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# Nucleic Acids Research Papers on Drugs Affecting Androgen Receptor Binding, Epigenetic Drugs, More

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A University of Michigan team describes distinct androgen receptor effects for compounds depending on their DNA response element interactions in prostate cancer. Using a combination of high-throughput drug screening, fluorescent reporter plasmids, transcriptomics, chromatin immunoprecipitation assays, and other approaches, the researchers screened thousands of compounds, searching for those that curbed cancer growth but spared growth by normal cells. In particular, they focused on drugs that appeared to have distinct effects on genes with consensus- or selective androgen response elements, which were predicted to affect proliferation and differentiation, respectively. The strategy led them to doxorubicin, which appeared to prompt distinct androgen receptor binding interactions when applied to cells at different concentrations.

University of California, Los Angeles, researchers outline an approach for evaluating the consequences of epigenetic modulating drugs. The approach — known as the "Massive and Parallel Measurement of Epigenetic Drug Sensitivity," or MAPMEDS — brings together DNA barcode, single-cell expression, reporter protein, and other data sources to compare locus-specific drug effects with effects across loci more broadly, the team says. "Using this platform, we discovered widespread loci-specific sensitivities to epi-drugs for three distinct epi-drugs that target histone deacetylase, DNA methylation, and bromodomain proteins," the authors report, adding that "[b]y leveraging ENCODE data on chromatin modification, we identified features of chromatin environments that are most likely to be affected by epi-drugs."

A University of Colorado team shares a method for finding and analyzing so-called "re-replicated DNA" — sequences in that undergo more than one round of replication per cell cycle, potentially contributing to some gene amplifications or aneuploidy. To assess such sequences, the researchers came up with a selective fragmentation- and enrichment-based sequencing method dubbed Rerep-seq, which they validated in a series of simulation analyses and experiments in human and *Saccharomyces cerevisiae* budding yeast cells. In the latter experiments, for example, the authors saw signs that re-replication is non-random and "spans multiple origins of replication and cross-topological boundaries."

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