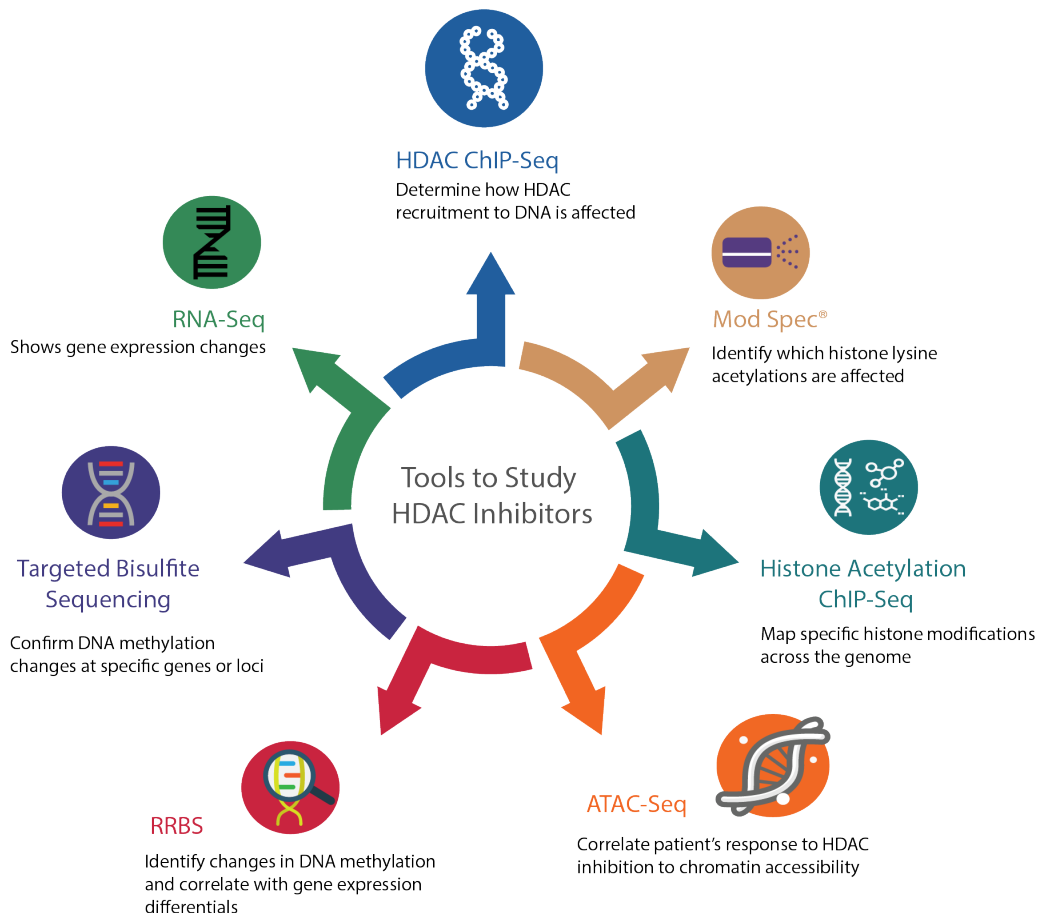# HDAC Assay Panel

Tools to study epigenetic and gene regulatory effects of HDAC inhibitors

- HDAC inhibitors are used clinically to treat diseases like cancer, neurodegenrative disorders, inflammatory and cardiovascular diseases.

- HDAC treatment results in changes in gene expression, chromatin accessibility, histone acetylation and DNA methylation.

- Our epigenetic and gene regulation services portfolio will help illuminate the mechanistic response of any experimental cell system to HDAC inhibition.


Tools to Study HDAC Inhibitors

HDAC ChIP-Seq
Determine how HDAC recruitment to DNA is affected

Mod Spec®
Identify which histone lysine acetylations are affected

Histone Acetylation ChIP-Seq
Map specific histone modifications across the genome

ATAC-Seq
Correlate patient's response to HDAC inhibition to chromatin accessibility

RRBS
Identify changes in DNA methylation and correlate with gene expression differentials

Targeted Bisulfite Sequencing
Confirm DNA methylation changes at specific genes or loci

RNA-Seq
Shows gene expression changes

HDAC assays can be carried out with cell culture, tissues from animal models, human blood cells, and human biopsies.

HDAC ASSAY PANEL

Using genome-wide data sets to understand mechanisms of HDAC inhibition

Next-Generation sequencing (NGS) based technologies have become integral to understanding mechanism of action for many therapeutic targets, like HDAC inhibitors. It is now increasingly common to perform multiple different "omics" assays and overlay the data in order to identify important genes and pathways. Active Motif offers multiple assays to explore gene expression, transcription factor binding, histone modifications, DNA methylation and chromatin structure.
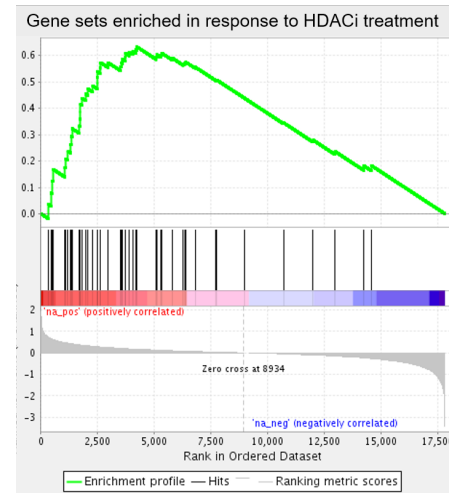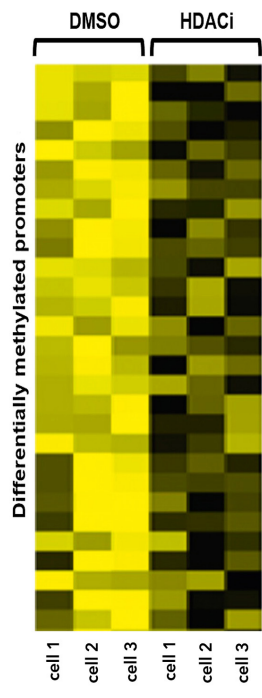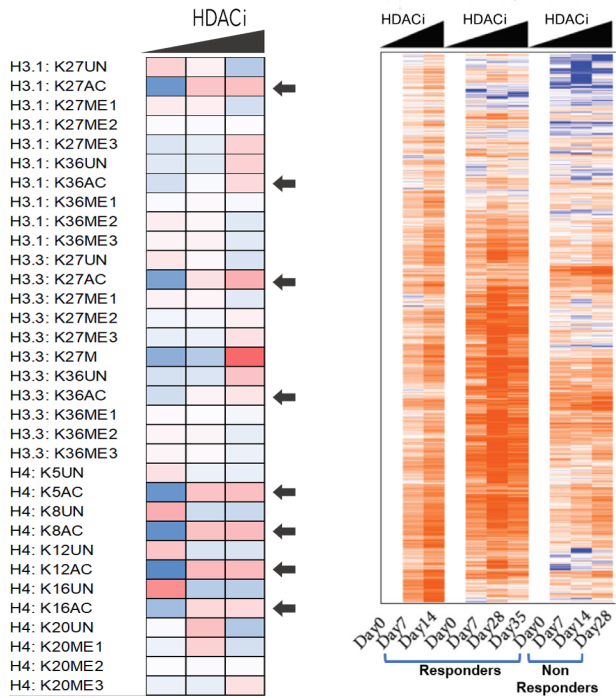

Data integration of RNA-Seq reveals important genes and pathways

Mod Spec® mass spec detection of histone modifications was used to identify histone lysine residues that become acetylated after HDACi treatment.

ATAC-Seq on patient blood cells. Chromatin openness after HDACi treatment correlated with positive patient outcomes.

RRBS was used to identify differentially methylated promoters from an untreated and HDACi treated human cell lines.
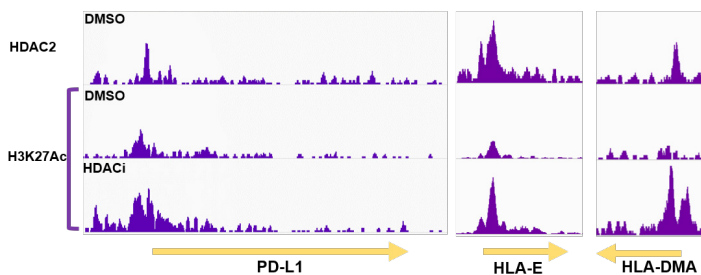
CHIP-Seq was used to map HDAC2 binding and H3K27ac occupancy across the genome in an immuno-oncology model. ChIP-Seq data show that HDACi augments checkpoint inhibitor therapy by promoting enhanced antigen presentation expression at checkpoint proteins through increased histone acetylation at those genes.

✓ **ATAC-Seq:** Profile open chromatin landscapes

✓ **RNA-Seq:** Measure gene expression

✓ **RRBS:** Quantify DNA methylation at over 4 million CpGs

✓ **Mod-Spec®:** Detect changes in global levels of histone modifications

✓ **HDAC ChIP-Seq**: Map HDAC binding across the genome

✓ **Histone ChIP-Seq**: Map histone modifications across the genome.