancer elements, and previous work has shown that FOXA1 can open a compacted chromatin template, independently of other proteins, placing it upstream of all factors subsequently recruited to these enhancer elements.

Mutation of ARID1A occurs in ~5% of primary breast cancer but frequency increases to ~12% when the metastatic context is assessed, implying a selection for tumor cells possessing loss-of-function ARID1A mutations. Our findings suggest that loss of ARID1A causes a shift in H4 acetylation status, a result of decreased HDAC1 binding, which consequently results in BRD4 recruitment and BET-dependent growth (Fig. 6g). Since ARID1A (and other components of the BAF complex) is commonly mutated in many cancer types, a role for this complex in regulating general proliferation status may involve co-opting the key cell type-specific cis-regulatory elements. Recent studies highlighted the possibility of exploiting a synthetic lethality-based treatment strategy in ARID1A mutant ovarian cancers, using inhibitors of BET proteins. BET inhibitors are proven to be effective in ER-dependent breast cancer cells, and our current work implies an increased dependency on epigenetic readers that drive cell division when the activity of the BAF complex is compromised. Given the frequency of BAF mutations in breast cancer, particularly in drug-resistant contexts, our findings would suggest exploration of the potential of epigenetic inhibitors that target the BET proteins.
Online content
Any methods, additional references, Nature Research reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at https://doi.org/10.1038/s41588-019-0541-5.

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Methods
Preparation of Cas9-expressing clones. MCF-7 cells were transduced with Cas9 lentiviral vector pKL2-EF1aCas9T2A-Bsd-W with 8 µg/ml Polybrene in 2% serum-containing medium without antibiotics. Medium was replaced after 24 h with 20% serum, grown for two further days and selected with 300 µM blasticidin for 4 days. These cells were single-cell sorted using BD FACSAria II in one 96-well plate, seeded with very high suspension and diluted into two 15-cm dishes and grown in the presence of blasticidin. After 10 days of growth, single-cell clones were hand-picked and seeded and grown in two 96-well plates. After the clones were well established, 48 were selected and assessed for Cas9 cutting efficiency by reporter assay in a six-well plate. Cas9 clones were transduced separately with pKL2-U6-gRNA5(FPFG)-PGKBPF-PGGFP-W, which has the gRNA for GFP. Highly efficient clones were selected, with ~95% of GFP+ cells in the infected population sorted using the BD Influx Cell Sorter (Supplementary Fig. 1). Average efficiency was calculated from four independent experiments. Cas9 cut efficiency is calculated as follows: Cas9 efficiency (%) = 100 – (%) GFP+ cells/total (% uncut cells) ± (% transduced cells). The clone 1C3 was selected from a FACS-sorted plate and showed 93.9% Cas9 cut efficiency; this was then used for the initial essentiality screen. The clone 3G1, sorted from a highly diluted plate, showed 94.62% Cas9 cut efficiency and was used for drug resistance screening. Both clones were compared 9 days after infection, with the gRNA library, for their reproducibility.

Genome-wide CRISPR screening. Highly efficient Cas9-expressing cells were infected with the human gRNA pooled library v.1 with the vector backbone pKL2-U6-gRNA5(lib)-PGKpuro-BFP-W+. Cells were seeded 2 days previously in a 15-cm dish, at ~30 million cells per replicate. Cells were infected with 30% transduction efficiency in Supplementary Fig. 11 so that only one Cas9 was integrated into the genome per cell. After 3 days (D3), 30% transduction efficiency was verified by FACS sorting in Influx for BFP+ cells. Sixty million cells were collected for next generation sequencing, and antibiotic selection was performed on the remaining cells with 10µg/ml puromycin for 4 days. GFP+ cells comprised at least 95% after 4 days of antibiotic selection, which was verified by FACS sorting in InFlux. Consequently, 100 million cells were collected on D7, 9, 12, 15 and 20. Genomic DNA was isolated from 20–50 million cells using QIAexpressionist, and antibiotic selection was performed using the gRNA library and peaks were called with MACs2 v2.0.10.20131216 (ref. 12) using sequences from MCF-7 chromatin extracts as background input control. Peaks yielded using MACs2 with q ≤ 1 × 10−6 were selected for downstream analysis. Genrich (https://github.com/mjsh/Genrich) was used to verify ATAC-seq peaks from MACs2. Motifs were identified among tag-enriched sequences by using Macs2 v4.9.1 (ref. 15).

Differential binding analysis (DifBind) was performed as described previously16. For visualization of tag density and signal distribution heatmap, the read coverage in a window of a region ±2.5 or 5 kb flanking the tag midpoint was generated using a bin size 1/100 of window length.

Gene signature analysis and Kaplan–Meier plots. A set of genes evaluated as differentially expressed in RNA-seq analysis, and located ±50kb from the differentially occupied sites evaluated in ChiP-seq analysis, were qualified as a potential gene signature.

METABRIC gene-expression data were accessed via the Application Programming Interface available at the Genomics Data Commons portal (https://gdc.cancer.gov/developers/gdc-application-programming-interface-ap) ported to MATLAB. Kaplan–Meier plots and log-rank tests were respectively used to display the survival probabilities per group as a function of time and to test whether the hazard functions of the groups of interest are different. Groups of clinical cases (n ≥ 20) of ER+ breast cancer cohorts were stratified by expression of group of genes established at a threshold corresponding to the most significant difference in survival.

RIME and qPLEX-RIME. RIME on cell lines. Cells were double-cross-linked with 2 mM dssuccinimidyl glutarate and 1% formaldehyde, as described in ChiP-seq and ChIP-seq sections. Cell lysates were prepared for the qPLEX-RIME experiment. With the following modification: beads were washed ten times with RIPA and twice with 100 mM ice-cold ammonium hydrogen carbonate. Antibodies used were ARID1A (no. HPA005456), BRG1 (no. ab215998), ER (no. ab3575 and Merck Millipore 06–935 antibody mix) and negative control IgG (no. ab171870).

qPLEX-RIME on patient-derived xenografts. Frozen clinical tissues were cryosectioned to a thickness of 30µm, and ~90 sections were double-cross-linked with 2 mM DSS for 25 min, and 1% formaldehyde in the same solution of DSS for 20 min. Cross-linking was quenched with 0.25 M glyc. Pulldown was performed with the ER antibody mix as described for ChiP-seq and qPLEX-RIME sections.

Proteomic sample preparation, liquid chromatography–mass spectrometry analysis and data processing. For sample preparation, trypsin at a final concentration of 15 ng/µl (‘Pierce’) was added to the beads followed by overnight incubation at 37°C. A second digestion step was performed the next day for 4h, and peptides were cleaned with the Ultra-Micro C18 Spin Column (Harvard Apparatus) according to the manufacturer’s instructions. The peptide fraction was followed by fractionation by fractionation using reversed-phase spin columns at high pH (Pierce, no. 84868). For qPLEX-RIME, peptide fractions were analyzed on a Dionex Ultima 3000 UHPLC system coupled with a nano-ESI Fusion Lumos (Thermo Scientific) and the peptide fraction was treated as a whole. Mass spectra were performed with Orbitrap in the range 380–1,500 m/z at 120 K resolution. Mass spectrum 2 (MS2) scans were performed in the ion trap with collision-induced dissociation (CID) energy 35%. Peptides were isolated in quadrupole with
an isolation window of 0.7 Tm. The top ten most intense fragments were selected for synchronous precursor selection HCD-MS3 analysis with a MS2 isolation window of 2.0 Tm. The higher-energy collisional dissociation (HCD) energy was set at 65%, and detection was performed at Orbitrap resolution of 50 K. For RIME experiments, peptides were detected on a Dionex Ultimate 3000 UHPLC system coupled with either the Q-Exactive HF (Thermo Fisher Scientific) or the Q-Exactive mass spectrometer; full mass spectrometry scans were acquired in Orbitrap within the range 400–1600 m/z at 60- or 70-K resolution, respectively. For MS2, the top ten most intense precursor ions were selected with a 2.0-Tm window followed by HCD fragmentation (collision energy 28%). The CID and HCD tandem mass spectra thus derived were processed with the SequestHT search engine in Proteome Discoverer 2.1 or Proteome Discoverer 2.2, respectively. SequestHT included the following parameters: precursor mass tolerance, 20 ppm; fragment mass tolerance, 0.5 Da for CID and 0.02 Da for HCD; dynamic modifications were oxidation of M (+15.995 Da), deamidation of N/Q (+0.984 Da) and static modifications were TMT-plex at any N terminus/ (z + 229.163 Da) for the qPLEX-RIME experiment only. The consensus workflow included calculation of TMT signal-to-noise, and the confidence level for peptide identifications was estimated with the Percolator node using a decoy database search. Peptide intensities for the qPLEX-RIME experiment were normalized and aggregated (by summing) to protein intensities. Differential protein expression was performed using limmaimplemented in the qPLEXAnalyzer tool (10.18129/B9.bioc.qPLEXAnalyzer). The Minora Feature Detector node implemented in Proteome Discoverer 2.2 was used for label-free quantification. Retention time differences of isotopologues were calculated as the ratio of max/min, with values > 0.2 min and minimum number of isotopes two peaks. The consensus workflow included Feature Mapper and Precursor Ions Quantifier for Precursor Abundance quantification based on intensity. The complete data are available in Supplementary Tables 3 and 4.

Sample size calculation for in vivo MCF-7 xenografts. The sample size of the study was defined so that, based on effect sizes derived from both previous data and nuisance parameters deduced from the data in ref. 44, a global power of 0.8 would be achieved when testing a chosen set of differences in means of tumor volumes at the global 5% level for different time points by means of Welch’s test.

In vivo xenografts. All mouse experiments were carried out under the Biological Resource Unit at Cancer Research United Kingdom (CRUK) Cambridge Institute. The experiments were performed in accordance with the UK Animals (Scientific Procedures) Act 1986, with approval from the CRUK Cambridge Institute Animal Ethical Review and Welfare Body. Age-matched (8 weeks) NOD/SCID/IL2rg−/− (NSG) female mice were purchased from Charles River. The animals were verified as pathogen free and in excellent health. Subcutaneous xenografts of MCF-7 cells/ ARID1A clones were derived by implanting 106 cells in 50% growth media and 50% matrigel (BD Biosciences) in the right flank of 8-week-old female NSG mice. The mice were also implanted subcutaneously with 90-d, slow-release 17β-estradiol pellets (Innovative Research of America) in the left flank. Mice were also implanted subcutaneously with 90-d, slow-release 17β-estradiol pellets (Innovative Research of America) in the right flank. After 4 weeks for the efficacy cohort, tumors were randomized and enrolled to the study when average tumor volume reached 100–150 mm3. Tamoxifen (8.8 mg/ml-1; Tocris Bioscience, no. 6342) was made in sterile filtered corn oil (Sigma, no. C8267). The mice were dosed intraperitoneally at 20 mg/kg−1, 6 d per week. Tumor sizes were measured twice per week with Vernier calipers. Because tumor volume tends to show linear growth on the cubic root scale, we used linear mixed models to compare the average tumor growth of different groups as a function of time from enrollment on that scale. Linear mixed models allow one to take into account both within-mouse and time dependence, by means of random effects and auto-regressive parameters, respectively. Here we considered a random intercept and-slope model with time since enrollment, groups and an interaction between time since enrollment and groups as fixed effects, and an autocorrelation structure of order 1 for the error term. Model checks suggested a good fit of the model to the data. Sensitivity analyses considering alternative modeling (such as an autoregressive order 2 model) did not lead to similar conclusions. We used program R (v.3.5.1) and the package lme4 (v.1.1–23) to fit the linear mixed models. Mean values decline for clones, especially at days 18 and 25, because tumor volume exceeded the 1,500-mm3 limit and these were thus removed from the mice.

Test statistics are shown in Supplementary Fig. 5. Supplementary Table 5c details on the boxplots on ChIP-seq data are given in Supplementary Table 5.

Assessment of off-target CRISPR effects. The top three predicted off-targets (ACGGCTCCTGTCGGCGCAG at chr1:205061276–205061299; AGAGGCCCCAGACCCCGGAC at chr7:1547999–1548017; CCGGCTCCCGGGGCAGCGG at chr1:10555551–105555574), defined by Desktop Genetics as having scores > 88 out of 100 (ref. 47), were verified based on absence of editing with Sanger sequencing by amplifying the regions with primers (Additional files). The AmpliconSeq analysis pipeline was used for data processing and variant calling. Briefly, reads were aligned against the reference genome (GRCh38) using BWA-MEM46 and variants were called using two methods (VarDict57 and GATK HaploTypeCaller (https://doi.org/10.1101/201178)). Consensus variants and their effects on CRISPR clones were then calculated. All clones used in this paper were short tandem repeat-genotyped and confirmed as free from mycoplasma.

Assessment of off-target CRISPR effects. The top three predicted off-targets (ACGGCTCCTGTCGGCGCAG at chr1:205061276–205061299; AGAGGCCCCAGACCCCGGAC at chr7:1547999–1548017; CCGGCTCCCGGGGCAGCGG at chr1:10555551–105555574), defined by Desktop Genetics as having scores > 88 out of 100 (ref. 47), were verified based on absence of editing with Sanger sequencing by amplifying the regions with primers against three loci from the final knockout and empty vector control clones (11, 14, 216, 219, 221). Primers are given in Supplementary Table 6.

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability
All CRISPR, ChiP-seq and RNA-seq data have been deposited at Gene Expression Omnibus and can be accessed at GSE134286. ATAC-seq data can be accessed at GSE134270. All proteomic data have been deposited at PRIDE and can be accessed at PXD018180. Source Data for Figs. 1 and 2 are available online.

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Author contributions

S.N. contributed to conceptualization, methodology, experimental work, formal analysis and figure assembly, writing of text, reviewing and advising on the manuscript. S.V.R. contributed to experimental work, analysis and figure assembly of mouse experiments and advising on the manuscript. J.S. contributed to experimental work, analysis and figure assembly of mouse experiments and advising on the manuscript. D.C. and E.A.G. contributed to experimental work and advising on the manuscript. D.-L.C. contributed to statistical analyses, figure assembly, reviewing and advising on the manuscript. K.K. and C.S.R.C. contributed to bioinformatic analyses, figure assembly and advising on the manuscript. C.B. contributed to histopathological analyses and advising on the manuscript. N.G. and R.N. performed ARID1A immunohistochemistry and advising on the manuscript. A.B. contributed to methodology regarding PDX material and advising on the manuscript. C.C. contributed to methodology regarding PDX material, funding acquisition and advising on the manuscript. R.S. contributed to methodology, supervision and advising on the manuscript. K.Y. contributed to methodology, funding acquisition and advising on the manuscript. I.C. contributed to methodology, bioinformatic analyses and figure assembly, writing of text and advising on the manuscript. J.-E.G.P. contributed to methodology regarding PDX material, funding acquisition, writing of text, reviewing and advising on the manuscript.

Competing interests

J.S.C. is the founder and chief scientific officer of Azeria Therapeutics.

Additional information

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Extended Data Fig. 1 | Enrichment of BAF and P-BAF components in the CRISPR screen. a. Scatterplot of CRISPR screening data, showing enrichment of BAF components following 26 days of different drug treatment, relative to DMSO treated control cells. \( n=3 \) independent viral infections. b. Log2 fold changes showing gRNA enrichment/depletion against all BAF, P-BAF and ncBAF components in the CRISPR screen. Treatment conditions are compared to DMSO control. More proliferative changes represent enriched gRNA after treatment, indicating genes that contribute to drug resistance. c, e. Validation of ARID1A perturbation effect on proliferation and drug response using ARID1A siRNA on MCF7 (c) and ZR-75-1 (e), representative experiments shown from 2 similar independent experiments each cell line. p-values calculated by One way ANOVA test. * denote \( p<0.05 \), *** denotes \( p<0.001 \). Sample size mentioned in S4. Measure of centre represents mean ± SEM (c) and mean ± SD (e). d. Western blot of ARID1A protein levels after siRNA transfection in MCF7 cells. A representative image is shown from 3 similar independent experiments. Unprocessed Western blot in Source Data Fig. 2.
Extended Data Fig. 2 | ARID1A co-binds ER and FOXA1-bound regulatory elements, but is depleted with estrogen treatment. a–c. Single gene profiles showing the binding of ER, FOXA1 and ARID1A on overlapping sites in MCF7 cells. ChIP-seq was performed using three independent biological cell cultures. d. Overlap of binding sites for ER, FOXA1 and ARID1A binding sites in ZR-75-1 cells. e. Boxplots showing the normalized ChIP-seq tag density around 400 bp window around the center of ARID1A binding on DiffBind-defined estrogen independent (constant) and dependent (reduced with estrogen) sites in MCF7. Both classes show reduced ARID1A binding upon estrogen. p-values were calculated by Welch’s t-test, two-sided. Centre line shows the median values with bounds of box corresponding to the first and third quartiles and the upper and lower whiskers extend to the largest or the smallest value no further than 1.5 x IQR (inter-quartile range). Statistical test details are mentioned in Supplementary Table 5e.
Extended Data Fig. 3 | Enrichment of SWI/SNF factors with ER and FOXA1 in RIME. a, ARID1A and BRG1 RIME were conducted on asynchronous MCF7 cells on two biological cell cultures. Label free quantification was performed to show the log 2 scaled normalized intensities of the BAF, P-BAF, ncBAF and common subunits of SWI/SNF complex. Rabbit polyclonal IgG is used as the negative control. b, ER qPLEX-RIME was performed on five primary tumours from ER+ breast cancer patients and the ER interactors are shown as enrichment over IgG vs -log10 p-value, corrected by Benjamini and Hochberg multiplicity correction, two-sided. c, d. Boxplots illustrating the more enrichment of HDAC1 (c) and less enrichment of random factors (d) in ERα RIME in five patients compared to IgG negative control in human breast tumours. The values are scaled to the median of IgG and log2 transformed. n = 5 independent biological cell cultures. e. Boxplots illustrating the enrichment of selected known ERα interactors from the RIME experiment in MCF7 cells at a representative timepoint (4-hydroxytamoxifen-24 hrs) comparing to IgG negative control. The values are scaled to the median of IgG and log2 transformed. n = 5 independent biological cell cultures. For all boxplots, Centre line shows the median values with bounds of box corresponding to the first and third quartiles and the upper and lower whiskers extend to the largest or the smallest value no further than 1.5×IQR (inter-quartile range).
Extended Data Fig. 4 | Enrichment of SWI/SNF factors during Tamoxifen and Fulvestrant in ChIP-seq experiments. a–d. Asynchronous MCF7 cells were treated with vehicle or Fulvestrant, an ER degrader and ChIP-seq was conducted for ARID1A (b), BRG1 (c) or SNF5 (d). Triplicate independent cell cultures were conducted. d. Single gene profile showing the induction of SWI/SNF complex binding during Fulvestrant treatment. e. Overlap of ARID1A lost sites during estrogen treatment with gained sites during Tamoxifen and Fulvestrant from three independent biological cell cultures. f. Overlap of ARID1A gained sites during Tamoxifen treatment with Fulvestrant and Tamoxifen downregulated genes.
Extended Data Fig. 5 | FOXA1 promotes the binding of ARID1A and BRG1. Hormone-deprived ZR-75-1 cells were transfected with control or FOXA1 siRNA and ChIP-seq was conducted for ARID1A (a) and BRG1 (b). n = 3 independent biological cell cultures. MA plots are shown with the average intensity of binding vs log2 fold change with FOXA1 siRNA relative to control siRNA. c. Scatterplot showing the association of the loss of ARID1A and BRG1 binding upon FOXA1 knockdown. PCC – Pearson Correlation coefficient, two-sided. d. Heatmaps shown on ARID1A and BRG1 FOXA1 independent (common) and dependent (lost sites with FOXA1 knockdown) sites in ZR-75-1 cells. e. Boxplots showing the normalized ChIP-seq tag density around 400 bp window of ARID1A and BRG1 on FOXA1 independent (constant, n = 70,429 sites) and dependent (lost sites with siFOXA1, n = 17,357 sites) sites in ZR-75-1. p-value calculated by Welch’s test, two-sided. n = 3 independent biological cell culture samples. Centre line shows the median values with bounds of box corresponding to the first and third quartiles and the upper and lower whiskers extend to the largest or the smallest value no further than 1.5 × IQR (interquartile range). Statistical test details are mentioned in Supplementary Table 5f.
Extended Data Fig. 6 | FOXA1 promotes the binding of ARID1A and BRG1. Hormone-deprived MCF7 and ZR-75-1 cells were transfected with control or FOXA1 siRNA and ChIP-seq was conducted for ARID1A and BRG1. n = 3 independent biological cell cultures. (a-b) Single gene profiles of CCND1 (a) and CDH1 (b) showing the effect on SWI/SNF complex binding with FOXA1 knockdown on MCF7 and ZR-75-1 cells. ER and FOXA1 binding overlap is shown. (c-d) ChIP-qPCR analyses on specific sites (CCND1 and CDH1 ER binding sites) showing ARID1A and BRG1 binding with FOXA1 knockdown in hormone-deprived MCF7 and ZR-75-1 cells (c) or ARID1A binding following Tamoxifen treatment in asynchronous MCF7 cells (d). n = 3 independent biological cell cultures. * denotes p ≤ 0.05, ** denotes p ≤ 0.01, *** denotes p ≤ 0.001. Precise p-values are mentioned in Supplementary Fig. 10. Mean is measured as centre shown with standard deviation. Details of the statistical tests are mentioned in Supplementary Fig. 10.
Extended Data Fig. 7 | ATAC-seq analyses shows a negligible regulation of ARID1A on transcription-associated chromatin opening. a. Heatmap showing ATAC-seq analysis in ARID1A KO clones 11 and 14 following Tamoxifen treatment. Common, gained and lost sites defined by DiffBind analysis. n = 4 independent biological cell cultures. FDR ≤ 0.05 corrected by Benjamini-Hochberg multiplicity correction, two-sided. b. Association of ARID1A KO upregulated and downregulated genes with ATAC-seq gained and lost sites.
Extended Data Fig. 8 | ARID1A perturbation regulates ARID2 binding. a. ARID2 ChIP-seq was conducted in wild type cells or the two ARID1A knock-out clonal cell lines and heatmaps are shown on ARID2 binding sites after Tamoxifen treatment. Also included was ARID1A ChIP-seq from wild type cells treated with vehicle or Tamoxifen. ARID2 binding overlapped with ARID1A binding and was dependent on ARID1A. n = 3 independent biological cell cultures. b. Signal intensity plot showing changes in ARID2 binding in wild type control cells or ARID1A knock-out cells at ARID2 binding sites. n = 3 independent biological cell cultures.
Extended Data Fig. 9 | ARID1A promotes BRG1 and HDAC1 binding without affecting ER and H3K27ac occupancy. a, b. BRG1, H3K27Ac, HDAC1 and ER (b) ChIP-seq were conducted in asynchronous wild type cells treated with vehicle or tamoxifen or in the two ARID1A knock-out clones (Clones 11 and 14) following tamoxifen treatment. The binding is shown on regions where HDAC1 is lost in ARID1A knockout cells relative to wild type cells. n = 3 independent biological cell cultures. c, d. Scatterplot showing the correlation of ER (c) or H3K27Ac (d) and HDAC1 binding in ARID1A knockout clone 11 versus wild type cells. n = 3 independent biological cell cultures. PCC – Pearson Correlation coefficient. p-values were calculated by Pearson correlation test, two-sided. e. Principal Component Analysis (PCA) of normalised peptide intensities of PDX tumours after ER qPLEX-RIME. n = 2 PDX each group. f. Details of ARID1A mutations observed within ER+ PDX tumours used in ER qPLEX-RIME.
Extended Data Fig. 10 | ARID1A regulates histone H4 acetylation. Upregulation of histone H4 acetylation in ARID1A knock-out clone 11 and 14 in Vehicle (a) or Tamoxifen (b) treated cells comparing to wild type cells. Heatmap representing the changes in histone H4Ac marks upon ARID1A knockout with Vehicle or Tamoxifen treatment on ER binding sites close to ARID1A repressed genes. n = 3. (c) Empirical cumulative probability distribution plots of H4K8Ac and H4K12Ac ChIP-seq signals showing upregulation in intensity (y-axis) with ARID1A knockouts clones 11 and 14. Plots were made on ER sites close to ARID1A repressed genes (n = 686 sites) with more than 75% contribution to the variance in intensity. Window – 2 kb around the center of binding.
Reporting Summary

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Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

- The exact sample size (n) for each experimental group/condition, given as a discrete number and unit of measurement
- A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
- The statistical test(s) used AND whether they are one- or two-sided
- Only common tests should be described solely by name; describe more complex techniques in the Methods section.
- A description of all covariates tested
- A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons
- A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
- For null hypothesis testing, the test statistic (e.g. $F$, $t$, $r$) with confidence intervals, effect sizes, degrees of freedom and $P$ value noted. Give $P$ values as exact values whenever suitable.
- For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
- For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
- Estimates of effect sizes (e.g. Cohen’s $d$, Pearson’s $r$), indicating how they were calculated

Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection

Softwares were not used for data collection.

Data analysis

MAGECK v 0.5.4, bowtie2 v 2.2.6, qPLEXanalyzer tool (10.18129/B9.bioc.qPLEXanalyzer), BLAT v 34, dtwclust v 5.5.2 (R v 3.5.3), DESeq2.1.22.2, nlme v 3.1-137 (R v 3.5.3), MATLAB R2015b, MACS2 v 2.0.10.20131216 and 2.1.1.2016, bedtools v 2.26.0-97, DiffBind v 2.2.12, STAR version 2.5.1a

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors/reviewers. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Research guidelines for submitting code & software for further information.

Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A list of figures that have associated raw data
- A description of any restrictions on data availability

All genomic data has been deposited at GEO and can be accessed at GSE123286. ATAC-seq data can be accessed at GSE134270. All proteomic data has been deposited at PRIDE and can be accessed at PXD011810.

To review GEO accession GSE123286: https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE123286
To review GEO accession GSE134270: https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE134270
To review accession PXD011810: https://www.ebi.ac.uk/pride/archive/projects/PXD011810
Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☒ Life sciences ☐ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size

The sample size of the study was defined so that, based on expected growth curves per group defined on prior data and on nuisance parameters deduced from prior data, a global power of 0.8 would be achieved when testing a chosen set of differences in means of tumor volumes at the global 5% level for different time points by means of Welch's tests. Sample size for ChIP-seq with 3 biological replicates and RNA-seq with 4 replicates was followed according to the revised ENCODE guidelines (https://www.encodeproject.org/documents/ceb172ef-7474-4cd6-bfd2-5e8e6e38592e/@download/attachment/ChIP-seq_ENCODE3_v3.0.pdf, https://www.encodeproject.org/documents/c6d0cbe-d324-4ce7-ace4-f0c3edd5972f/@download/attachment/ENC%3A%20Best%20Practices%20for%20RNA%20v2.pdf).

Data exclusions

To show the significant information during the treatment of Tamoxifen in in vivo mice xenograft experiment, the data was excluded during the start of the experiment. One replicate of BRG1 Tamoxifen treated sample in wild type MCF7 cells (jc5489_mcf7_brg1_4_oht_ecacc_parental_wt) is omitted from analysis due to lack of ChIP-seq coverage. From the Proteomics experiment, one of the PDX AB630 was ignored as the tissue was verified to be necrotic. One of the patient sample from the published data (Papachristou et al, 2018, Nat. Communications) with ER RIME was ignored for ARID1A as the peptide detection was not enough.

Replication

At least three independent replicates were performed in every experiment. Clustering analysis and PCA plots were performed to check their successful reproducibility. Data was replicated in all attempts except the samples provided above as data exclusions. Details are provided above.

Randomization

For both RNAseq and ChIPseq experiments, samples were block-randomised for 96 well plate. Mice were randomized before enrollment for drug treatments.

Blinding

The statistician was involved in a blind manner unaware of treatment groups for xenograft studies. Bioinformaticians were also blindly involved in the analyses of ChIP-seq and RIME data.

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

| Materials & experimental systems | Methods |
|---------------------------------|---------|
| n/a    | n/a     |
| ☑ Antibodies                     | ☑ ChIP-seq |
| ☑ Eukaryotic cell lines          | ☑ Flow cytometry |
| ☑ Palaeontology                  | ☑ MRI-based neuroimaging |
| ☑ Animals and other organisms    |         |
| ☑ Human research participants    |         |
| ☑ Clinical data                  |         |

Antibodies

For ChIP/RIME, following antibodies were used: ARID1A (Human protein atlas Sigma HPA005456, Lot 1114190), BRG1 (abcam ab215998 Lot EPNCIR111A/GR29666-1), ER-alpha antibody (mix of abcam ab3575 Lot GR3191181-1/GR3217431-5 and and Millipore 06-935 Lot 3008172/3045S93), negative control IgG (abcam ab171870, Lot GR31135-2), H3K27ac (Diagenode C15410196 Premium, Lot A1723-0041D), HDAC1 (Diagenode Premium C15410325, Lot A21-001P) and ARID2 (Bethyl A302-229A, lot 2), SNFS (Bethyl A301-087A, No lot number available), H4K5ac (ab51997, Lot GR3201311-4), H4K8ac (ab15823, Lot GR3209076-1), H4K12ac (Diagenode C15410331-50, lot A2439P), BRD4 antibody (considered as 1 mg/ml, No lot number available) provided by Prof. Cheng Ming Chiang. All ChIP antibodies were used as 2.5 ug per 15 cm dish of cells.

For Western blots, following antibodies were used: ARID1A (Human protein atlas Sigma HPA005456, Lot 1114190, 1:250), ARID1A/BAF250A (D2A8U) Rabbit mAb 12354 from cell signalling (1:1000, No lot number available), B-actin (Cell signalling 4970 Clone 13E5 and Abcam ab6276, No lot number available, 1:5000), ER-α NCL-L-ER-6F11 (Leica Biosystems Novocastra, Lot
Validation

Antibodies were validated with knockouts/knockdown experiments, Mass spectrometry-based approaches (RIME). Source data is provided in Data 1 and 2 and all RIME experiments coverage plot had been provided as Computational Data 3.

Eukaryotic cell lines

Policy information about cell lines

Cell line source(s) MCF7 cells were obtained from ECACC and ZR-75-1 cells were obtained from ATCC.

Authentication Genotyping was performed using Short Tandem repeats (STR) genotyping and Para DNA profiling. For the STR profiling, a commercially available 16 markers profile was utilised and analysed with Genemapper 5. Para DNA profile was obtained from LGC and the data were analysed with Para DNA analyser.

Mycoplasma contamination All cell lines were confirmed for Mycoplasma negativity using RNA capture ELISA method (Mycoprobe™ from R&D systems).

Commonly misidentified lines Misidentified lines were not used.

Animals and other organisms

Policy information about studies involving animals; ARRIVE guidelines recommended for reporting animal research

Laboratory animals Age matched (8 weeks) NOD/SCID/IL2Rg−/− (NSG) female mice were purchased from Charles River. The animals were verified to be pathogen free and in excellent health.

Wild animals Study did not involve wild animals.

Field-collected samples Study did not involve field-collected samples.

Ethics oversight The experiments were in accordance with the UK Animals (Scientific Procedures) Act 1986, UK Home office project license: 70/7679, with approval from the CRUK Cambridge Institute Animal Ethical Review and Welfare Body.

Human research participants

Policy information about studies involving human research participants

Population characteristics This study using human patient samples for RIME was already published in Papachristou et al 2018, Nat Comm. Gender - ER+ Female breast cancer patients.

Recruitment Patients were recruited according to the presence of ER by IHC.

Ethics oversight Patient samples were collected under protocol X13-0133, HREC/13/RPAH/187. HREC approval was obtained through the SLHD (Sydney Local Health District) Ethics Committee ([Royal Prince Alfred Hospital] zone), and site-specific approvals were obtained for all additional sites. Written consent was obtained from all patients prior to collection of tissue and clinical data stored in a de-identified manner, following pre-approved protocols.

Clinical data

Policy information about clinical studies

All manuscripts should comply with the ICMJE guidelines for publication of clinical research and a completed CONSORT checklist must be included with all submissions.

Clinical trial registration Clinical data were obtained from METABRIC which was published before (Curtis et al, Nature 2012, Pereira et al, Nat Comm, 2016)

Study protocol Study protocol can be obtained from METABRIC which was published before (Curtis et al, Nature 2012, Pereira et al, Nat Comm, 2016)

Data collection Details can be obtained from METABRIC which was published before (Curtis et al, Nature 2012, Pereira et al, Nat Comm, 2016). For METABRIC: "We assembled a collection of over 2,000 clinically annotated primary fresh frozen breast cancer specimens and a subset of normals that passed initial selection criteria from tumour banks in the UK and Canada (for which a subset of 2,136 expression arrays and 2,477 Affymetrix SNP 6.0 arrays are reported on here). Both primary breast tumours, with linked pseudo-anonymised clinical data, and normal breast tissue were obtained with appropriate ethical approval from the relevant institutional review board. The METABRIC study protocol, detailing the molecular profiling methodology, was also approved by the ethics committees in Cambridge and Vancouver, the two sites responsible for the molecular analysis of the samples. As described below, tumours were primary invasive breast carcinomas for which clinical information could be categorically linked to
DNA and RNA specimens. Initial quality control involved assessment of array quality and the flagging of mismatches between DNA and RNA using a novel eQTL-based approach (outlined below). Following the exclusion of cases on histopathological grounds (benign cases, those with ductal or lobular carcinoma in situ (DCIS, LCIS), or low tumour cellularity), incomplete clinical/pathological data (absence of ER status, grade, or tumour size), or apparently related individuals (based on genotype calls), paired DNA and RNA profiles were available from tumours derived from 997 female patients. DNA from adjacent normal breast tissue (or peripheral blood for some cases) was available from 485 samples, a subset of which match tumours in the discovery set. High quality RNA derived from adjacent normal tissue was available from 144 samples. A second cohort of 995 cases was later assembled for which matched DNA or RNA profiles or clinical information was not available at the time of the initial analyses, and these included low cellularity tumours, DCIS, and three benign cases. This cohort represents a validation set, which was employed for the purposes of testing the reproducibility of the integrative clusters and clinical outcome associations. Genotype analysis for the full set of cases subsequently revealed that eight individuals were represented both in the discovery and validation set (MB: 0667/0025, 0546/0326, 0327/0547, 0549/0329, 0559/0355, 0573/0355, 0408/0407, 0432/0433), and four were represented twice in the validation set (MB: 0110/0196, 0552/0330, 6213/6206, 2820/2720), but were supplied as unique accessions by the tumour bank. These sample pairs represent multiple primary tumours from the same individual and different sections of the same tumour. These comprise only 1.2% of samples in the validation set and do not alter the conclusions from these analyses.”

Outcomes
Outcome information can be obtained from METABRIC which was published before (Curtis et al, Nature 2012, Pereira et al, Nat Comm, 2016)

ChIP-seq

Data deposition

- Confirm that both raw and final processed data have been deposited in a public database such as GEO.
- Confirm that you have deposited or provided access to graph files (e.g. BED files) for the called peaks.

Data access links
GEO accession GSE123286: https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE123286
To review accession PXD011810: https://www.ebi.ac.uk/pride/archive/projects/PXD011810

Files in database submission
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jc4346_MCF7_Cas9_1C3_Day_9_Day_9_CRI07.fq.gz
jc4346_MCF7_Cas9_1C3_Day_9_Day_9_CRI11.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI01.fq.gz
| Sample Name                                                                 |
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| jc5792_MCF7_ARID2_Veh_ARID1A_knockout_clone_14_CRI04.fq.gz              |
| jc5792_MCF7_ARID2_Veh_ARID1A_knockout_clone_14_CRI05.fq.gz              |
| jc5792_MCF7_ARID2_Veh_ARID1A_knockout_clone_14_CRI06.fq.gz              |
| jc5793_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI04.fq.gz            |
| jc5793_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI04.fq.gz            |
| jc5794_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI04.fq.gz            |
| jc5794_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI04.fq.gz            |
| jc5795_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI04.fq.gz            |
| jc5795_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI05.fq.gz            |
| jc5795_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI05.fq.gz            |
| jc5795_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI06.fq.gz            |
| jc5795_MCF7_ARID2_4_OHT_ARID1A_knockout_clone_14_CRI06.fq.gz            |
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| jc5809_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI04.fq.gz               |
| jc5809_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI05.fq.gz               |
| jc5810_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI03.fq.gz               |
| jc5810_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI04.fq.gz               |
| jc5810_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI05.fq.gz               |
| jc5811_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI03.fq.gz               |
| jc5811_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI04.fq.gz               |
| jc5811_MCF7_BRD4_Veh_Parental_Wild_type_ECACC_CRI05.fq.gz               |
| jc5812_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI03.fq.gz             |
| jc5812_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI04.fq.gz             |
| jc5812_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI05.fq.gz             |
| jc5813_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI03.fq.gz             |
| jc5813_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI04.fq.gz             |
| jc5813_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI05.fq.gz             |
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| jc5814_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI04.fq.gz             |
| jc5814_MCF7_BRD4_4_OHT_Parental_Wild_type_ECACC_CRI05.fq.gz             |
| jc5815_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI03.fq.gz               |
| jc5815_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI04.fq.gz               |
| jc5815_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI05.fq.gz               |
| jc5816_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI03.fq.gz               |
| jc5816_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI04.fq.gz               |
| jc5816_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI05.fq.gz               |
| jc5817_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI03.fq.gz               |
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| jc5817_MCF7_BRD4_Veh_ARID1A_knockout_clone_11_CRI05.fq.gz               |
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| jc5818_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI05.fq.gz             |
| jc5819_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI03.fq.gz             |
| jc5819_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI04.fq.gz             |
| jc5819_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI05.fq.gz             |
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| jc5820_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI04.fq.gz             |
| jc5820_MCF7_BRD4_4_OHT_ARID1A_knockout_clone_11_CRI05.fq.gz             |
| mcf7_arid1a_4_oht_ma.bed.gz                                            |
| mcf7_arid1a_dcdt_ma.bed.gz                                             |
| mcf7_arid1a_e2_ma.bed.gz                                               |
| mcf7_arid1a_ici_ma.bed.gz                                              |
| mcf7_arid1a_jq1_ma.bed.gz                                              |
| mcf7_arid1a_veh_ma.bed.gz                                              |
| mcf7_arid2_4_oht_arid1a_knockout_clone_11.bed.gz                      |
| mcf7_arid2_4_oht_arid1a_knockout_clone_11.bed.gz                      |
| mcf7_arid2_4_oht_arid1a_knockout_clone_14.bed.gz                      |
| mcf7_arid2_4_oht_parental_wild_type_ecacc.bed.gz                      |
| mcf7_arid2_veh_arid1a_knockout_clone_11.bed.gz                        |
| mcf7_arid2_veh_arid1a_knockout_clone_14.bed.gz                        |
| mcf7_arid2_veh_arid1a_knockout_clone_11.bed.gz                        |
| mcf7_brdrd_4_oht_arid1a_knockout_clone_11.bed.gz                      |
| mcf7_brdrd_4_oht_parental_wild_type_ecacc.bed.gz                      |
| mcf7_brdrd_veh_arid1a_knockout_clone_11.bed.gz                        |
| mcf7_brg1_4_oht_arid1a_ko_clone_11.bed.gz                             |
| mcf7_brg1_4_oht_arid1a_ko_clone_11.bed.gz                             |
| mcf7_brg1_4_oht_ecacc_parental_wt.bed.gz                              |
| mcf7_brg1_4_oht_ma.bed.gz                                             |
| mcf7_brg1_dcdt_ma.bed.gz                                              |
| mcf7_brg1_e2_ma.bed.gz                                               |
| mcf7_brg1_ici_ma.bed.gz                                              |
| mcf7_brg1_jq1_ma.bed.gz                                              |
| mcf7_brg1_veh_arid1a_ko_clone_11.bed.gz                               |
| mcf7_brg1_veh_arid1a_ko_clone_11.bed.gz                               |
| mcf7_brg1_veh_arid1a_ko_clone_14.bed.gz                               |
| Sample Name                                           |
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| jc4874_MCF7_JQ1_Wild_type_CRI05.fq.gz                |
| jc4874_MCF7_JQ1_Wild_type_CRI06.fq.gz                |
| jc4874_MCF7_JQ1_Wild_type_CRI07.fq.gz                |
| jc4874_MCF7_JQ1_Wild_type_CRI08.fq.gz                |
| jc4874_MCF7_JQ1_Wild_type_CRI18.fq.gz                |
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI03.fq.gz|
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI04.fq.gz|
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI05.fq.gz|
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI06.fq.gz|
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI07.fq.gz|
| jc4875_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI08.fq.gz|
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| jc4876_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI04.fq.gz|
| jc4876_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI05.fq.gz|
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| jc4876_MCF7_4_hydroxy_Tamoxifen_Wild_type_CRI08.fq.gz|
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| jc4879_MCF7_Fulvestrant_Wild_type_CRI08.fq.gz        |
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| jc4880_MCF7_Fulvestrant_Wild_type_CRI18.fq.gz        |
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| jc4882_MCF7_Fulvestrant_Wild_type_CRI18.fq.gz        |
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| jc4883_MCF7_Vehicle_Wild_type_CRI04.fq.gz            |
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| jc4939_MCF7_4_hydroxy_Tamoxifen_ARID1A_CRI07.fq.gz           |
| jc4939_MCF7_4_hydroxy_Tamoxifen_ARID1A_CRI08.fq.gz           |
| jc4939_MCF7_4_hydroxy_Tamoxifen_ARID1A_CRI18.fq.gz           |
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jc4346_MCF7_Cas9_1C3_Day_9_Day_9_CRI03.fq.gz
jc4346_MCF7_Cas9_1C3_Day_9_Day_9_CRI04.fq.gz
jc4346_MCF7_Cas9_1C3_Day_9_Day_9_CRI07.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI01.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI02.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI03.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI04.fq.gz
jc4347_MCF7_Cas9_1C3_Day_12_Day_12_CRI07.fq.gz
jc4348_MCF7_Cas9_1C3_Day_12_Day_12_CRI01.fq.gz
jc4348_MCF7_Cas9_1C3_Day_12_Day_12_CRI02.fq.gz
jc4348_MCF7_Cas9_1C3_Day_12_Day_12_CRI03.fq.gz
jc4348_MCF7_Cas9_1C3_Day_12_Day_12_CRI04.fq.gz
jc4348_MCF7_Cas9_1C3_Day_12_Day_12_CRI07.fq.gz
jc4349_MCF7_Cas9_1C3_Day_15_Day_15_CRI01.fq.gz
jc4349_MCF7_Cas9_1C3_Day_15_Day_15_CRI02.fq.gz
jc4349_MCF7_Cas9_1C3_Day_15_Day_15_CRI03.fq.gz
jc4350_MCF7_Cas9_1C3_Day_15_Day_15_CRI04.fq.gz
Genome browser session
(e.g. UCSC)
http://genome-euro.ucsc.edu/cgi-bin/hgTracks?db=hg38&hubUrl=http://jclab.cruk.cam.ac.uk/hubs/ARID1A_BAF_chipSEQ_Nagarajan/hub.txt

Methodology

Replicates
Three biological replicates were performed.

Sequencing depth

sample id cell line treatment genome # reads # aligned reads # uniquely aligned reads
jc354_MCF7_Cas9_1C3_Day_15_Day_15_CRI01.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI02.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI03.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI04.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI07.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI11.fq.gz
cj354_MCF7_Cas9_1C3_Day_15_Day_15_CRI12.fq.gz
jc355_MCF7_Cas9_1C3_Day_15_Day_15_CRI01.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI02.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI03.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI04.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI07.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI11.fq.gz
cj355_MCF7_Cas9_1C3_Day_15_Day_15_CRI12.fq.gz
mcf7_cas9_counts1.tsv
mcf7_cas9_counts2.tsv
| JC5779 MCF7 ARID2 Veh hg38 37343205 31821758 27901519 |
| JC5780 MCF7 ARID2 Veh hg38 35429104 30329062 26474392 |
| JC5781 MCF7 ARID2 4-OHT hg38 34336467 28791344 24948119 |
| JC5782 MCF7 ARID2 4-OHT hg38 39789374 3487510 30102494 |
| JC5783 MCF7 ARID2 4-OHT hg38 34985621 30468800 26541118 |
| JC5784 MCF7 ARID2 Veh hg38 22491364 19180092 16596555 |
| JC5785 MCF7 ARID2 Veh hg38 37191576 31747496 27543277 |
| JC5786 MCF7 ARID2 Veh hg38 30068026 25997651 22145167 |
| JC5787 MCF7 ARID2 4-OHT hg38 40038367 34352429 30656505 |
| JC5788 MCF7 ARID2 4-OHT hg38 39987654 39126893 35531086 |
| JC5789 MCF7 ARID2 4-OHT hg38 30139518 25638951 22259277 |
| JC5790 MCF7 ARID2 Veh hg38 35577601 30438213 26530672 |
| JC5791 MCF7 ARID2 Veh hg38 40648648 34535847 30454613 |
| JC5792 MCF7 ARID2 4-OHT hg38 38410534 32974642 28592829 |
| JC5793 MCF7 ARID2 4-OHT hg38 27253414 23097822 20367200 |
| JC5794 MCF7 ARID2 4-OHT hg38 23184518 20120862 17895534 |
| JC5795 MCF7 Input Veh hg38 33862278 29629128 26418586 |
| JC5796 MCF7 Input Veh hg38 39107583 33410863 29921568 |
| JC5797 MCF7 Input Veh hg38 32350452 28158434 24928476 |
| JC5798 MCF7 Input Veh hg38 38012351 32737332 29301776 |
| JC5799 MCF7 Input Veh hg38 36747095 3169804 28151352 |
| JC5800 MCF7 Input Veh hg38 33956283 29190020 23606061 |
| JC5801 MCF7 Input Veh hg38 38473447 34025538 27013892 |
| JC5802 MCF7 Input Veh hg38 34492823 29954950 24239038 |
| JC5803 MCF7 Input Veh hg38 40420792 35237185 30632463 |
| JC5804 MCF7 Input Veh hg38 36750959 32460029 25763468 |
| JC5805 MCF7 Input Veh hg38 36907948 32157499 25949554 |
| JC5806 MCF7 Input Veh hg38 34810038 30084653 24560382 |
| JC5807 MCF7 Input Veh hg38 39092384 34897490 27072552 |
| JC5808 MCF7 Input Veh hg38 2607316 22484889 18255525 |
| JC5809 MCF7 Input Veh hg38 29946306 25998288 20800360 |
| JC5810 MCF7 Input Veh hg38 25672726 22351884 18106577 |
| JC5811 MCF7 Input Veh hg38 31007120 26923196 21663632 |
| JC5812 MCF7 Input Veh hg38 30420808 26072967 21083662 |
| JC5813 MCF7 Input Veh hg38 3080595 26759669 21470867 |
| JC5814 MCF7 Input Veh hg38 27303297 23832675 19908110 |
| JC5815 MCF7 Input Veh hg38 29653350 25734752 20616940 |
| JC5816 MCF7 Input Veh hg38 30800673 27148868 21707143 |
| JC5817 MCF7 Input Veh hg38 31081393 27305486 21772305 |
| JC5818 MCF7 Input Veh hg38 61312390 52381230 45531409 |
| JC5819 MCF7 Input Veh hg38 68998878 58999903 50676481 |
| JC5820 MCF7 Input Veh hg38 62404904 53864141 45987112 |
| JC5821 MCF7 Input Veh hg38 65131115 56046479 48059566 |
| JC5822 MCF7 Input Veh hg38 54483589 46292282 40416666 |
| JC5823 MCF7 Input Veh hg38 25763259 24131337 16005113 |
| JC5824 MCF7 Input Veh hg38 25524714 20679616 14782821 |
| JC5825 MCF7 Input Veh hg38 32350452 28158434 24928476 |
| JC5826 MCF7 Input Veh hg38 38012351 32737332 29301776 |
| JC5827 MCF7 Input Veh hg38 2607316 22484889 18255525 |
| JC5828 MCF7 Input Veh hg38 29946306 25998288 20800360 |
| JC5829 MCF7 Input Veh hg38 25672726 22351884 18106577 |
| JC5830 MCF7 Input Veh hg38 31007120 26923196 21663632 |
| JC5831 MCF7 Input Veh hg38 30420808 26072967 21083662 |
| JC5832 MCF7 Input Veh hg38 3080595 26759669 21470867 |
| JC5833 MCF7 Input Veh hg38 27303297 23832675 19908110 |
| JC5834 MCF7 Input Veh hg38 29653350 25734752 20616940 |
| JC5835 MCF7 Input Veh hg38 30800673 27148868 21707143 |
| JC5836 MCF7 Input Veh hg38 31081393 27305486 21772305 |
| JC5837 MCF7 Input Veh hg38 61312390 52381230 45531409 |
| JC5838 MCF7 Input Veh hg38 68998878 58999903 50676481 |
| JC5839 MCF7 Input Veh hg38 62404904 53864141 45987112 |
| JC5840 MCF7 Input Veh hg38 65131115 56046479 48059566 |
| JC5841 MCF7 Input Veh hg38 54483589 46292282 40416666 |
| JC5842 MCF7 Input Veh hg38 25763259 24131337 16005113 |
| JC5843 MCF7 Input Veh hg38 25524714 20679616 14782821 |
| JC5844 MCF7 Input Veh hg38 32350452 28158434 24928476 |
| Sample ID | Description | Chromosome | Start Position | End Position | Length |
|-----------|-------------|-------------|----------------|--------------|--------|
| jc5129    | MCF7 SNF5 JQ1 | hg38        | 26309562       | 23387954     | 15387723 |
| jc5130    | MCF7 SNF5 JQ1 | hg38        | 22665994       | 21912135     | 15430466 |
| jc5131    | MCF7 SNF5 4-OHT | hg38       | 24199036       | 21750646     | 14028128 |
| jc5132    | MCF7 SNF5 4-OHT | hg38       | 22655994       | 19921036     | 15387723 |
| jc5133    | MCF7 SNF5 4-OHT | hg38       | 23387954       | 21750646     | 14028128 |
| jc5134    | MCF7 SNF5 ICI | hg38        | 19047272       | 17210584     | 11738617 |
| jc5135    | MCF7 SNF5 ICI | hg38        | 28802176       | 25703016     | 14483818 |
| jc5136    | MCF7 SNF5 ICI | hg38        | 23882086       | 19439230     | 12346105 |
| jc5137    | MCF7 Input DCDT | hg38         | 22316665       | 21750646     | 14028128 |
| jc5138    | MCF7 Input DCDT | hg38        | 36076968       | 32728572     | 36021717 |
| jc5139    | MCF7 Input DCDT | hg38        | 35790484       | 31309656     | 13219005 |
| jc5140    | MCF7 Input DCDT | hg38        | 35747777       | 31440918     | 13219005 |
| jc5141    | MCF7 Input DCDT | hg38        | 41511061       | 36535728     | 26014669 |
| jc5142    | MCF7 Input DCDT | hg38        | 35747777       | 31440918     | 13219005 |
| jc5143    | MCF7 Input DCDT | hg38        | 40583489       | 35483403     | 25100466 |
| jc5144    | MCF7 Input DCDT | hg38        | 40583489       | 35483403     | 25100466 |

*Note: The above table provides information on different samples with their respective genomic coordinates and lengths.*
### Antibodies

For ChIP, following antibodies were used: ARID1A (Human protein atlas Sigma HPA005456, Lot I114190), BRG1 (abcam ab215998 Lot EPNCIR111A/GR296616-1), ER-alpha antibody (mix of abcam ab3575 Lot GR3217431-5 and Millipore 06-935 Lot 3045593), H3K27ac (Diagenode C15410196 Premium, Lot A1723-0041D), HDAC1 (Diagenode Premium C15410325, Lot A21-001P), ARID2 (Bethyl A302-229A, lot 2), SNF5 (Bethyl A301-087A), BRD4 antibody provided by Prof. Cheng Ming Chiang.

### Peak calling parameters

macs2 callpeak -t <chip> -c <input> -f BAM -g hs -n <chip name>-X-<input name> -q 0.05 -m 5 50 --nomodel

### Data quality

| Sample | # peaks |
|--------|---------|
| MCF7 ARID1A 4 OHT MA 38948 | |
| MCF7 ARID1A E2 MA 12663 | |
| MCF7 ARID1A ICI MA 26438 | |
| MCF7 ARID1A JQ1 MA 22916 | |
| MCF7 ARID1A VEH MA 21226 | |
| MCF7 ARID2 4 OHT ARID1A KNOCKOUT CLONE 11 1741 | |
| MCF7 ARID2 4 OHT ARID1A KNOCKOUT CLONE 14 4925 | |
| MCF7 ARID2 4 OHT PARENTAL WILD TYPE ECACC 18865 | |
| MCF7 ARID2 VEH ARID1A KNOCKOUT CLONE 11 1403 | |
| MCF7 ARID2 VEH ARID1A KNOCKOUT CLONE 14 993 | |
| MCF7 ARID2 VEH PARENTAL WILD TYPE ECACC 41128 | |
| MCF7 BRD4 4 OHT ARID1A KNOCKOUT CLONE 11 39181 | |
| MCF7 BRD4 4 OHT ARID1A KNOCKOUT CLONE 14 44153 | |
| MCF7 BRD4 4 OHT PARENTAL WILD TYPE ECACC 47501 | |
| MCF7 BRD4 VEH ARID1A KNOCKOUT CLONE 11 41575 | |
| MCF7 BRD4 VEH ARID1A KNOCKOUT CLONE 14 32261 | |
| MCF7 BRD4 VEH PARENTAL WILD TYPE ECACC 28840 | |
| MCF7 BRG1 4 OHT MA 61156 | |
| MCF7 BRG1 DCDT MA 59225 | |
| MCF7 BRG1 E2 MA 55731 | |
| MCF7 BRG1 ICI MA 55332 | |
| MCF7 BRG1 ICI MA 55332 | |
| MCF7 BRG1 ICI MA 54635 | |
| MCF7 BRG1 VEH MA 38195 | |
| MCF7 SNFS 4 OHT MA 46329 | |
| MCF7 SNFS DCDT MA 28960 | |
| MCF7 SNFS E2 MA 41828 | |
| MCF7 SNFS ICI MA 28737 | |
| MCF7 SNFS ICI MA 28800 | |
| MCF7 SNFS VEH MA 23893 | |
| MCF7 BRG1 4 OHT ARID1A KO CLONE 11 16018 | |
| MCF7 BRG1 4 OHT ARID1A KO CLONE 14 36144 | |
| MCF7 BRG1 4 OHT ECACC PARENTAL WT 65144 | |
| MCF7 BRG1 VEH ARID1A KO CLONE 11 14332 | |
| MCF7 BRG1 VEH ARID1A KO CLONE 14 32837 | |
| MCF7 BRG1 VEH ECACC PARENTAL WT 56301 | |
| MCF7 ER 4 OHT ARID1A KO CLONE 11 1839 | |
| MCF7 ER 4 OHT ARID1A KO CLONE 14 15618 | |
| MCF7 ER 4 OHT ECACC PARENTAL WT 28840 | |
| MCF7 ER VEH ARID1A KO CLONE 11 10247 | |
| MCF7 ER VEH ARID1A KO CLONE 14 11747 | |
| MCF7 ER VEH ECACC PARENTAL WT 20360 | |
| MCF7 H3K27AC 4 OHT ARID1A KO CLONE 11 30412 | |
| MCF7 H3K27AC 4 OHT ARID1A KO CLONE 14 39338 | |
| MCF7 H3K27AC 4 OHT ECACC PARENTAL WT 49142 | |
| MCF7 H3K27AC VEH ARID1A KO CLONE 11 32360 | |
| MCF7 H3K27AC VEH ARID1A KO CLONE 14 41045 | |
| MCF7 H3K27AC VEH ECACC PARENTAL WT 56864 | |
| MCF7 HDAC1 4 OHT ARID1A KO CLONE 11 13818 | |
| MCF7 HDAC1 4 OHT ARID1A KO CLONE 14 17413 | |
| MCF7 HDAC1 4 OHT ECACC PARENTAL WT 24430 | |
| MCF7 HDAC1 VEH ARID1A KO CLONE 11 15899 | |
| MCF7 HDAC1 VEH ARID1A KO CLONE 14 17358 | |
| MCF7 HDAC1 VEH ECACC PARENTAL WT 22619 | |
| MCF7 ARID1A DCDT SICONT 28649 | |
| MCF7 ARID1A DCDT SIFOXA1 23471 | |
Flow Cytometry

**Plots**

Confirm that:

- [x] The axis labels state the marker and fluorochrome used (e.g. CD4-FITC).
- [ ] The axis scales are clearly visible. Include numbers along axes only for bottom left plot of group (a 'group' is an analysis of identical markers).
- [ ] All plots are contour plots with outliers or pseudocolor plots.
- [ ] A numerical value for number of cells or percentage (with statistics) is provided.

**Methodology**

**Sample preparation**

Describe the sample preparation, detailing the biological source of the cells and any tissue processing steps used.

**Instrument**

Identify the instrument used for data collection, specifying make and model number.

**Software**

Describe the software used to collect and analyze the flow cytometry data. For custom code that has been deposited into a community repository, provide accession details.

**Cell population abundance**

Describe the abundance of the relevant cell populations within post-sort fractions, providing details on the purity of the samples and how it was determined.

**Gating strategy**

Describe the gating strategy used for all relevant experiments, specifying the preliminary FSC/SSC gates of the starting cell population, indicating where boundaries between "positive" and "negative" staining cell populations are defined.

[ ] Tick this box to confirm that a figure exemplifying the gating strategy is provided in the Supplementary Information.