



Broaden the impact of your screening with scalable transcriptomics.

The challenge:

Phenotypic screens show what happened, but not always why.

MoA blind spot

Phenotypic readouts rank hits by severity but often provide limited insight into mechanism.

Hit prioritization cap

Without transcriptional context, morphological hits advance with similar confidence, increasing risk of pursuing off-target or low-value compounds.

Off-target risk undetected

Imaging and viability may not capture pathway-level off-target activity. Gene-expression signatures reveal unintended biology before *in vivo* studies.

The solution:

Alithea's MERCURIUS™ DRUG-seq & Total DRUG-seq technology

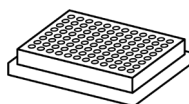
Revvity is an authorized distributor of:

ALITHEA
GENOMICS



Extraction-free

Direct from cells. Eliminates the need for RNA isolation.



Early multiplexing

Sample barcoding enables pooling of up to 1,000+ conditions in a single tube.



Genome-wide coverage

Gene-expression signatures across the genome.



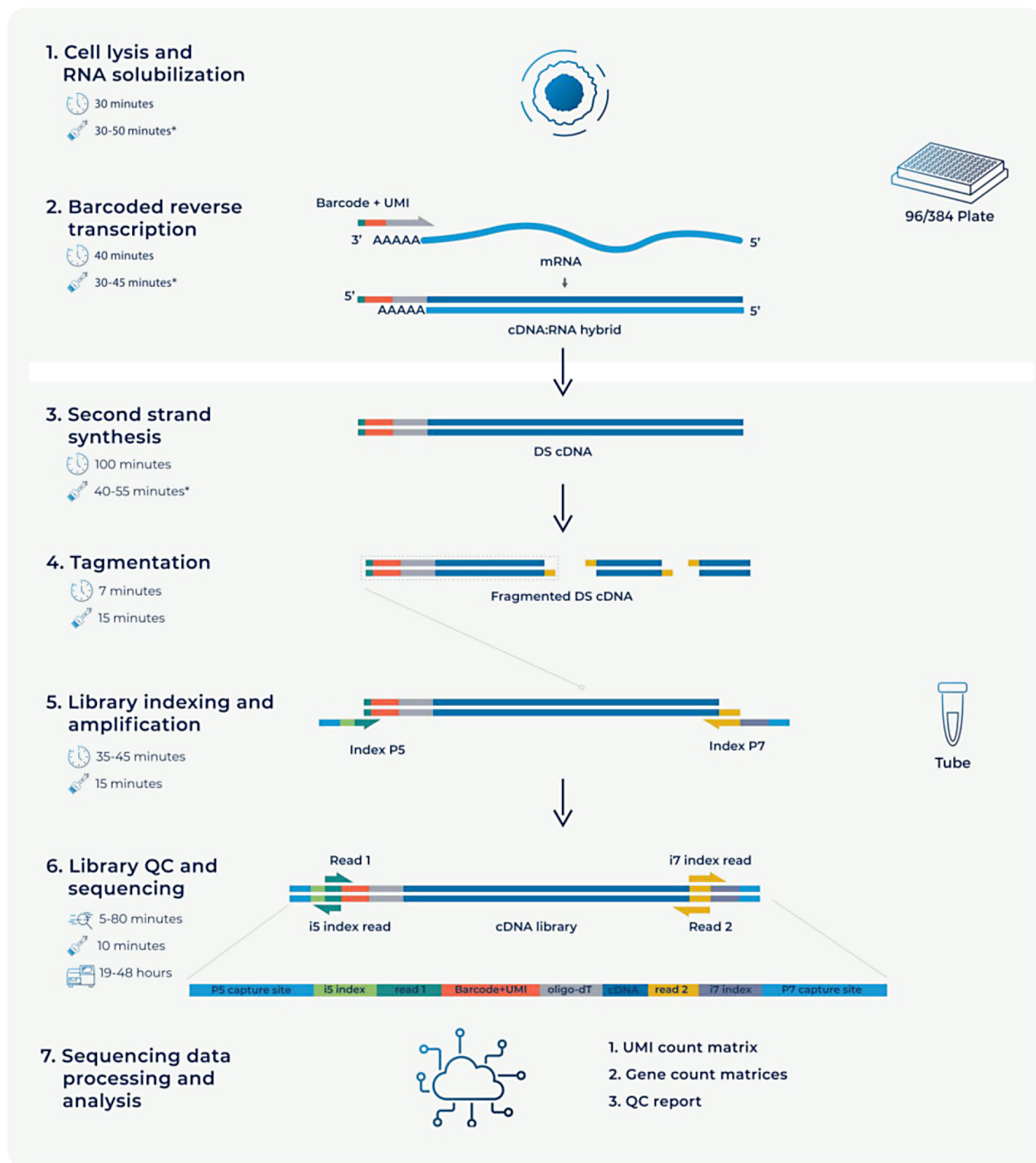
Ultra low-cost

Significantly cheaper than conventional alternatives.

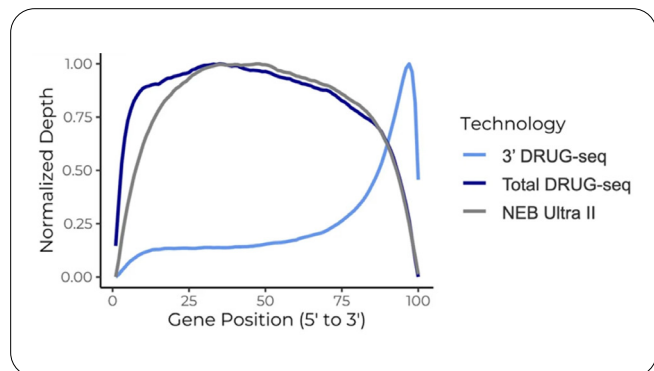
100+ labs using this technology worldwide **50+** publications

Workflow at a glance

The DRUG-seq workflow begins by adding the proprietary lysis buffer directly to pre-washed and frozen cell in 96- or 384-well-plates. The lysates can then be used directly in the DRUG-seq reverse transcription reaction, in which individual RNA samples are “tagged” with a specific barcode and each RNA molecule is marked with a unique molecular identifier (UMI). All samples are subsequently pooled into one single tube and purified. Library amplification is performed with unique dual indexes to maximize the efficiency of library demultiplexing during next-generation sequencing.



Comparable performance



Total DRUG-seq displays uniform coverage across the full transcript length, matching NEBNext® Ultra™ II Total RNA preparations. Standard DRUG-seq shows characteristic 3' enrichment, reflecting its 3'-end capture design.

Up to **384** preps in a single tube

3,000+ genes detected
at 1M reads/sample

0 extraction steps needed

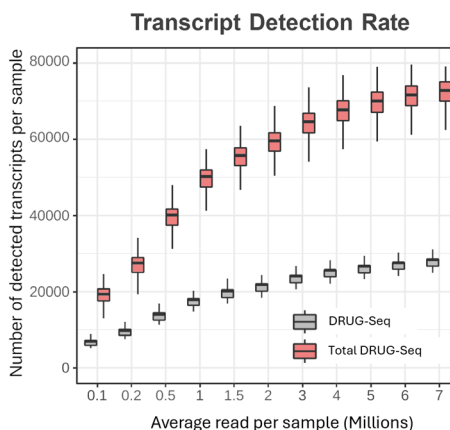
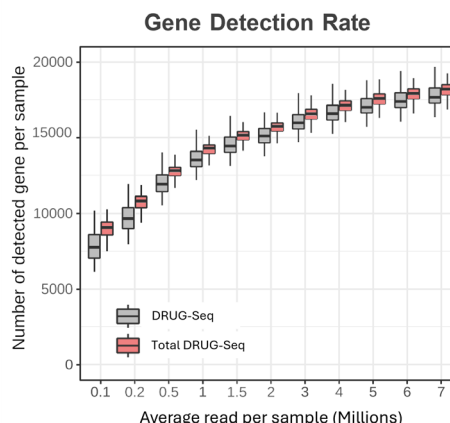
Selection guide

MERCURIUS™ DRUG-seq

Part number	Total preps	Plate format
NOVA-10841	96	1x96
NOVA-11041	384	4x96
NOVA-10851	384	1x384
NOVA-11051	1,536	4x384

MERCURIUS™ Total DRUG-seq

Part number	Total preps	Plate format
NOVA-10705	96	1x96
NOVA-11661	384	4x96
NOVA-10706	384	1x384
NOVA-11662	1,536	4x384



Both protocols detect comparable numbers of genes across all read depths. At the transcript level, Total DRUG-seq detects up to ~2.5-fold more transcripts than 3' DRUG-seq at equivalent depths, reflecting its full-length coverage and ability to distinguish isoforms.

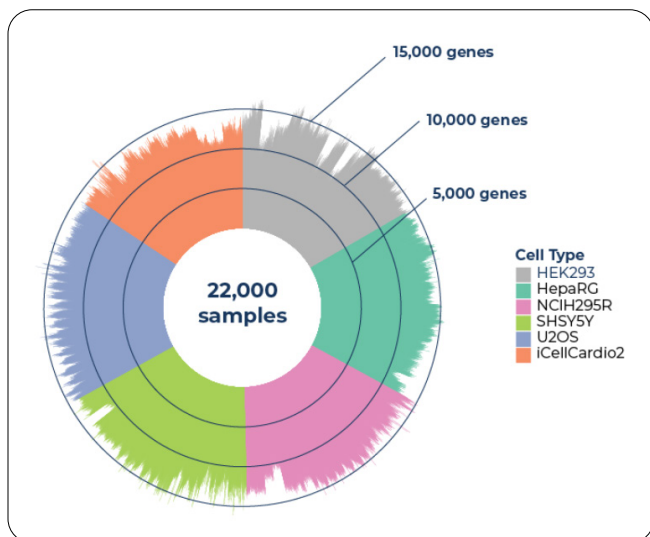
- **DRUG-seq** enables high-throughput profiling with efficient gene-expression signatures for large-scale screens.
- **Total DRUG-seq** extends this approach to full transcriptome resolution, capturing isoforms and non-coding RNA for deeper biological insight.

Cell input range

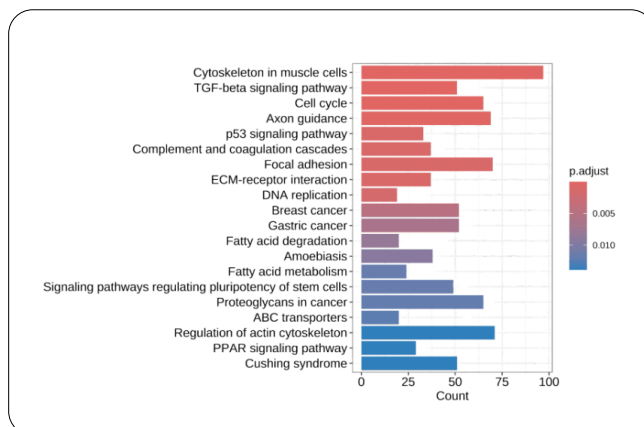
- **DRUG-seq:** 15k-50k (96-well) / 2k-10k (384-well)
- **Total DRUG-seq:** 5k-25k (96-well) / 2k-10k (384-well)
- List of validated cell lines/tissues can be provided upon request.

MERCURIUS™ DRUG-seq in action

The examples below show how transcriptome-wide readouts can reveal mechanisms of action, detect combination effects, and generate actionable insights across large screening campaigns.



Large scale studies: Gene detection overview of a case study on 22,000 samples and 51 compounds in four doses, on 6 cell lines, sequenced at 1M reads per sample. Should we maybe add in writing that the average detection was 13000 genes detected per sample?



Enables detailed pathway analysis of biological processes: Interrogate the molecular pathways and identify altered biological processes.

What do customers say

I was so satisfied that I've already recommended to my colleagues.

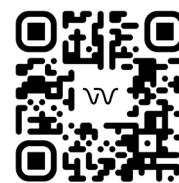
Yvan Martineau, Ph.D.
Senior Scientist, CRCT

The DRUG-seq platform enabled us to systematically profile compound-induced transcriptional responses at scale, generating a rich dataset that helped prioritize testable hypothesis.

Mikołaj Stabicki, Ph.D.
PI at the MGH Krantz Family Center for Cancer Research, Assistant Professor of Medicine at Harvard Medical School

Recent publications

- Anstett et al. (2025). *In Vitro* Evaluation of the Safety and Efficacy of Cibisatamab Using Adult Stem Cell-Derived Organoids and Colorectal Cancer Spheroids. *Cancers* (Basel). 17(2):291. doi: 10.3390/cancers17020291.
- Sousa et al. (2025). CRIPTO's multifaceted role in driving aggressive prostate cancer unveiled by in vivo, organoid, and patient data. *Oncogene* 44(7):462-475. doi: 10.1038/s41388-024-03230-x.



Learn more

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