CeMM Research Center for Molecular Medicine of the Austrian Academy of Sciences

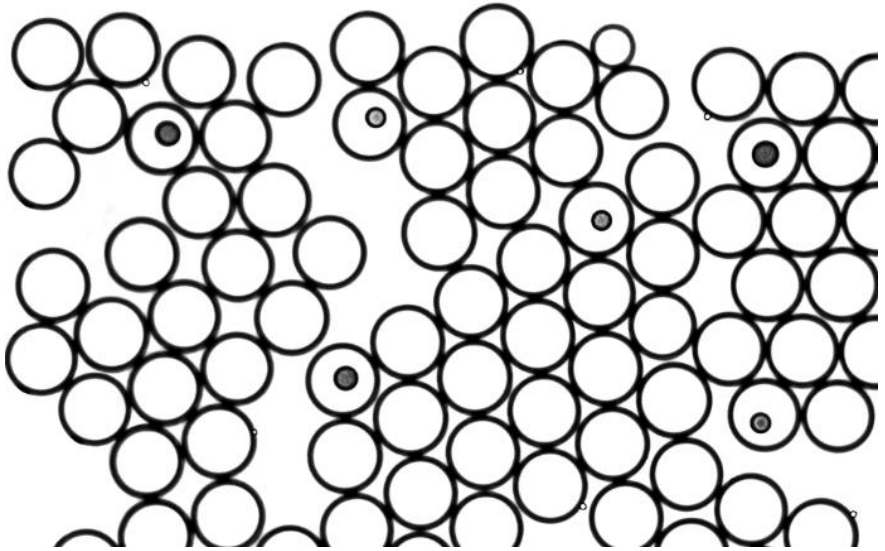
MEDICAL UNIVERSITY OF VIENNA

Programmed Cells?

Epigenetics and engineering of immunity

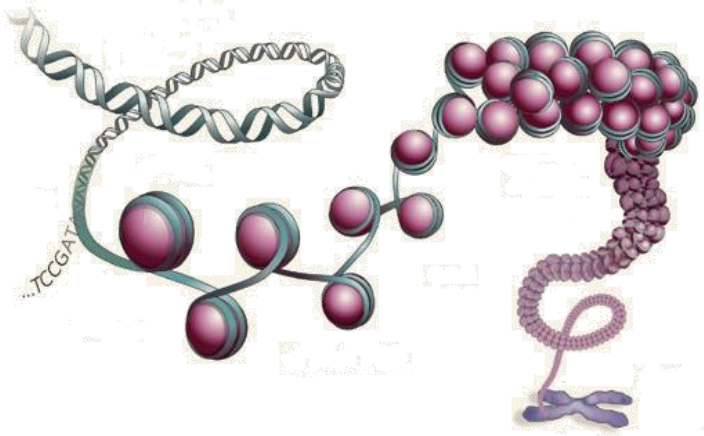
Christoph Bock

21 September 2023



https://bocklab.org
https://twitter.com/BockLab

Epigenetic mechanisms constitute a layer of genome regulation beyond the DNA sequence

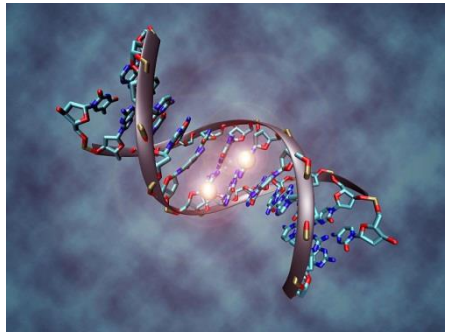


1D: Genome sequence

- Protein-coding genes
- Regulatory elements

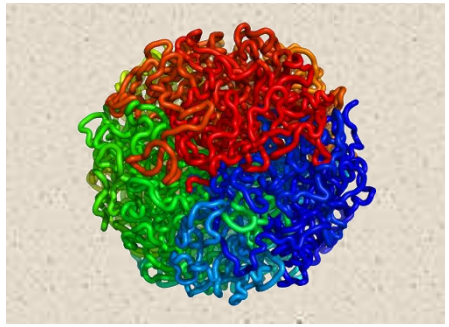
“2D”: Epigenetic marks

- DNA methylation
- Histone modifications



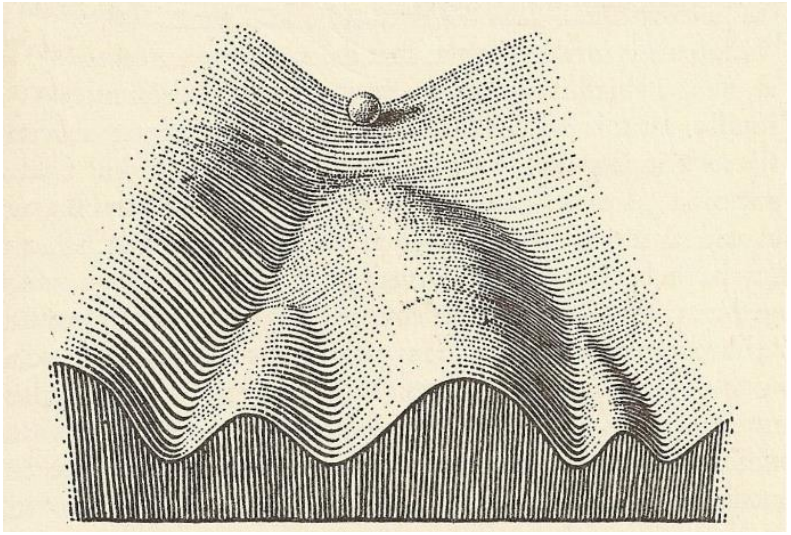
3D: Nuclear organization

- Chromatin domains
- Promoter-enhancer links

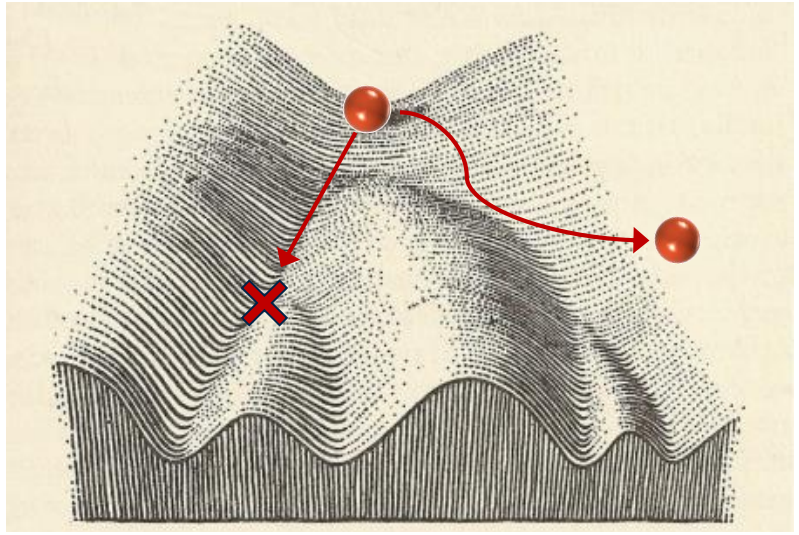


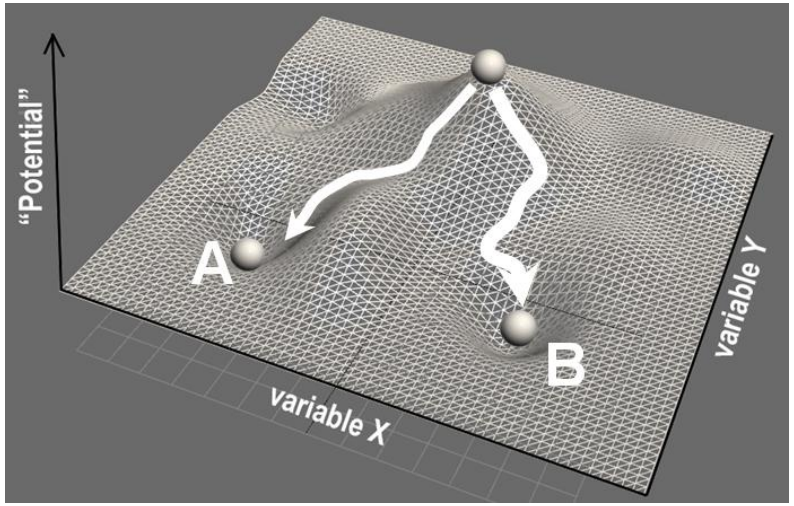
The epigenetic landscape visualizes cell states, their developmental past & future potential

Epigenetic landscape (Waddington 1957)

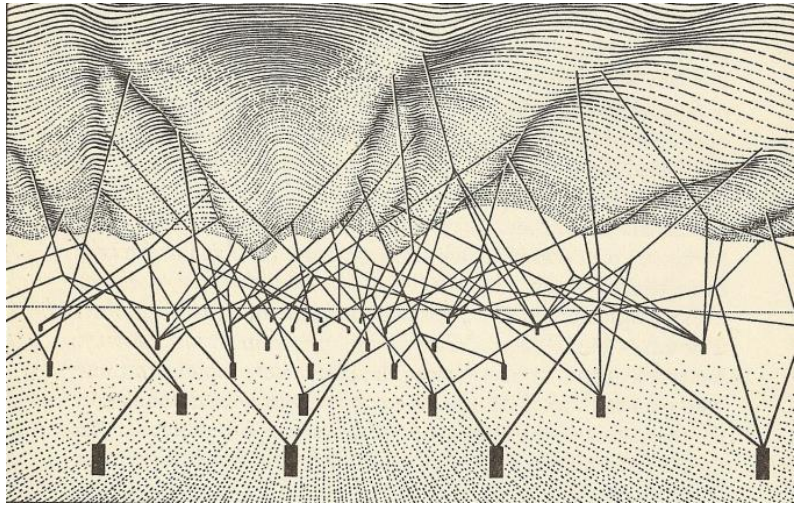


Differentiation blocks and aberrations in cancer



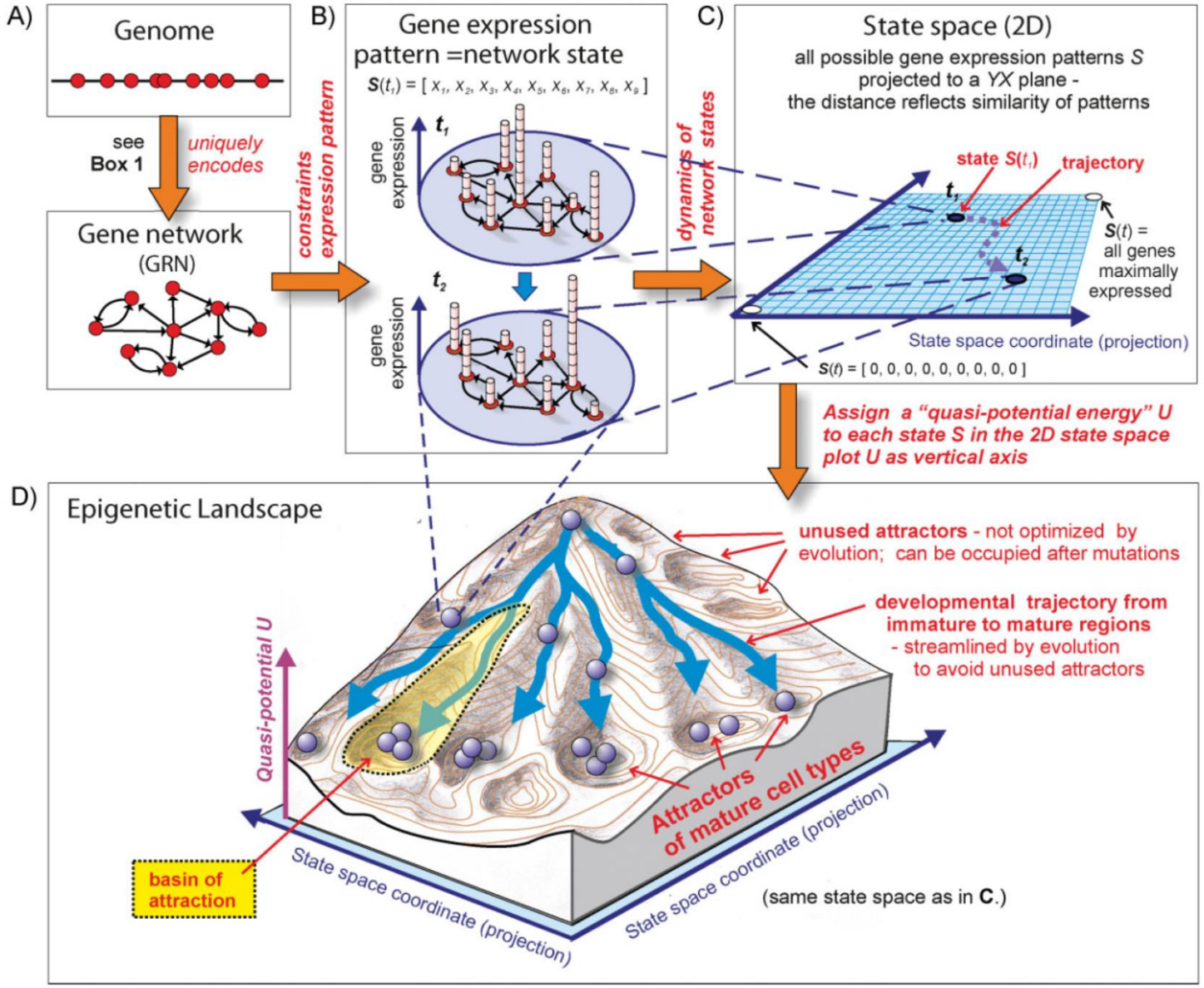


Epigenetic memory of cell state trajectories



Gene regulatory network underlying the landscape

Goal: A quantitative, disease-relevant & predictive model of the epigenetic landscape



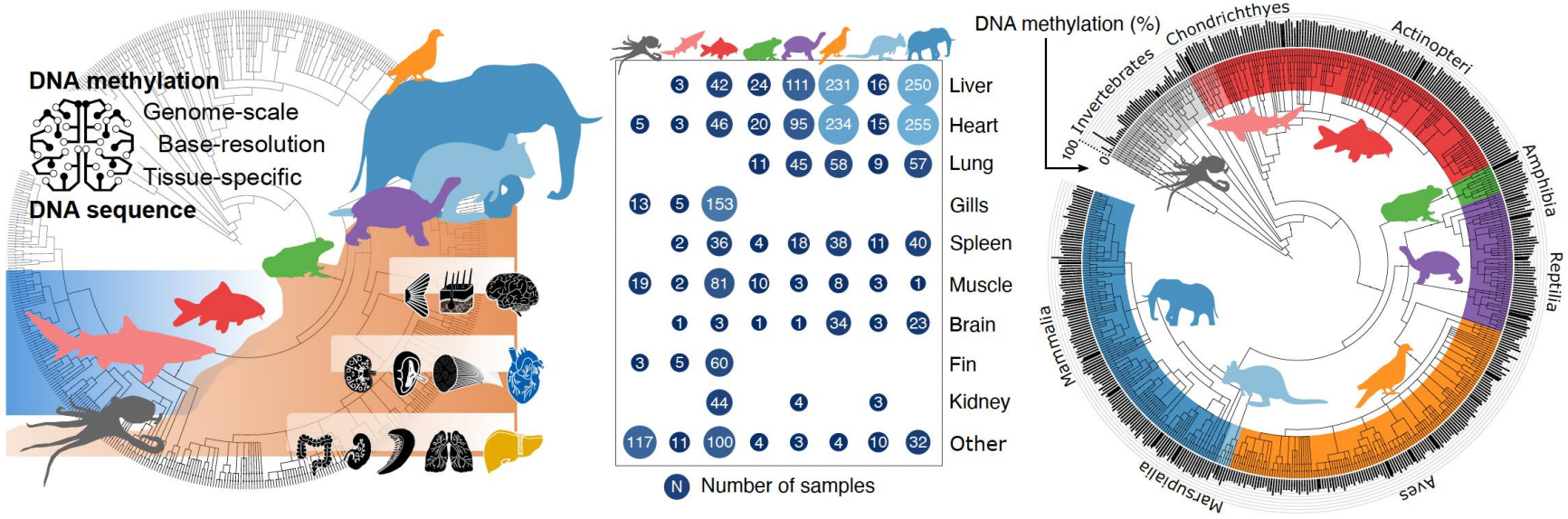
Definition & diagram by Sui Huang (ISB Seattle)

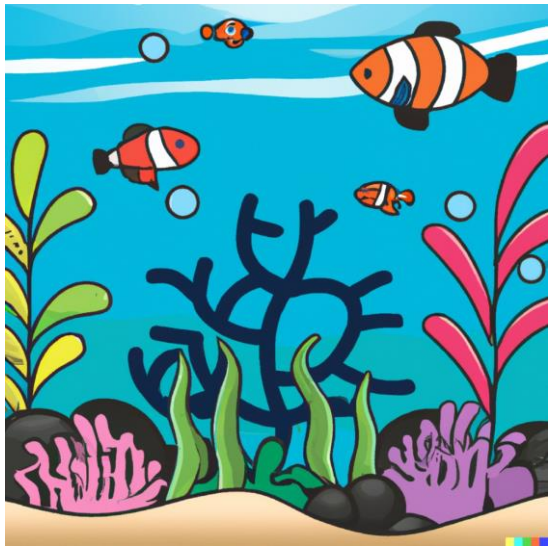
Huang (2011) BioEssays (http://doi.org/10.1002/bies.201100031)

Epigenetic regulation by DNA methylation is deeply conserved in vertebrate evolution

DNA methylation mapped (by RRBS) and analyzed across 580 animal species & 2443 tissue samples

- DNA methylation and DNA sequence are closely linked (DNA trimers predict local DNA methylation)
- We can predict DNA methylation profiles across species, for example octopus to elephant (ROC-AUC 0.76)
- Cancer risk and Peto's paradox: DNA methylation may help protect long-lived birds & mammals against cancer

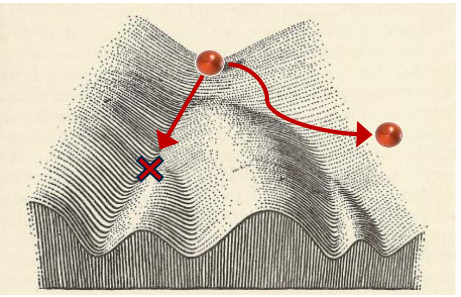




Presentation outline

1. Developmental history and epigenetic cell states in immune diseases

Immune cells “remember” their differentiation history and re-use regulatory processes of normal development in immune diseases



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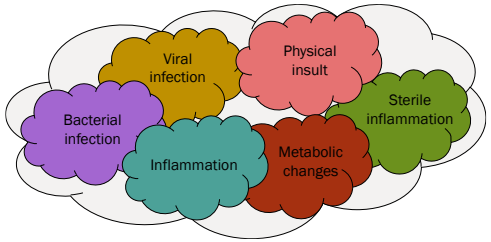
Hematopoietic and non-hematopoietic structural cells implement an epigenetic potential for rapid immune gene activation upon challenge



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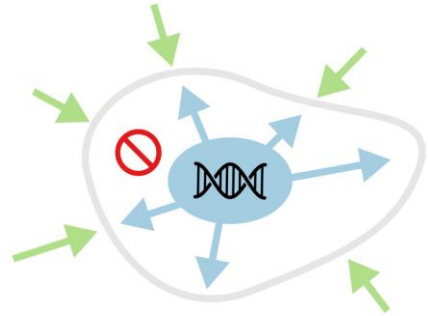
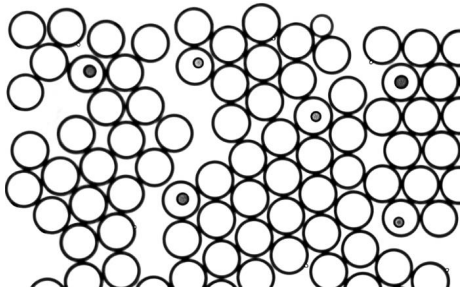
Epigenetic and transcription-regulatory profiles identify cancer cells-of-origin, detect disease progression, and prioritize potential therapies



4. Rational programming of human cells for biomedical applications

Interpretable deep learning, CRISPR single-cell sequencing, and patient-derived organoids facilitate mechanistic biology at scale

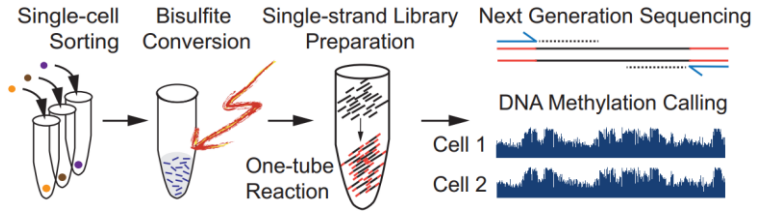


Reconstructing cellular differentiation hierarchies from epigenetic data

An early proof-of-concept focusing on the human blood lineage

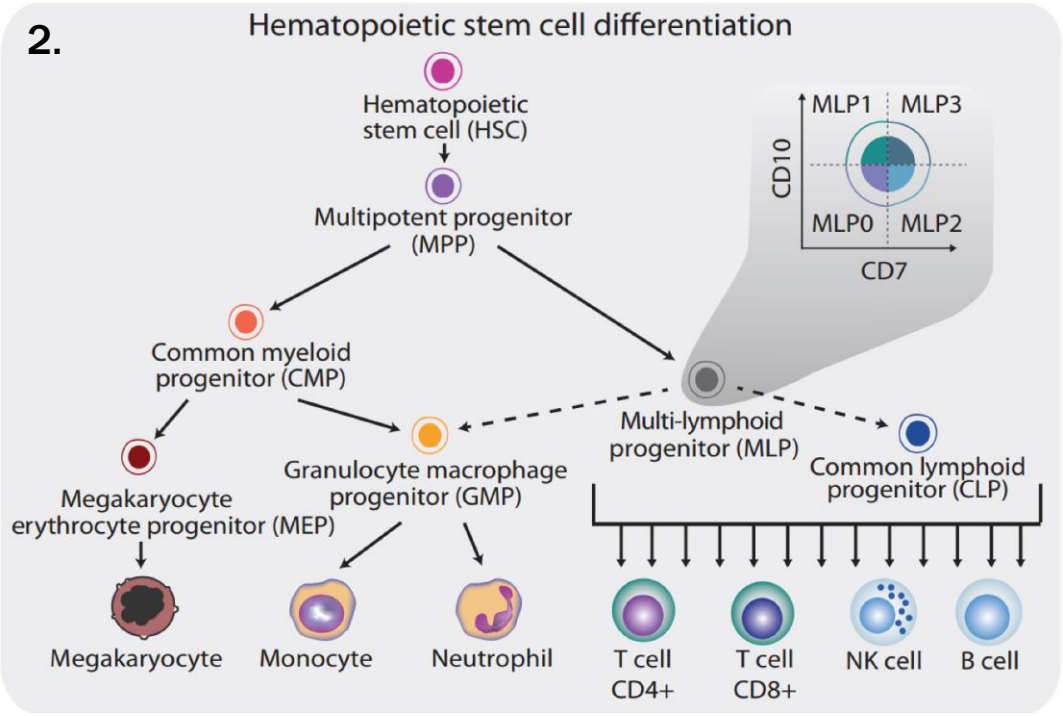
1. We developed a scalable method for single-cell DNA methylation profiling
2. Applied it to the epigenomes of FACS-enriched stem/progenitor cells
3. Performed bioinformatic lineage reconstruction using machine learning

1.



scWGBS: Farlik, Sheffield et al. (2015) Cell Reports (http://doi.org/10.1016/j.celrep.2015.02.001)

2.



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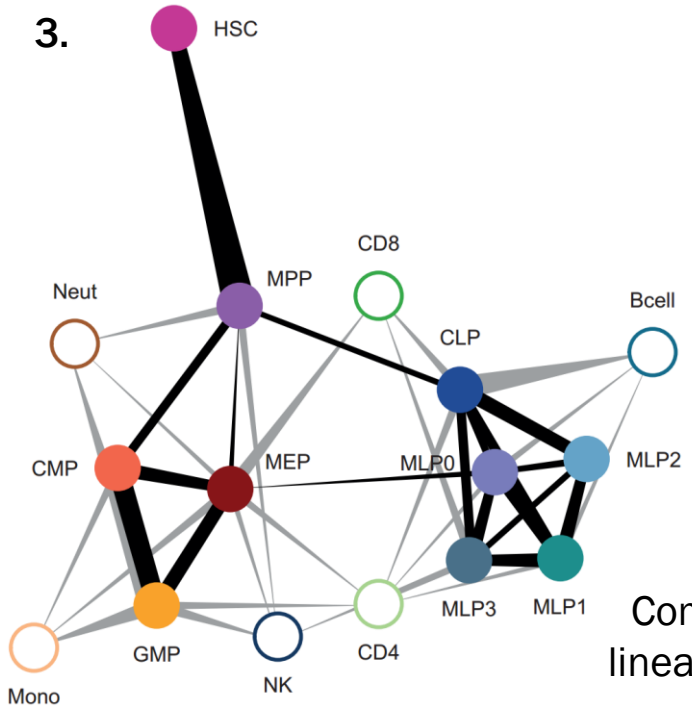


Computational lineage inference

Collaboration:

BLUEPRINT epigenome

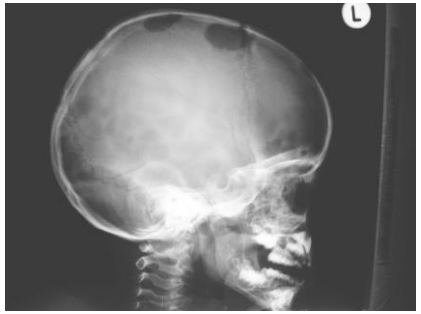
IHEC International Human Epigenome Consortium

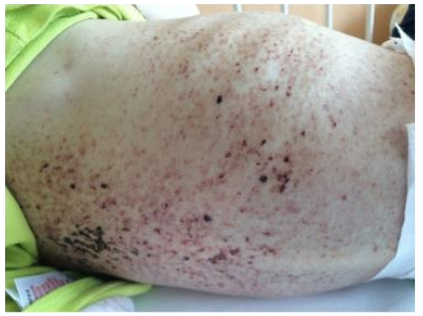
Farlik, Halbritter, Müller et al. (2016) Cell Stem Cell (http://doi.org/10.1016/j.stem.2016.10.019)

An unexpected epigenetic landscape in a rare childhood disease

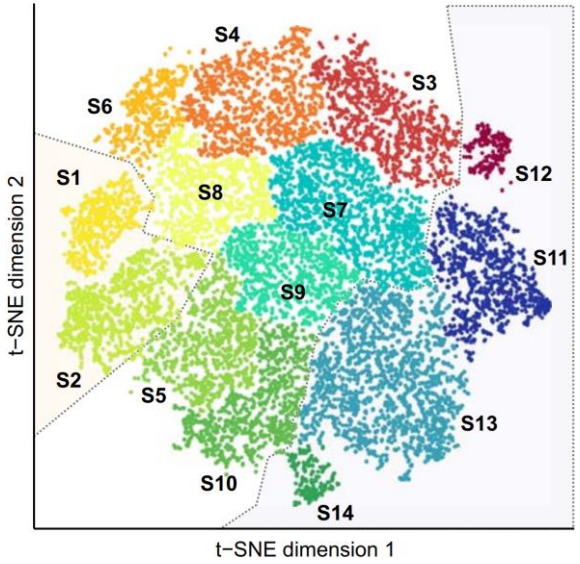
Langerhans cell histiocytosis: A cancer? An autoimmune disease?

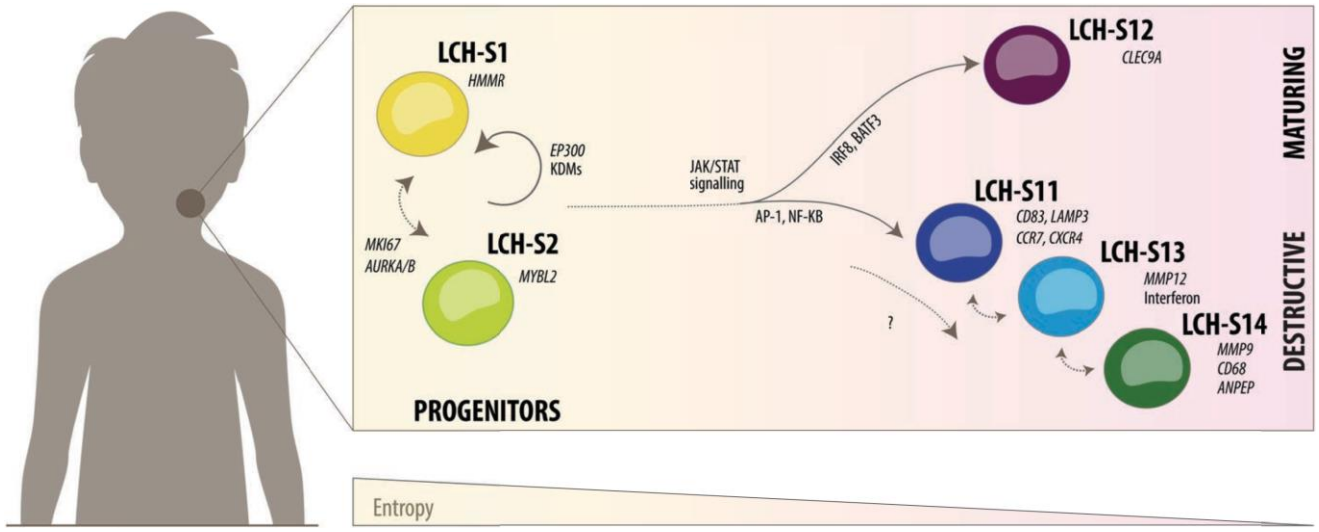
- Rare pediatric disease: <1 case per 100,000, most patients survive
- Pathology: Accumulation of CD1a+ CD207+ cells in various tissues
- Hybrid position between a cancer (BRAF V600E) and an autoimmune disease (inflammation, no genetic evolution)





Single-cell RNA-seq, ATAC-seq unravels developmental hierarchy *in situ*:





Collaboration:

St. Anna Kinderkrebs Forschung

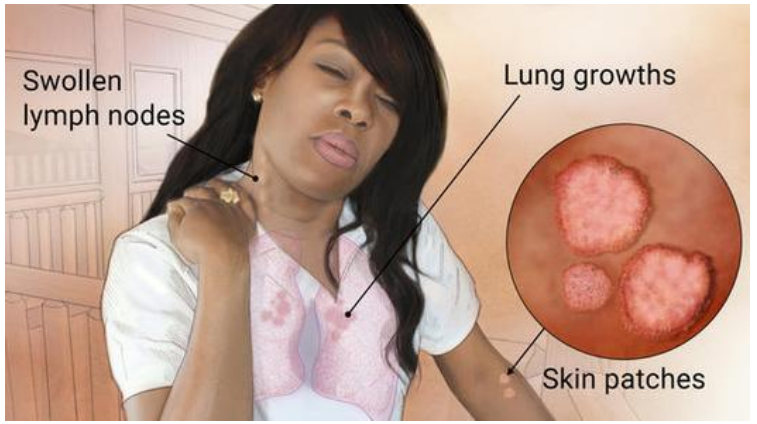
LUDWIG BOLTZMANN INSTITUTE Rare and Undiagnosed Diseases

Halbritter, Farlik et al. (2019) Cancer Discovery (http://dx.doi.org/10.1158/2159-8290.CD-19-0138)

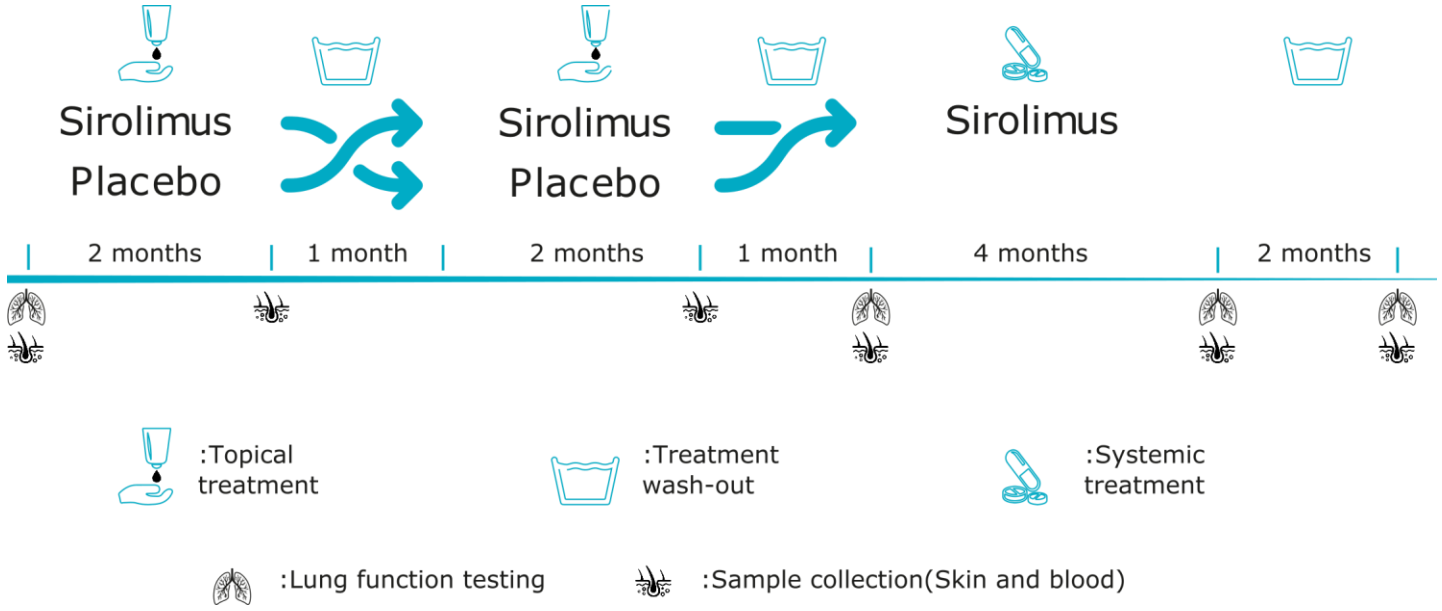
Single-cell and spatial profiling for clinical trials monitoring

Sarcoidosis: Granuloma formation of unknown cause

- 5 to 40 cases per 100,000, frequency highly variable by genetic ancestry
- Affecting skin, lung, and other organs, <5% mortality, substantial morbidity
- Few treatment options – but initial data that mTOR is critical for granuloma formation in a mouse model of sarcoidosis (Linke 2017 Nature Immunology)



N-of-1 clinical trial for mTOR inhibition



Collaboration:

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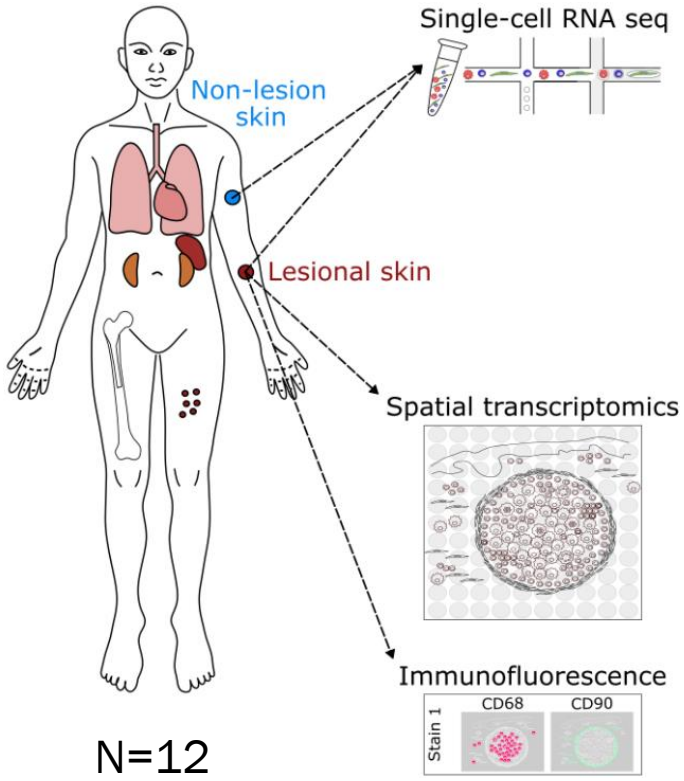
LUDWIG BOLTZMANN INSTITUTE Rare and Undiagnosed Diseases

Redl et al. (2023) Lancet Rheumatology, in press

Results of single-cell and spatial profiling at baseline of the trial



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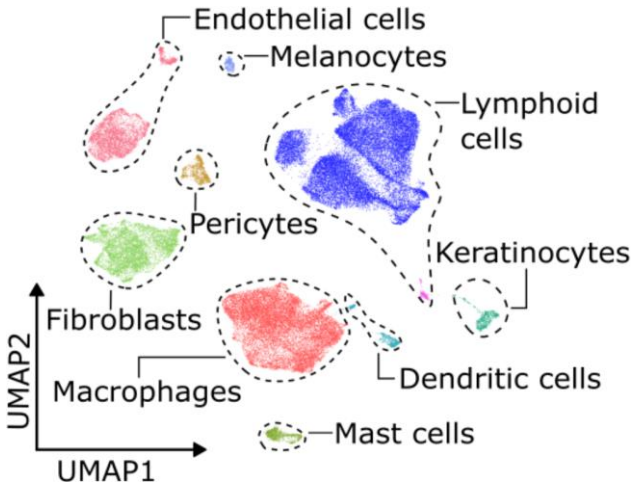

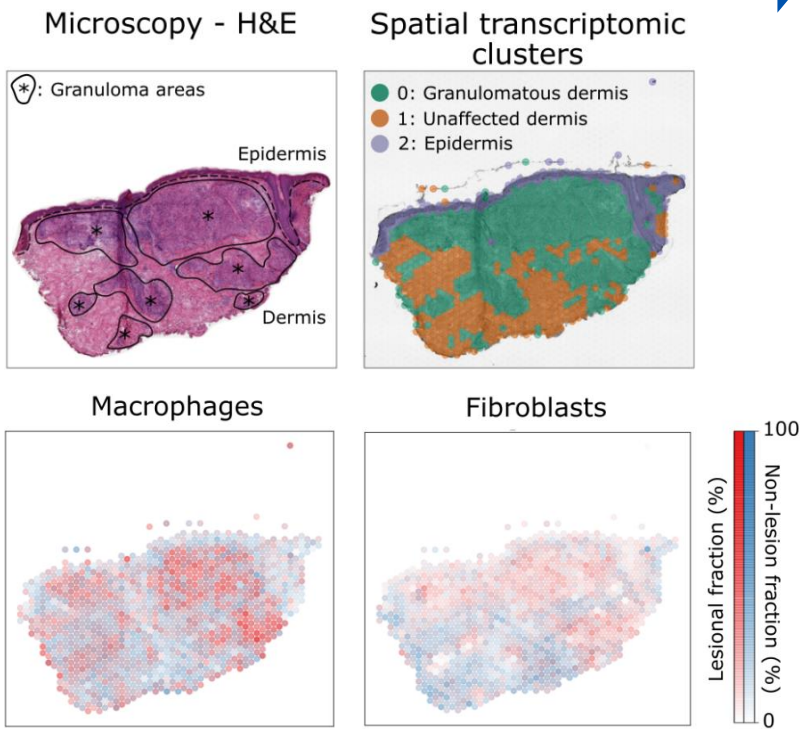

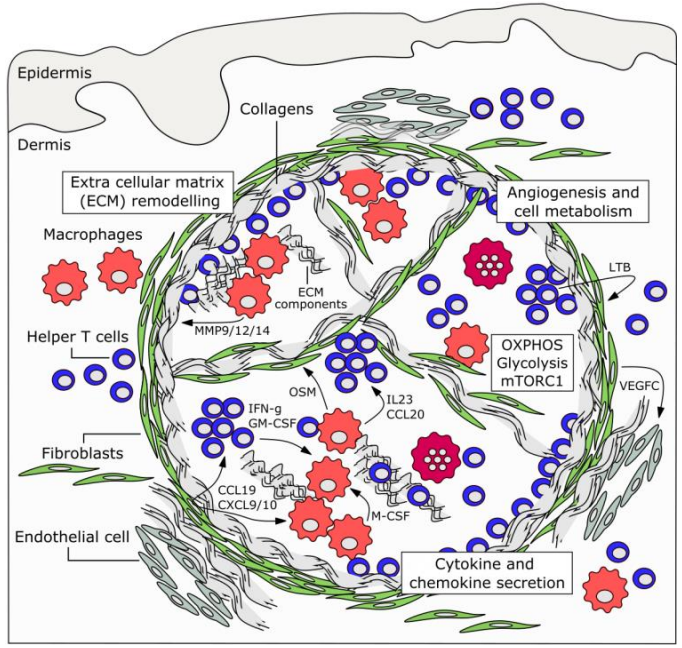

Conclusions

- Pathogenic macrophages and fibroblasts support granulomas
- Granulomas exploit elements of lymphoid organ development

Krausgruber, Redl, Barreca et al. (2023) Immunity (http://dx.doi.org/10.1158/2159-8290.CD-19-0138)

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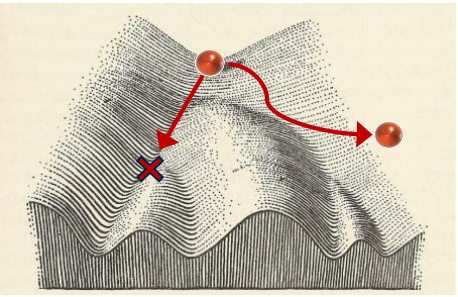

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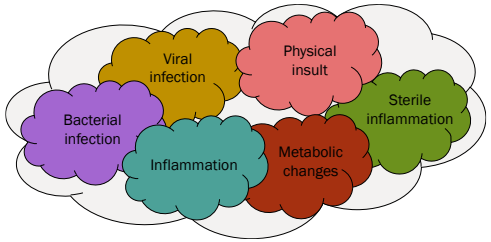

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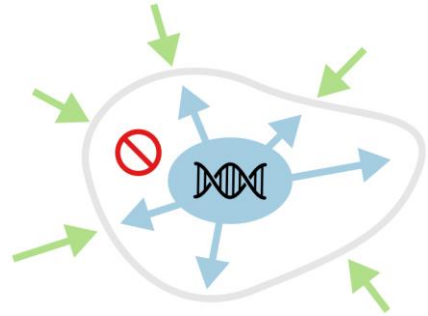

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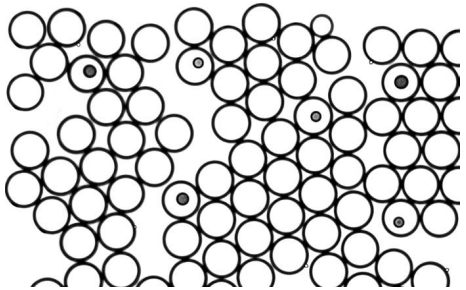
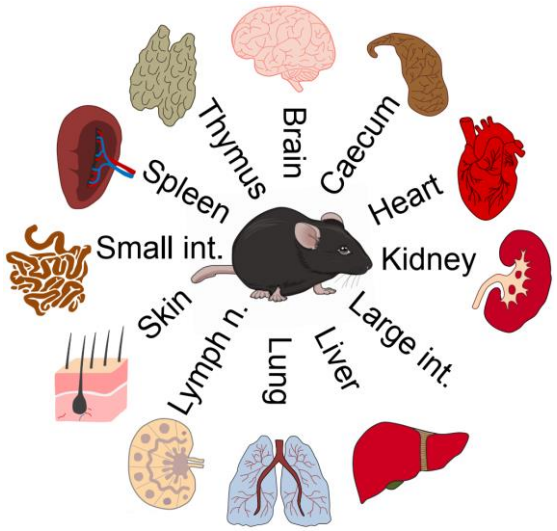
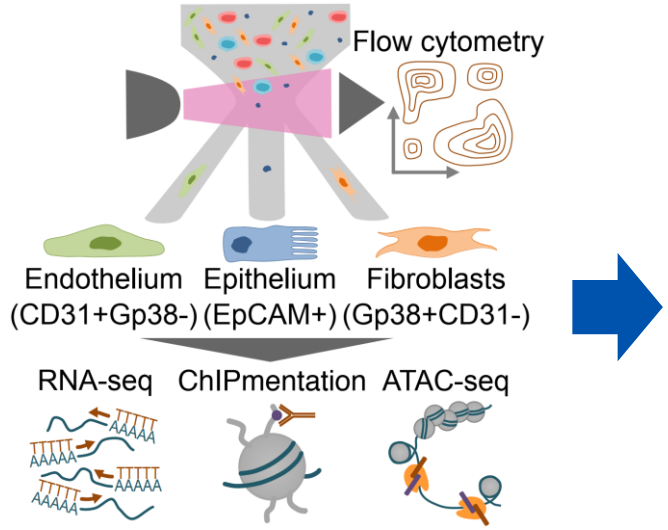


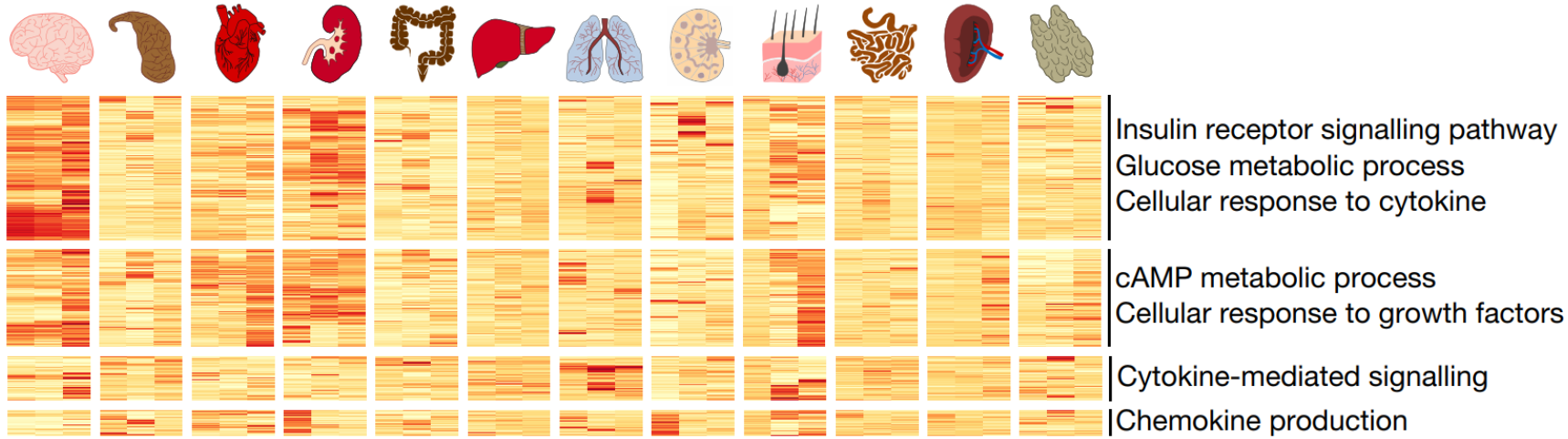
Epigenetic states capture the cells' developmental past – can they predict their future?





Structural cells: Giving shape to our body, contributing to host immunity?

- We performed epigenome & transcriptome profiling of "structural cells"
- Epithelium, endothelium, fibroblasts were analyzed across 12 organs
- We found widespread activity of immune genes in these non-immune cells

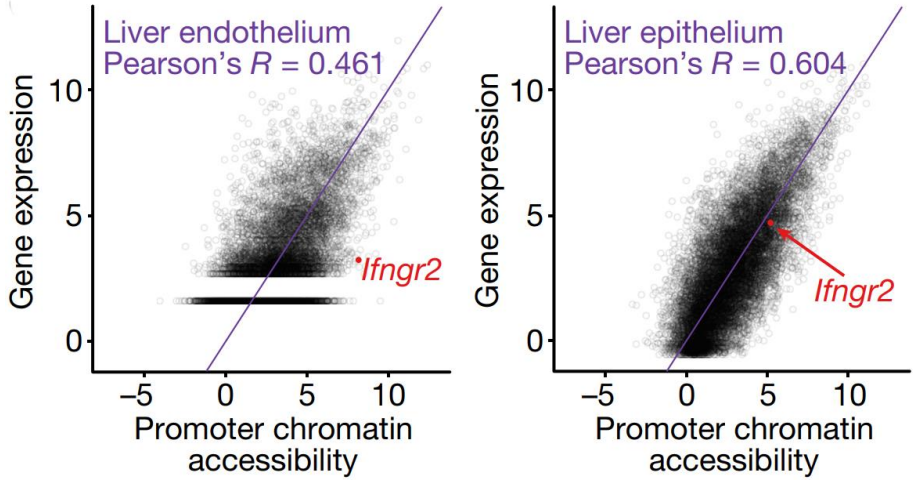


Krausgruber, Fortelny et al. 2020 Nature (https://nature.com/articles/s41586-020-2424-4)

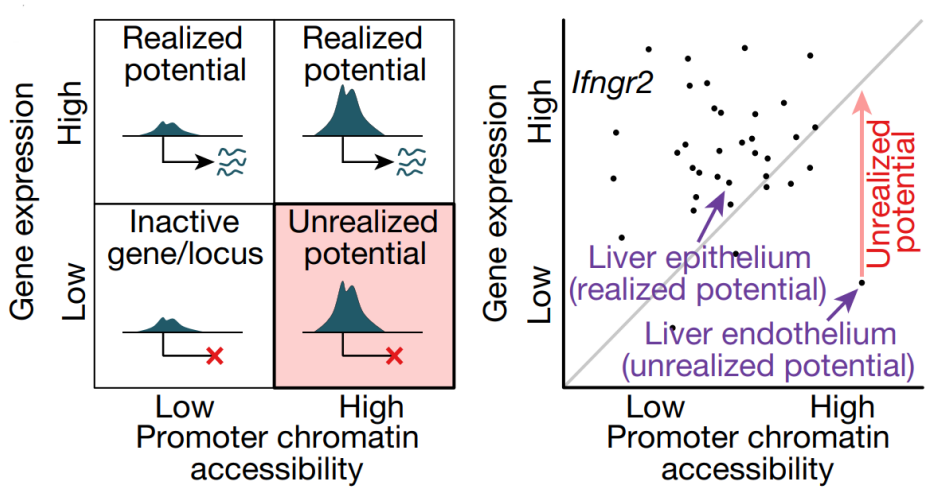
An epigenetic potential for immune gene activation in structural cells

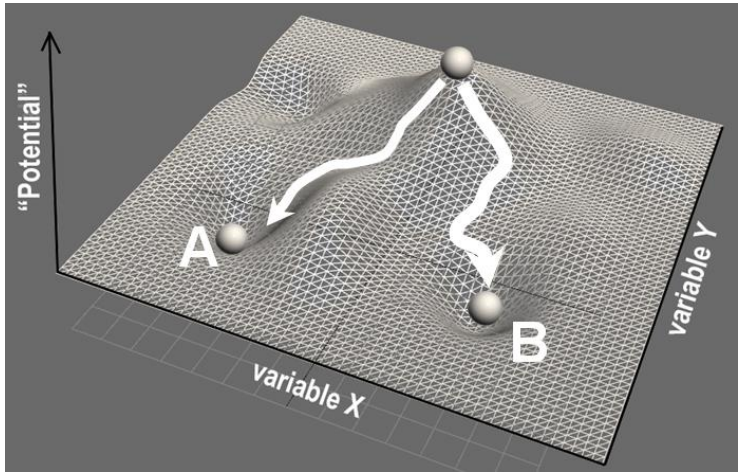
Integrative analysis of epigenome (ATAC-seq) and transcriptome (RNA-seq) data

- Open chromatin at promoter regions is correlated with high gene expression
- BUT: Some genes have widely open promoters yet low levels of gene expression
- These genes are enriched for immune functions



Hypothesis: These immune genes carry an epigenetic potential for rapid activation

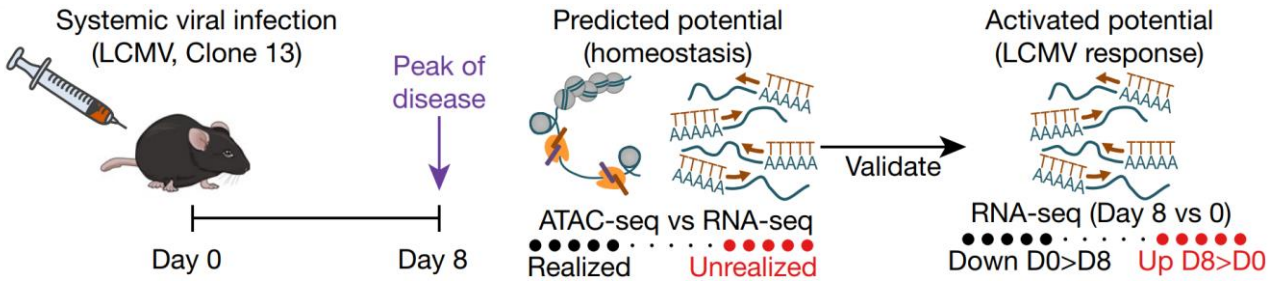


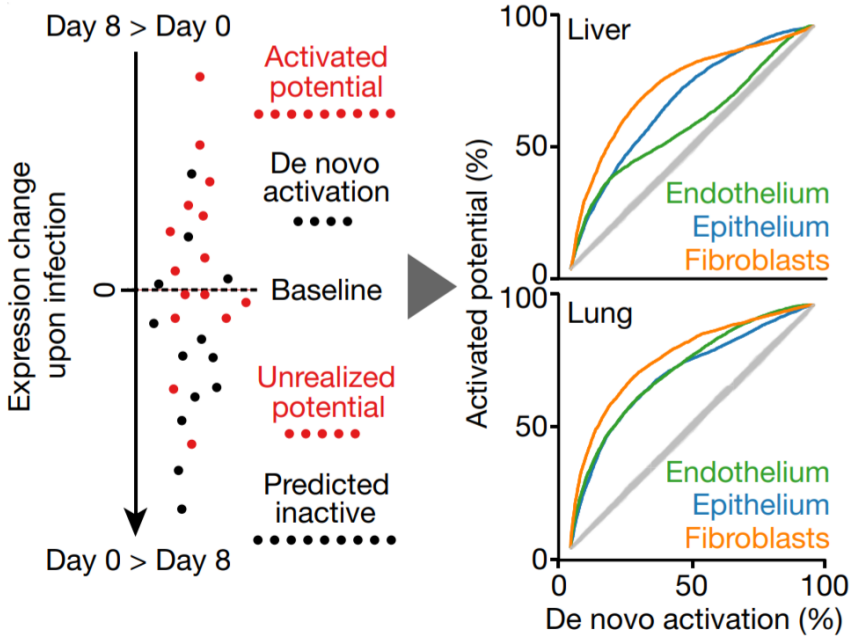


Systemic viral infection activates the epigenetic immune potential of structural cells

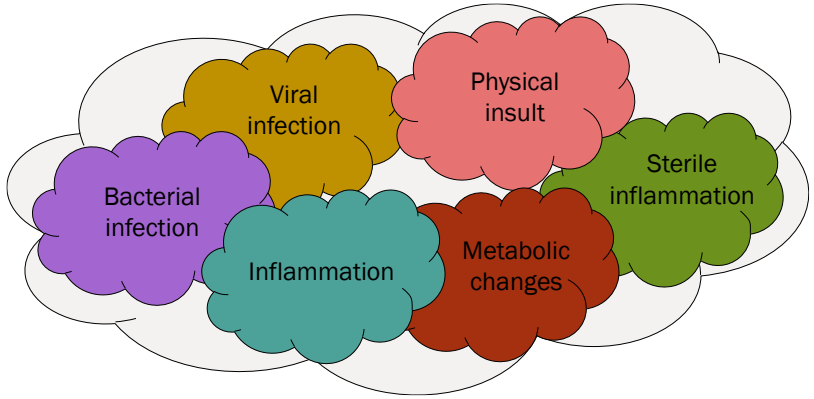
Validating the epigenetic potential of structural cells

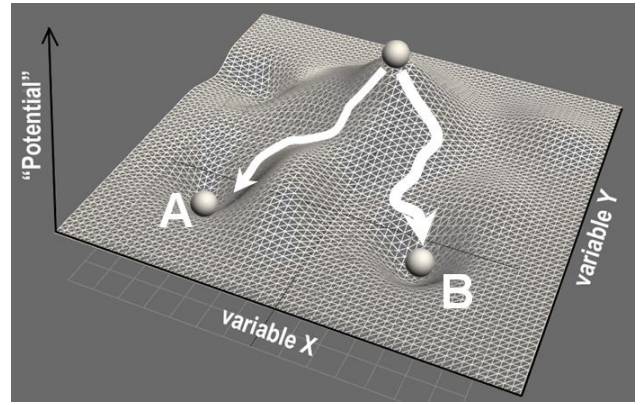
- We challenged mice with a systemic infection model (LCMV, collaboration with Andreas Bergthaler at CeMM)
- Preferential activation of “poised” immune genes





Model: Stimulus-dependent activation of epigenetic potential





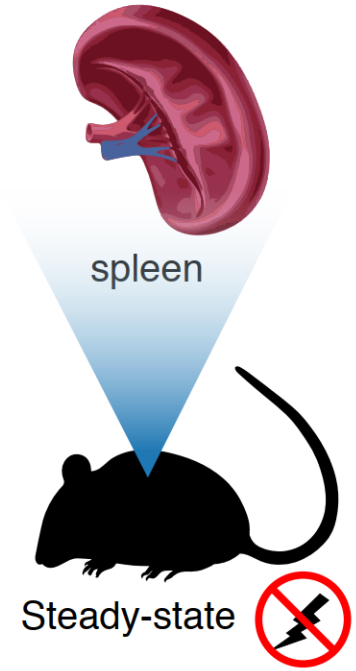
Krausgruber, Fortelny et al. 2020 Nature (https://nature.com/articles/s41586-020-2424-4)

How to immune cells retain their epigenetically primed state?

Hypothesis: Baseline immune signaling keeps target genes in open chromatin state

- We tested this hypothesis with a focus on JAK-STAT signaling in homeostasis
- We performed RNA-seq & ATAC-seq for T cells & macrophages from 12 mutant mouse models

Homeostatic model (*in vivo*)



JAK-STAT transgenic mice

Knockout

Stat1-ko	$Stat1^{-/-}$
Stat2-ko	$Stat2^{-/-}$
Stat3-ko	$Stat3^{-/-}$
Stat4-ko	$Stat4^{-/-}$
Stat5-ko	$Stat5^{-/-}$
Stat6-ko	$Stat6^{-/-}$
Irf9-ko	$Irf9^{-/-}$
Tyk2-ko	$Tyk2^{-/-}$

Isoform-specific

Stat1a-only	$Stat1^{\alpha/\alpha}$
Stat1b-only	$Stat1^{\beta/\beta}$

Hyperactive

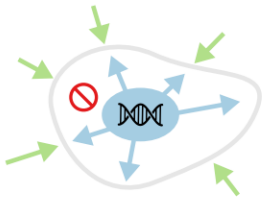
Stat5-hyp	$Stat5b^{N642H}$

Kinase-dead

Tyk2-inact	$Tyk2^{K923E}$

Cell types

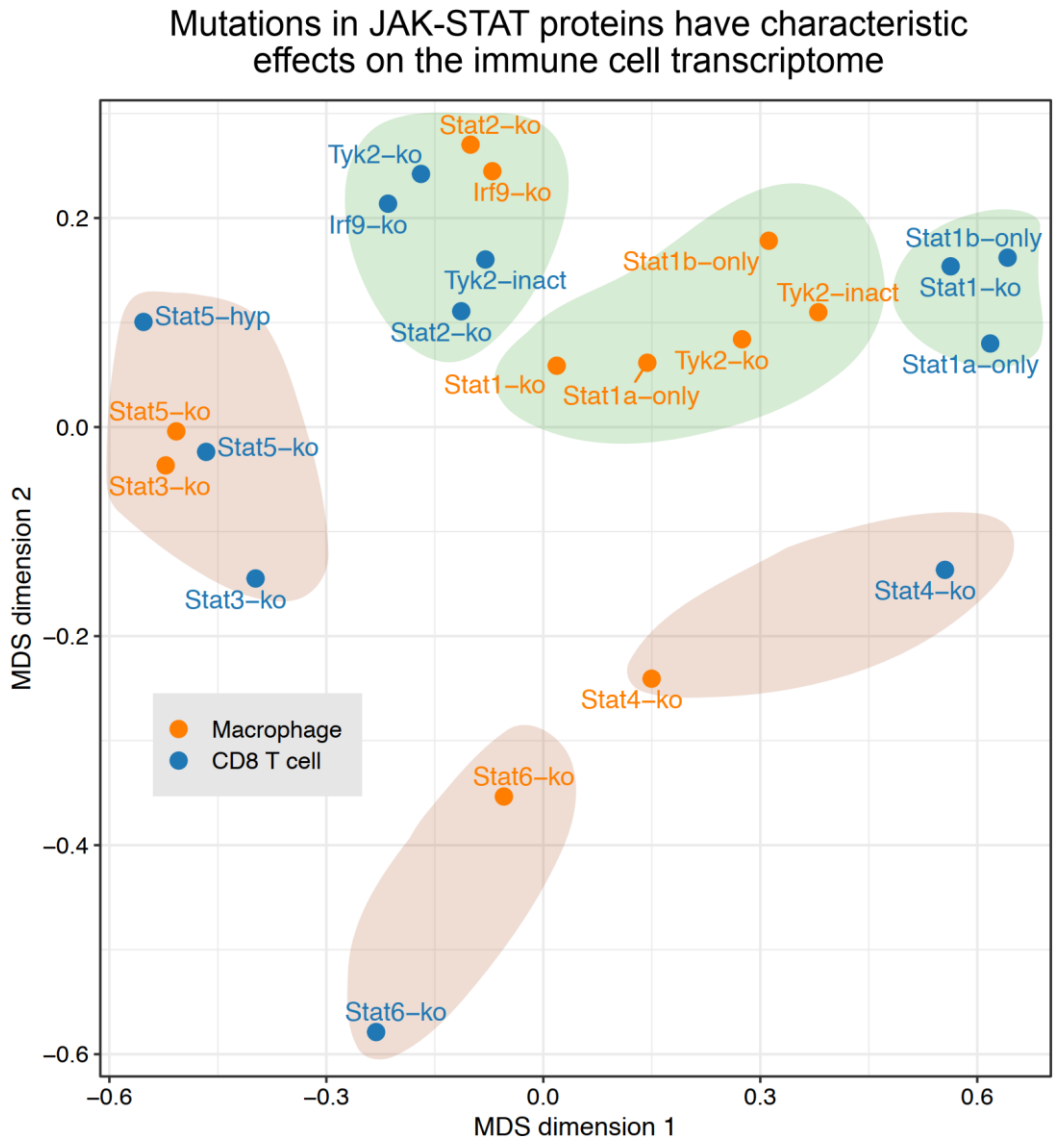
Myeloid lineage	Lymphoid lineage
Macrophage Dendritic cell	CD8+ T cell B cell NK cell

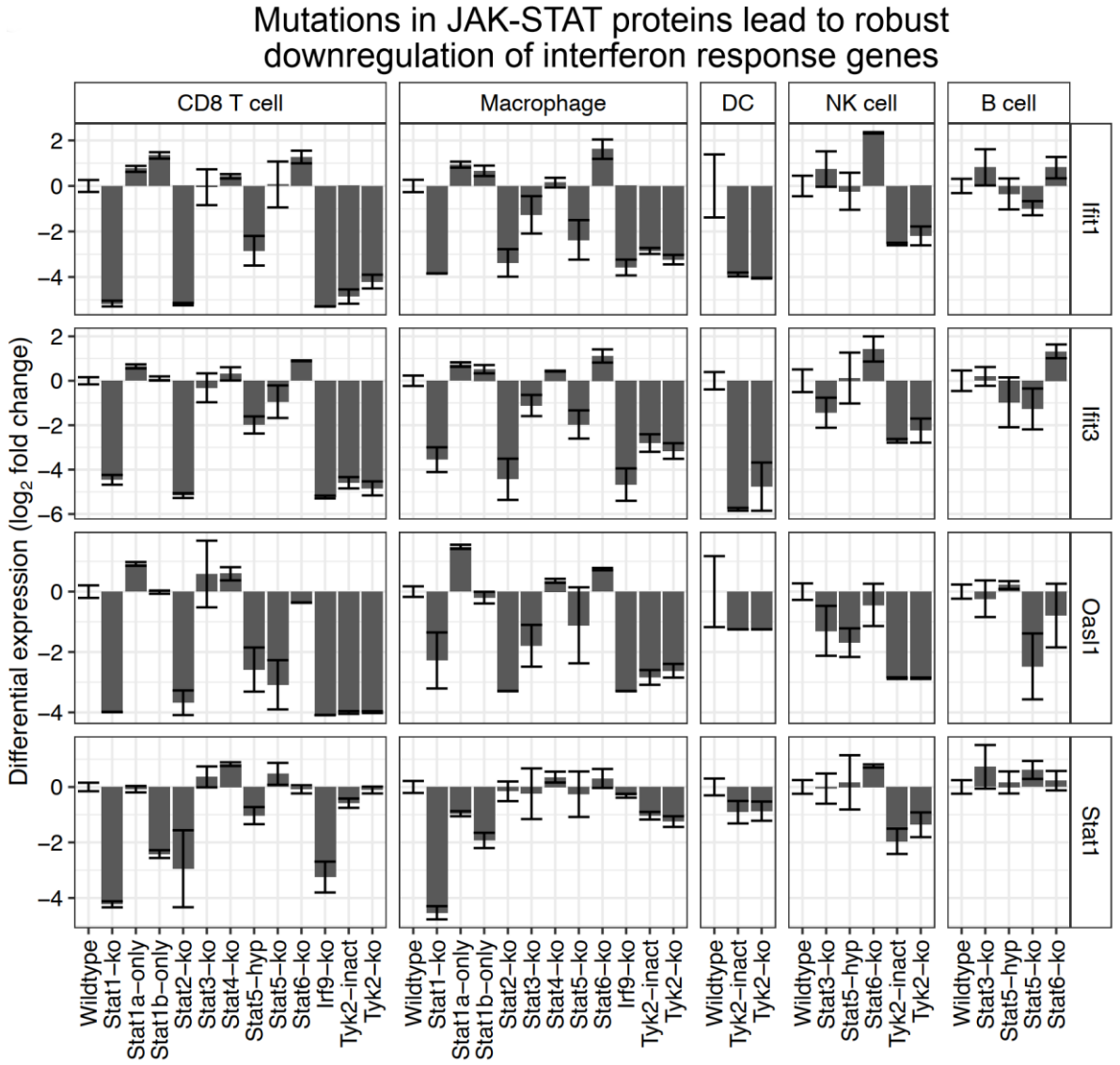


Perturbed homeostatic signaling

RNA-seq
ATAC-seq

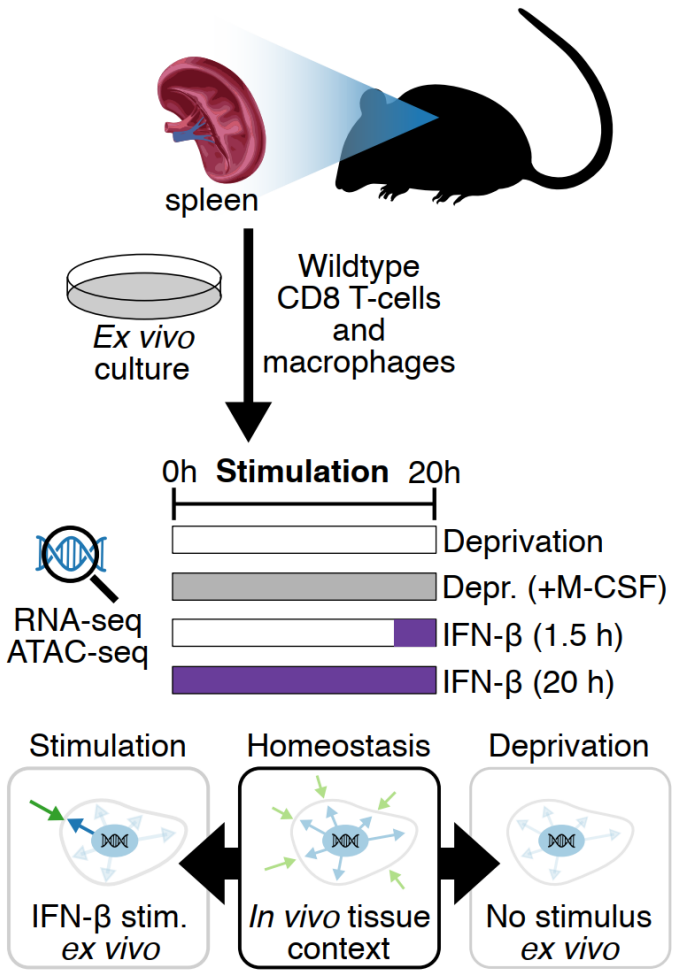
STAT knockout mice show reduced immune gene activity even at homeostasis

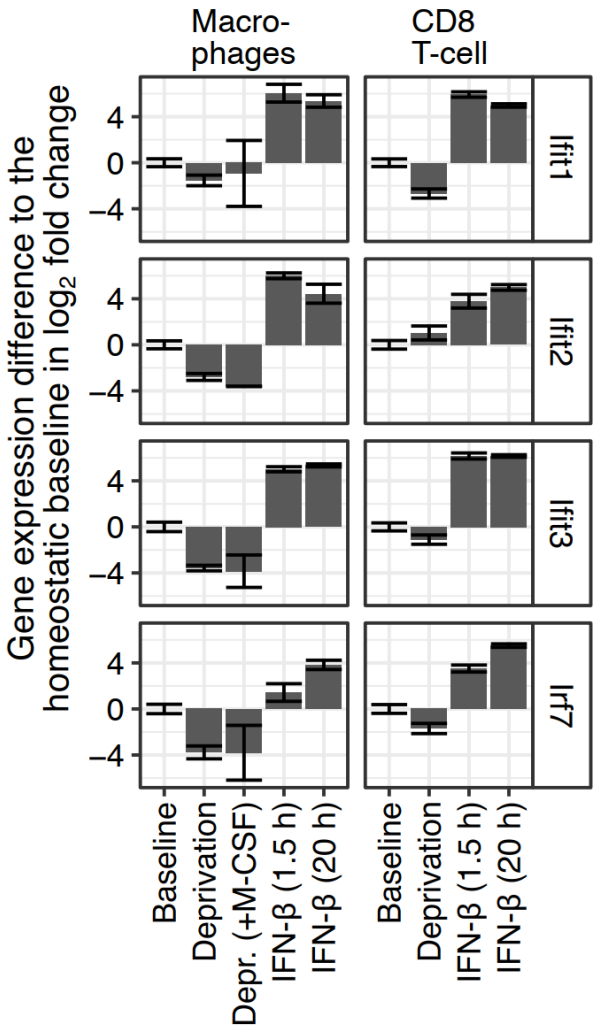




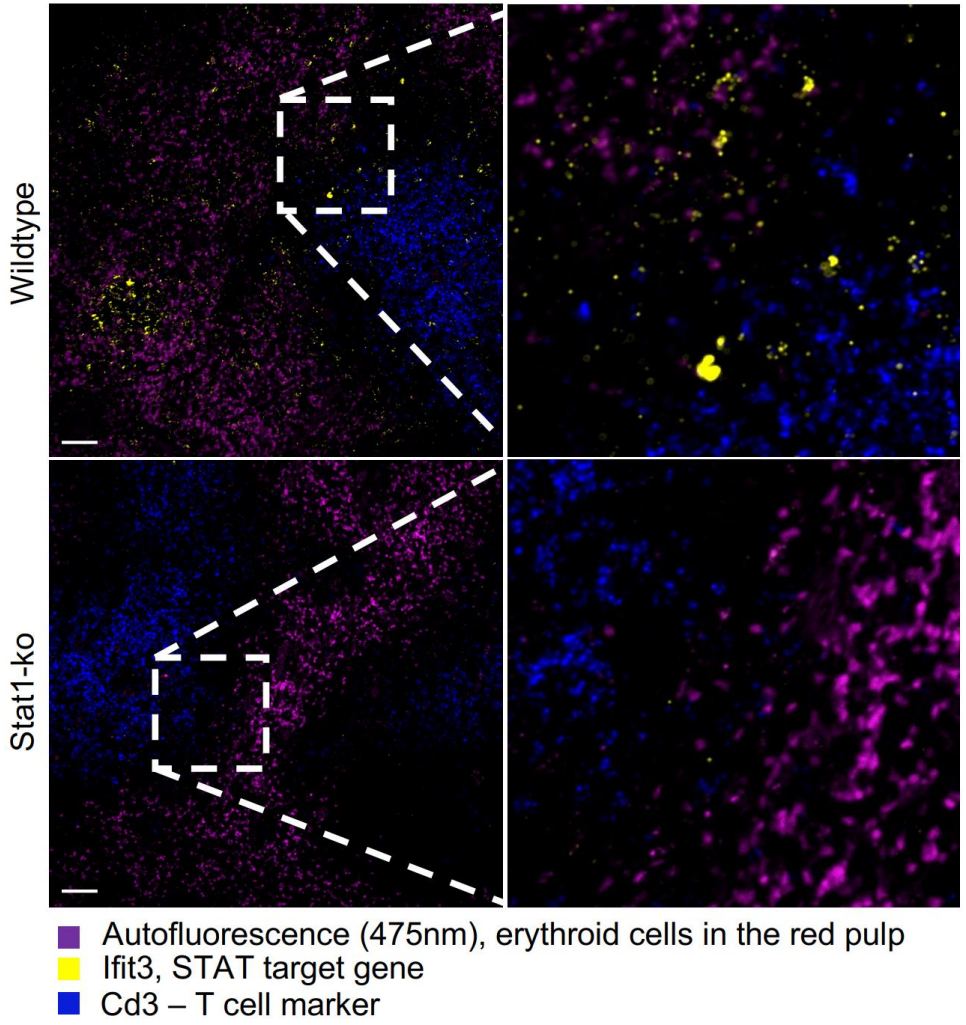
Baseline JAK-STAT signaling at homeostasis is driven by the tissue environment

Ex vivo culture deprives immune cells of baseline stimulation





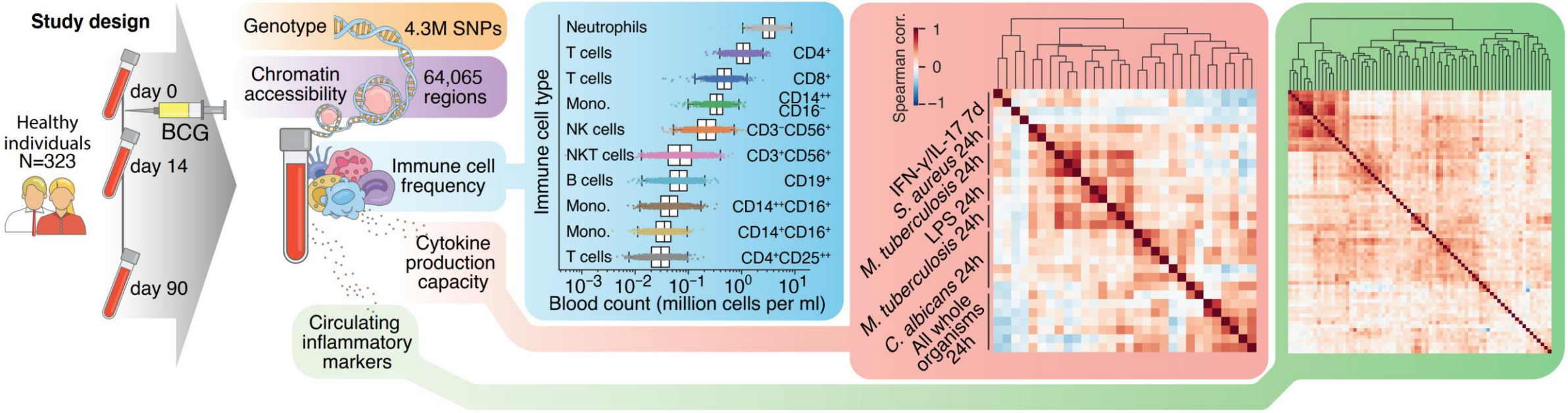
In vivo expression at sub-cellular resolution reveals *Ifit3* dependency on STAT1



How does epigenetic priming affect immune responses in humans?

BCG stimulates both innate & adaptive immunity, allowing us to compare these effects

- 323 individuals vaccinated with BCG, three time points, clinical study led by Mihai Netea (Nijmegen)
- We performed ATAC-seq on the entire cohort and analyzed the data with various immune readouts
- Time series character of the dataset enables investigation of baseline versus BCG effect



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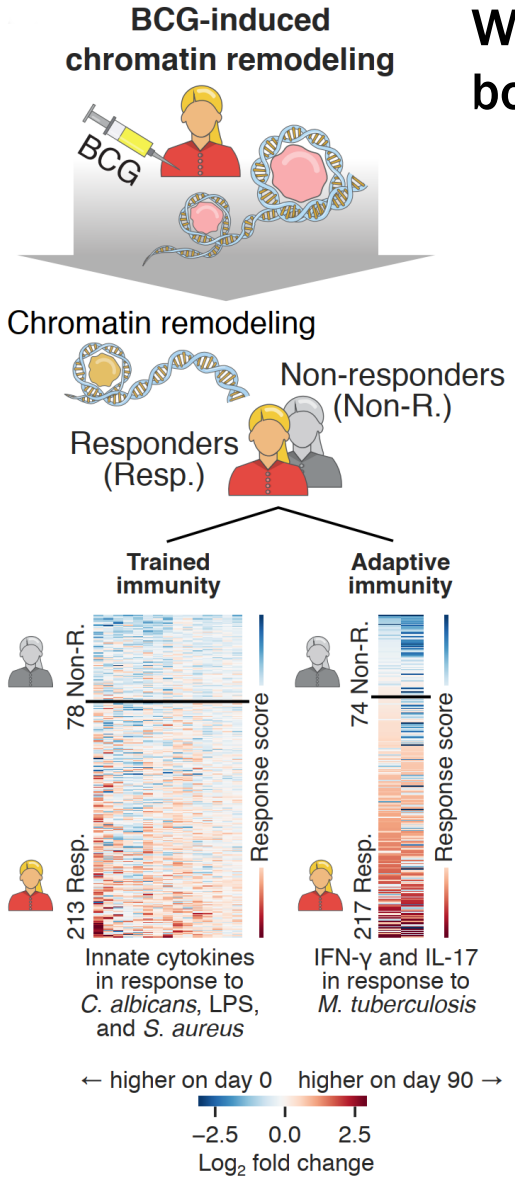
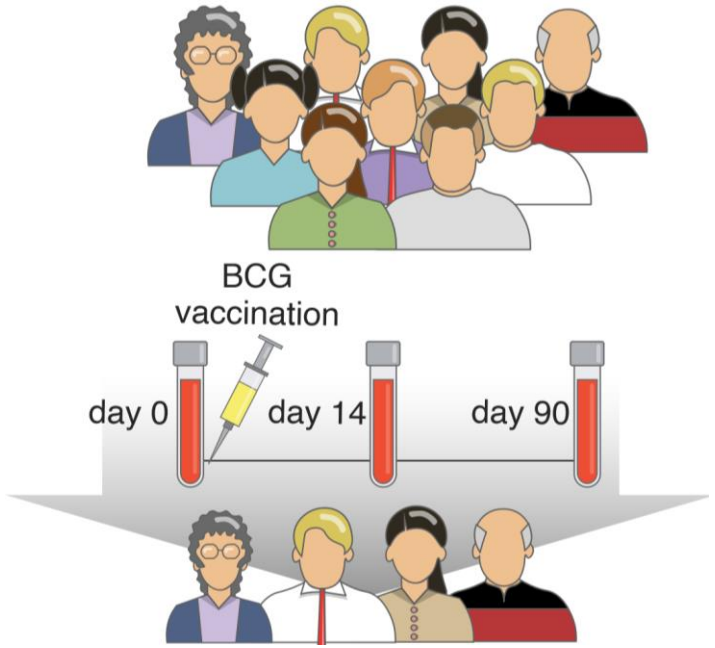
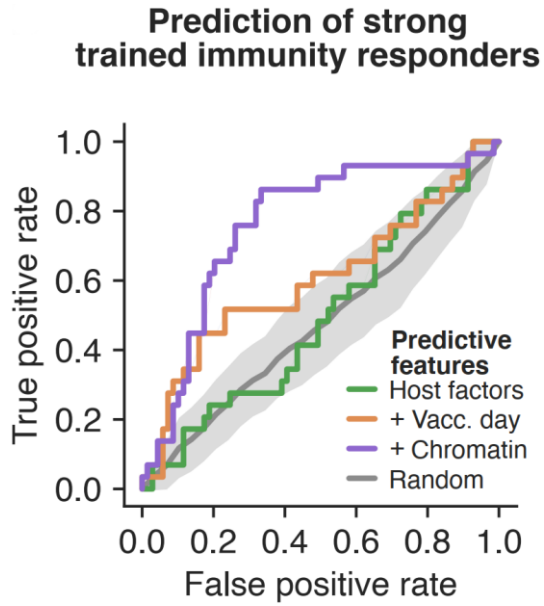
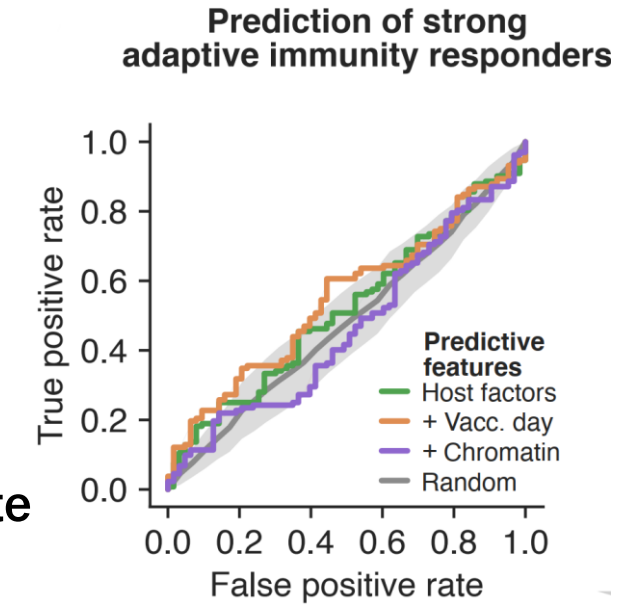


We observe responders vs. non-responders for both innate and adaptive immune responses





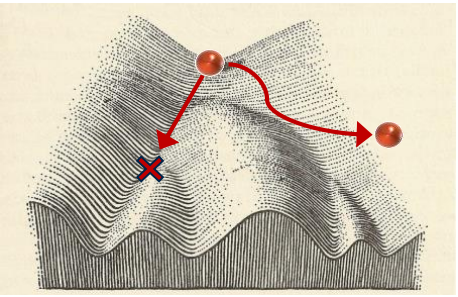


Chromatin accessibility predicts innate but not adaptive immune responders

Presentation outline

1. Developmental history and epigenetic cell states in immune diseases

Immune cells “remember” their differentiation history and re-use regulatory processes of normal development in immune diseases



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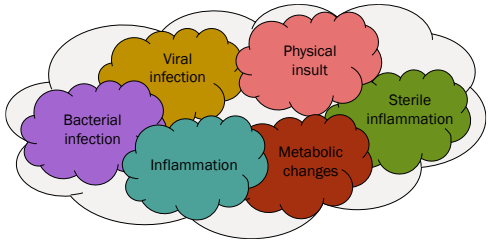

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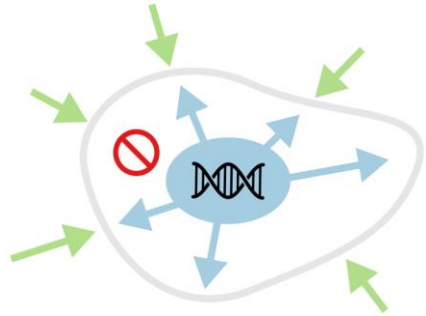
Epigenetic and transcription-regulatory profiles identify cancer cells-of-origin, detect disease progression, and prioritize potential therapies



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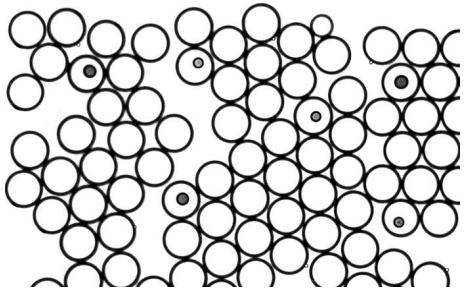
Interpretable deep learning, CRISPR single-cell sequencing, and patient-derived organoids facilitate mechanistic biology at scale

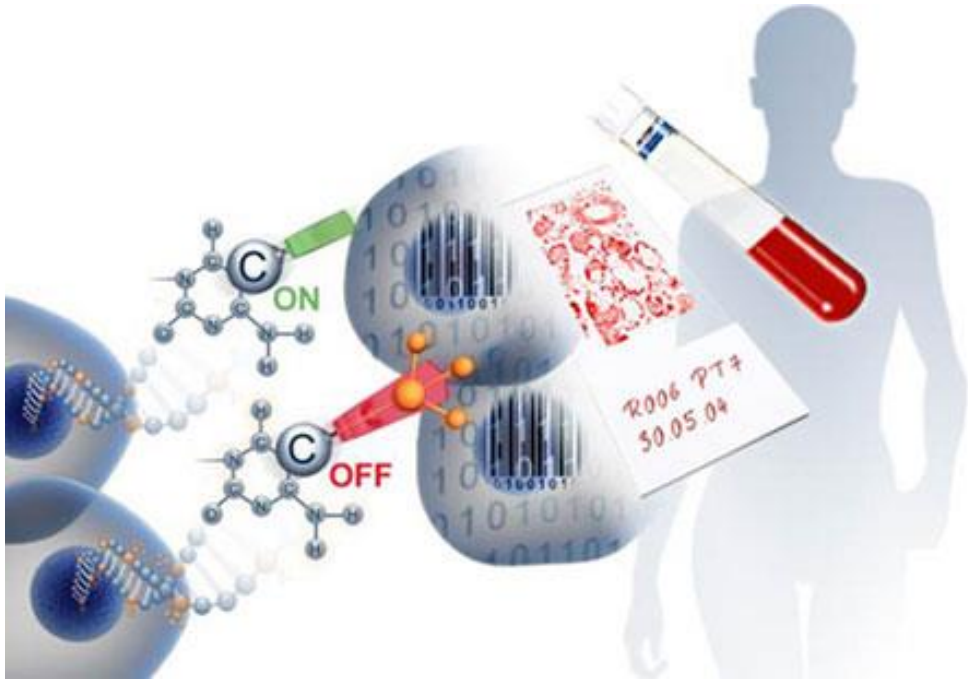


Using epigenetic information to guide personalized medicine

Genome profiling	Epigenome profiling		Transcriptome profiling
Stable	Cell type-specific		Fluctuating
DNA	DNA methylation	Chromatin	RNA

Clinical utility of epigenetic information

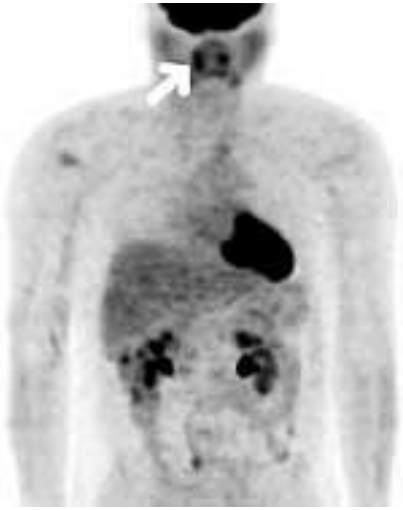
- Disease stratification for precision medicine
- Monitoring epigenetic drug response
- Detecting footprints of environmental exposure
- Inferring tissue type from DNA
- Quantifying immune cell infiltration

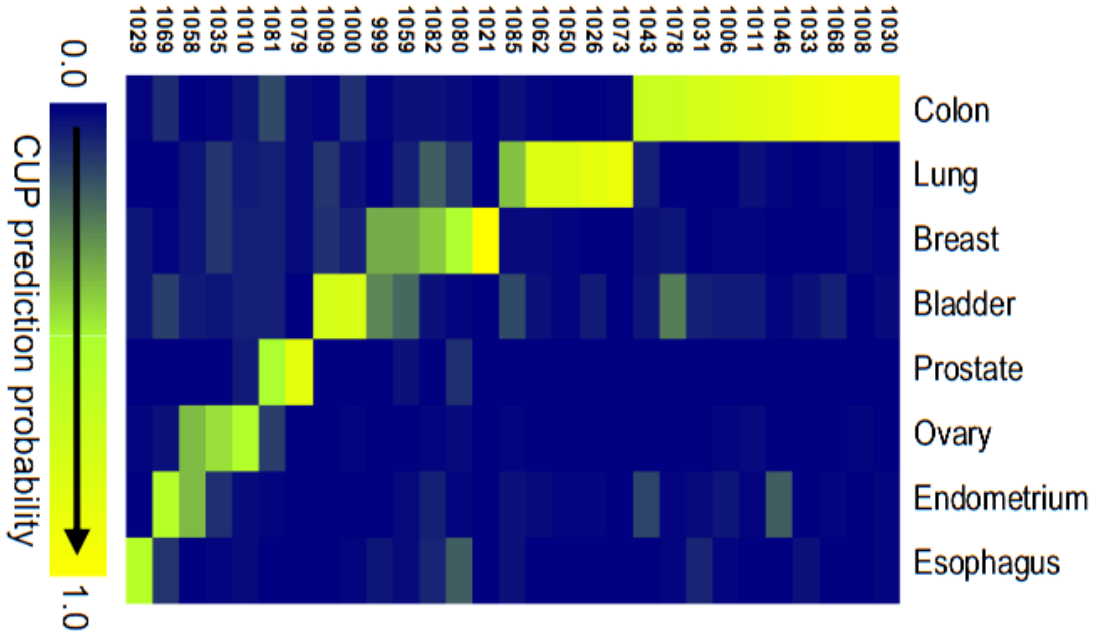


Example 1: Bioinformatics enables epigenetic cancer diagnostics

Cancers of unknown primary site (CUPs)

- **Metastatic cancers** of unknown primary site are hard to treat
- DNA methylation mapping established in a **reference set** of cell type signatures
- Bioinformatic analysis of **DNA methylation in CUPs** readily identified the tissue of origin





Bioinformatic approach

1. Training and cross validation of an elastic net classifier
2. Application to an independent test set of tumor samples
3. Validation based on clinical diagnostics data

Fernandez, Assenov et al. (2012) Genome Research (http://dx.doi.org/10.1101/gr.119867.110)

Example 1: The EPICUP biomarker for tissue-of-origin in cancer

Five years later – a validated biomarker

- **Public-private partnership** by Manel Esteller and Ferrer Biotech (Barcelona)
- **Retrospective validation study** done on 10,000 clinical samples
- **Biomarker CE-certified** with **97% sensitivity** and **>99% specificity**

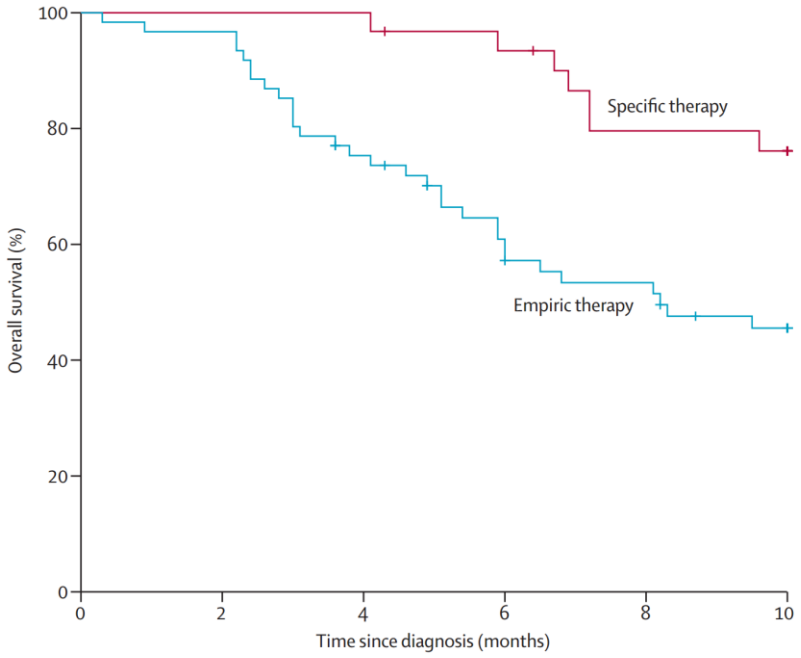
Validated Accuracy

Necropsy	100%
Further appearance of primary tumour	87%
Light microscopy evaluation	96%
IHC with tissue-specific markers	100%

Epigenetic profiling to classify cancer of unknown primary: a multicentre, retrospective analysis

Sebastian Moran, Anna Martínez-Cardús, Sergi Sayols, Eva Musulén, Carme Balañá, Anna Estival-Gonzalez, Cátia Moutinho, Holger Heyn, Angel Diaz-Lagares, Manuel Castro de Moura, Giulia M Stella, Paolo M Comoglio, Maria Ruiz-Miró, Xavier Matias-Guiu, Roberto Pazo-Cid, Antonio Antón, Rafael Lopez-Lopez, Gemma Soler, Federico Longo, Isabel Guerra, Sara Fernandez, Yassen Assenov, Christoph Plass, Rafael Morales, Joan Carles, David Bowtell, Linda Mileshkin, Daniela Sia, Richard Tothill, Josep Tabernero, Josep M Llovet, Manel Esteller

Promising Clinical Utility



Moran et al. (2016) Lancet Oncology (http://dx.doi.org/10.1016/S1470-2045(16)30297-2)

Example 2: Dissecting epigenetic heterogeneity in Ewing sarcoma

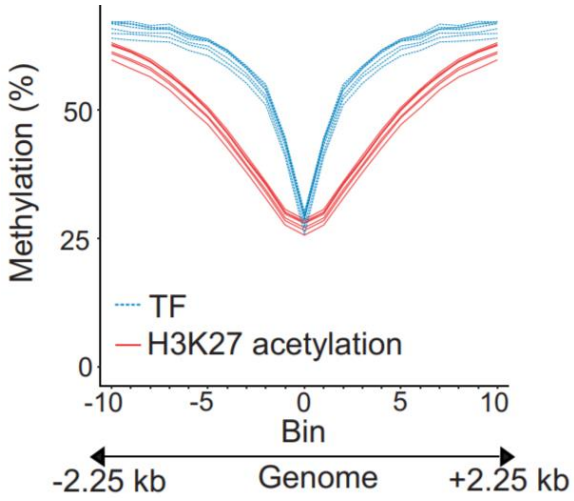
Collaboration:

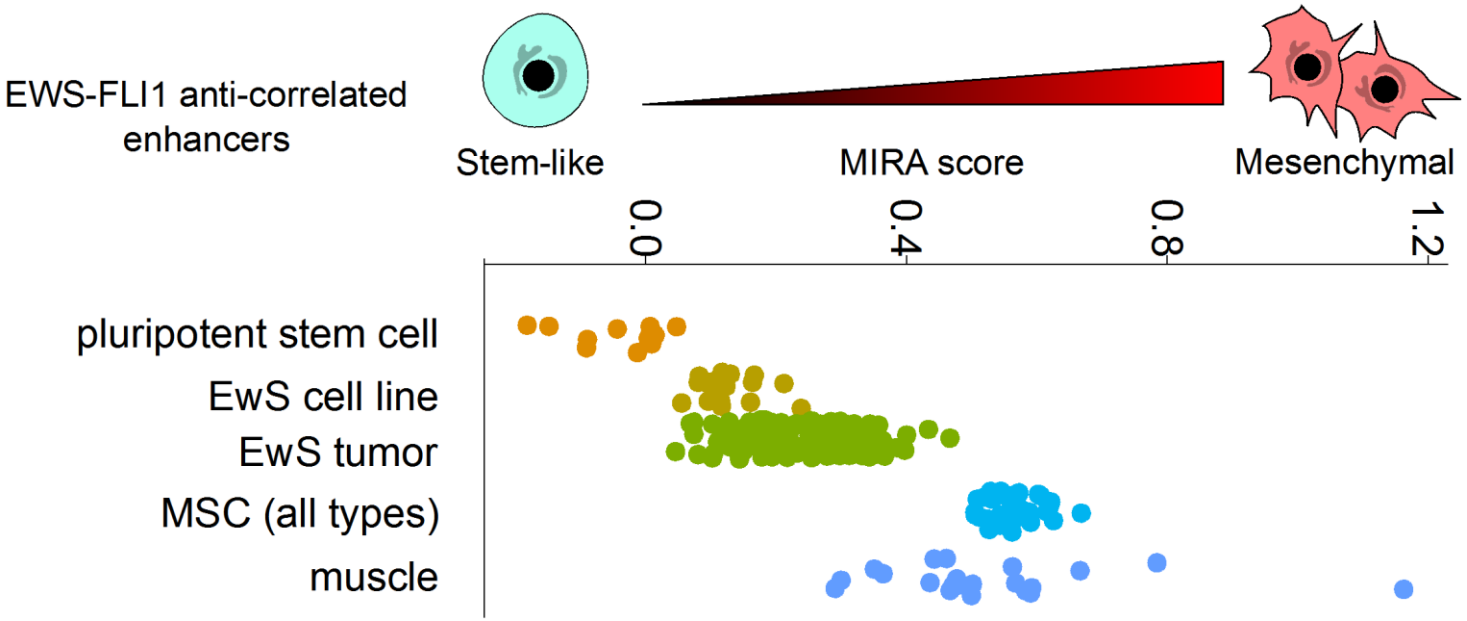
Ewing sarcoma: Aggressive childhood cancer with unexplained heterogeneity

- Driven by a single genetic event (EWS-ETS gene fusion), few other genetic defects
- **Hypothesis:** Epigenetic heterogeneity may explain the observed clinical variability

A spectrum of epigenetic states reflecting cell-of-origin

MIRA (Methylation-based Inference of Regulatory Activity) method



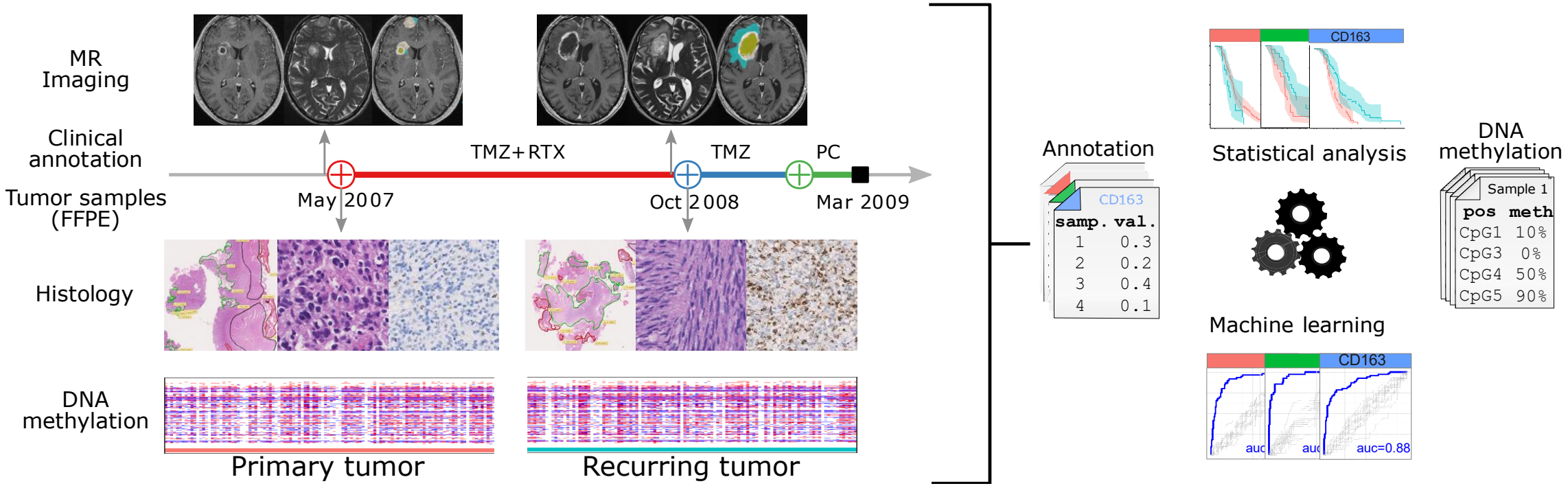


Sheffield et al. (2017) Nature Medicine (http://dx.doi.org/10.1038/nm.4273)

Example 3: Glioblastoma progression based on a national registry

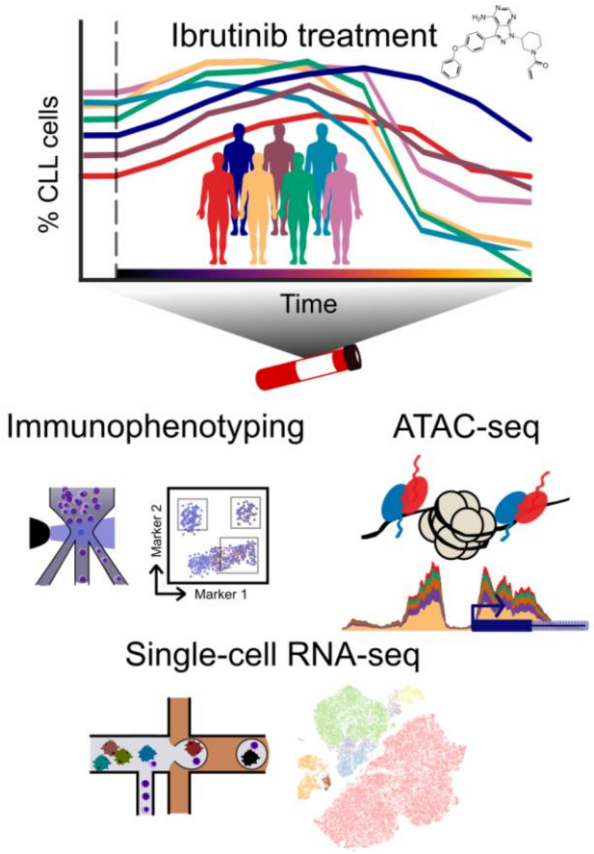
Approach:

- Cohort: 112 primary, IDH wildtype glioblastoma patients, each with 2-4 time points
- Selected from the Austrian Brain Tumor Registry (FFPE tumor blocks)
- DNA methylation profiling using FFPE-optimized RRBS protocol in 499 tumor samples
- Multimodal data integration by statistical methods and machine learning

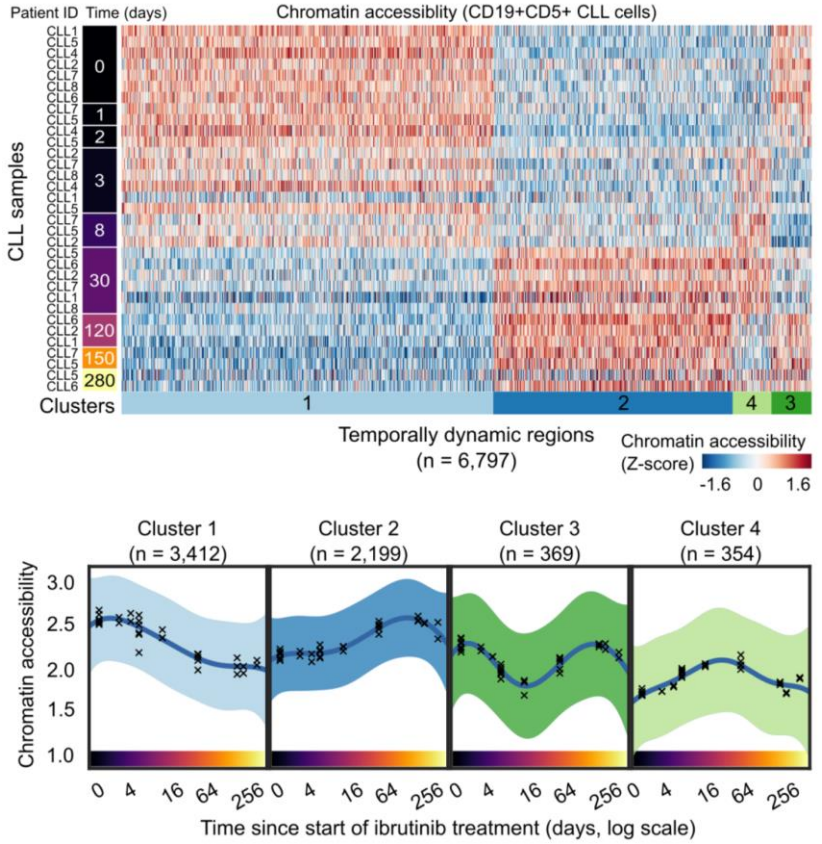


Klughammer et al. (2018) Nature Medicine (http://dx.doi.org/10.1038/s41591-018-0156-x)

Example 4: Time series analysis of the response to targeted leukemia therapy



Comp. Modeling



Dataset

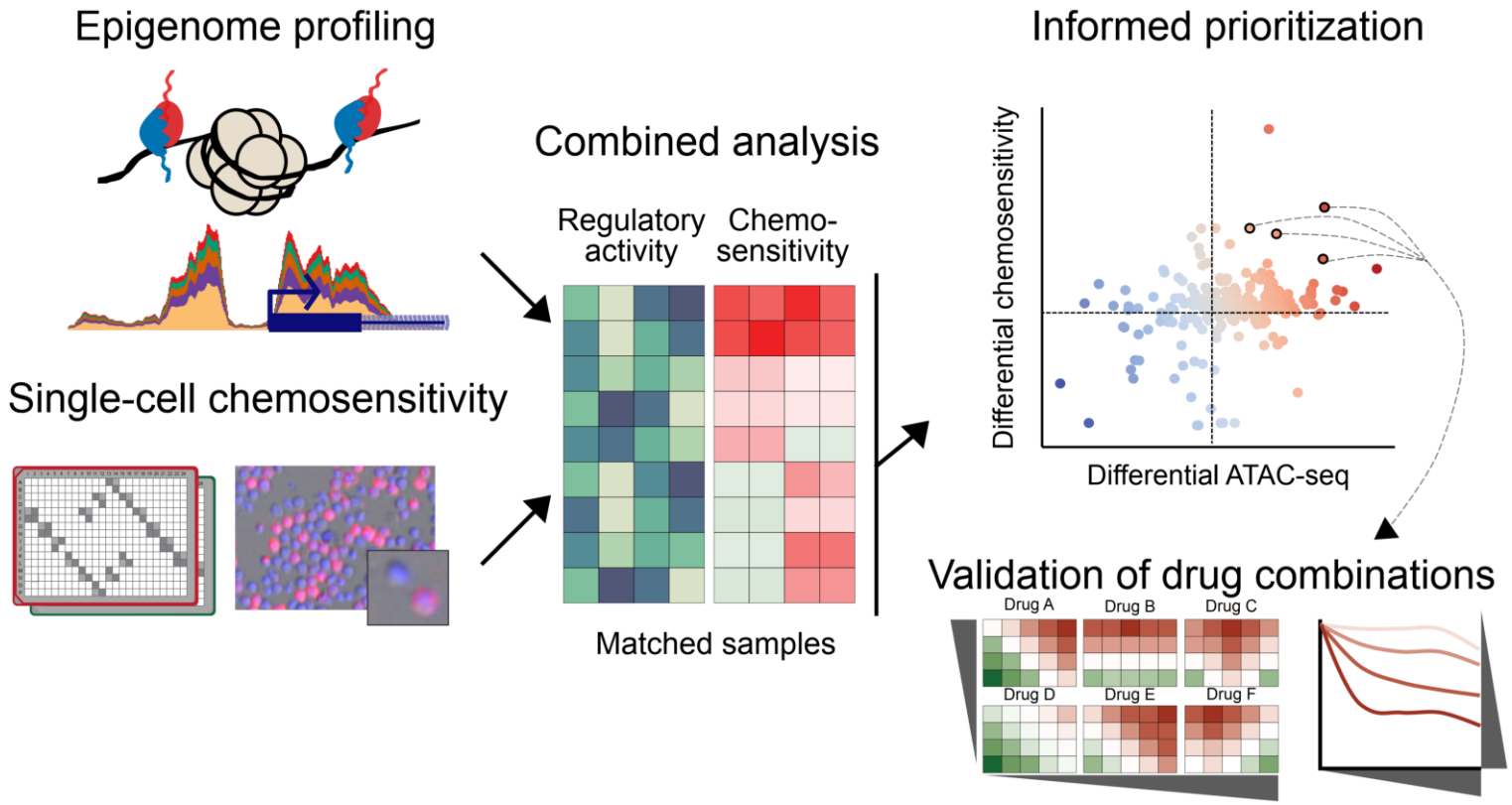
- ATAC-seq: 7 patients, 8 time points, 6 cell types
- >43,000 single-cell transcriptomes

Model / Interpretation

- Conserved response, heterogeneous speed
- NF-κB binding down ➔ lineage TFs down ➔ erosion of CLL cell identity ➔ quiescence

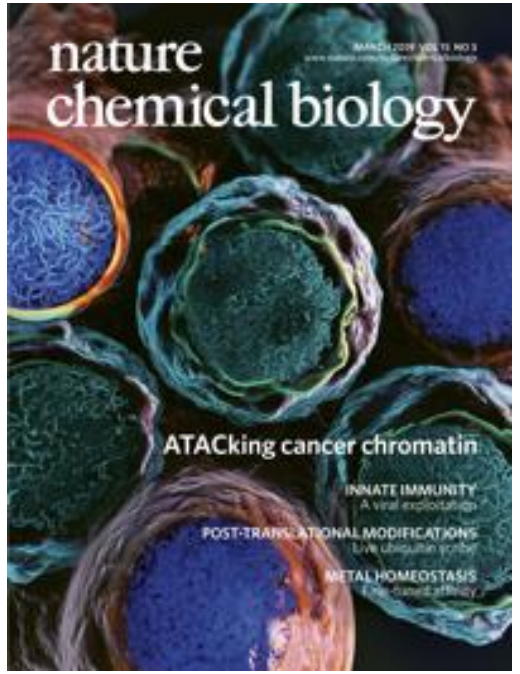
Example 4: Drug-response profiling & epigenetics prioritize drug combinations

Identifying drugs to enhance ibrutinib's anti-CLL effect



Collaboration:

MEDICAL UNIVERSITY OF VIENNA

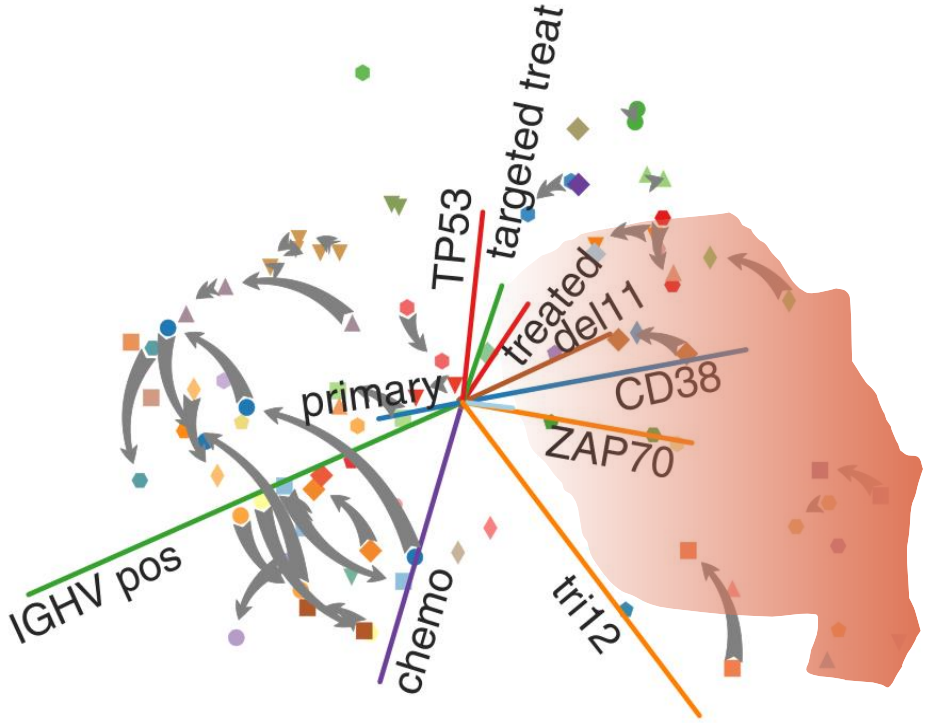


Integration of epigenome profiling and single-cell chemosensitivity profiling prioritizes drug sensitivities for combination therapy

Schmidl, Vladimer, Rendeiro et al. (2019) Nature Chemical Biology (http://dx.doi.org/10.1038/s41589-018-0205-2)

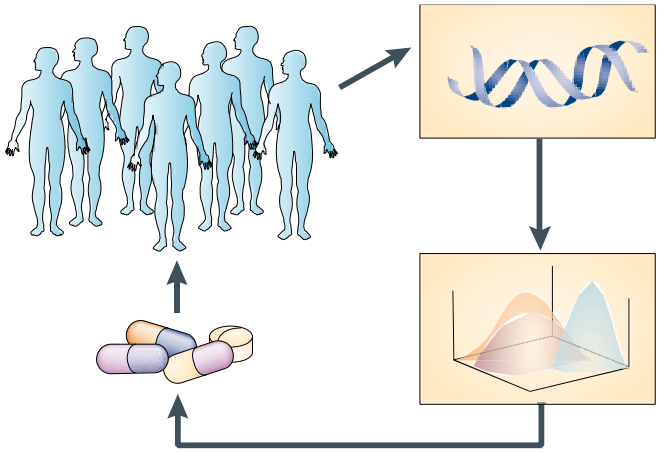
Vision: Patient-specific ‘route planning’ on disease landscapes

Toward adaptive therapy in leukemia (CLL)



- Goal: **Delay disease progression** until the patient dies for unrelated reasons
- Concept: **Manipulate the evolutionary dynamics** on the cells’ fitness landscape

HIV therapy as proof-of-principle



PERSPECTIVES

OPINION

Managing drug resistance in cancer: lessons from HIV therapy

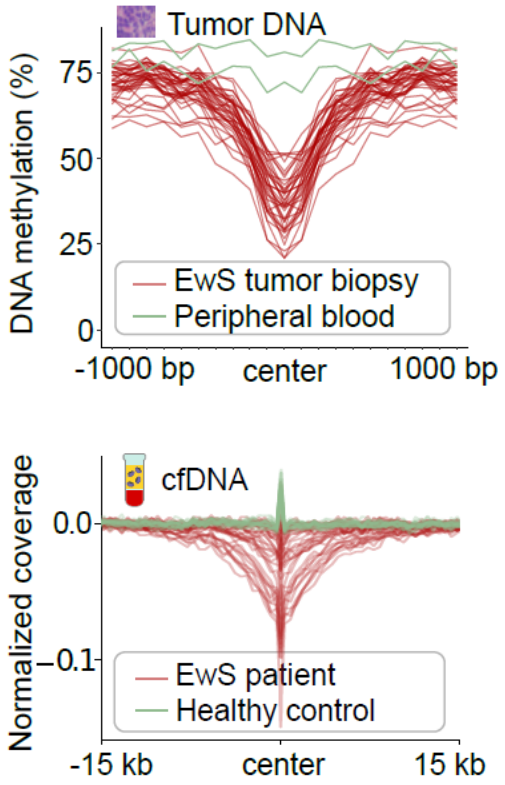
Christoph Bock and Thomas Lengauer

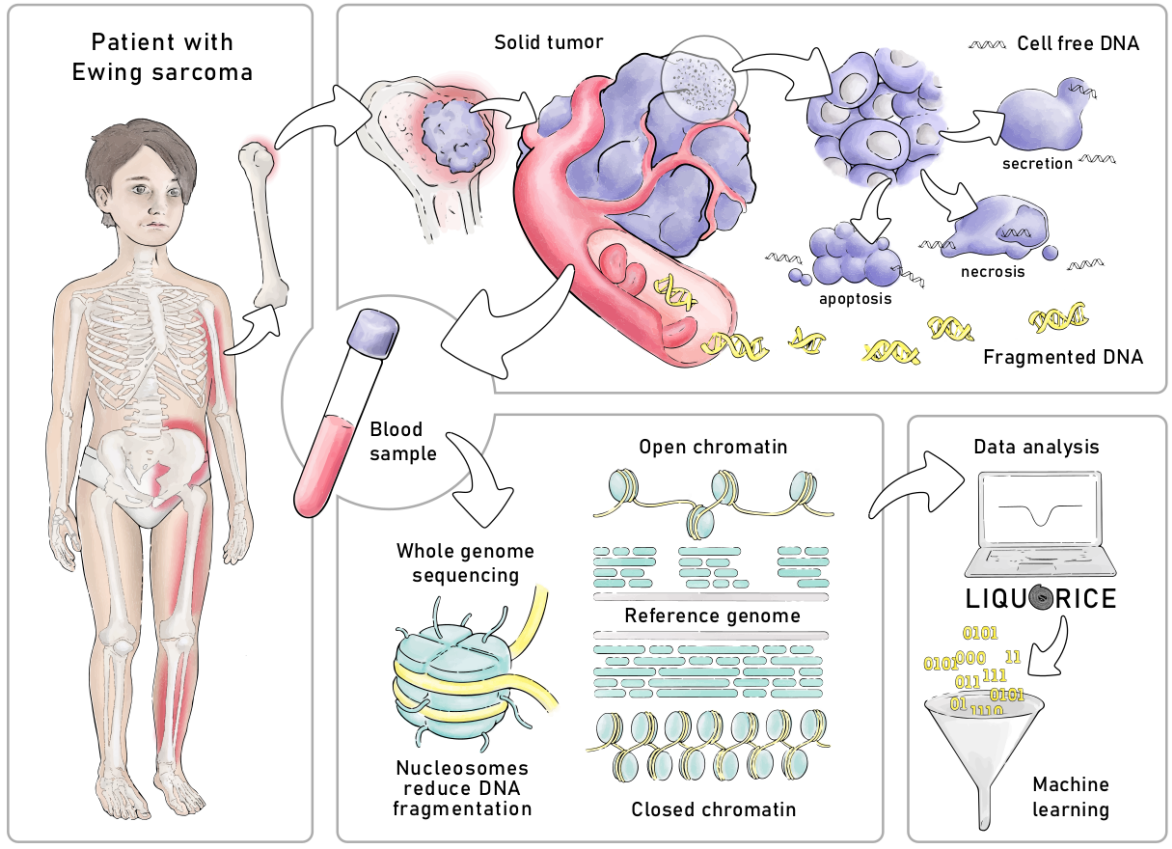
Abstract | Drug resistance is a common cause of treatment failure for HIV infection and cancer. The high mutation rate of HIV leads to genetic heterogeneity among viral populations and provides the seed from which drug-resistant clones emerge in response to therapy. Similarly, most cancers are characterized by extensive genetic, epigenetic, transcriptional and cellular diversity, and drug-resistant cancer cells outgrow their non-resistant peers in a process of somatic evolution. Patient-specific combination of antiviral drugs has emerged as a powerful approach for treating drug-resistant HIV infection, using genotype-based predictions to identify the best matched combination therapy among several hundred possible combinations of HIV drugs. In this Opinion article, we argue that HIV therapy provides a ‘blueprint’ for designing and validating patient-specific combination therapies in cancer.

Detecting epigenetic footprints of Ewing sarcoma in cell-free DNA

A whole genome sequencing based liquid biopsy assay that does not depend on genetic alterations

- Many childhood cancers have few genetic alterations, making liquid biology analysis challenging
- Fragmentation patterns of tumor-derived DNA in the blood reflect tumor-specific epigenetic signatures





Collaboration:

St. Anna Kinderkrebs Forschung

TOMAZOU LAB

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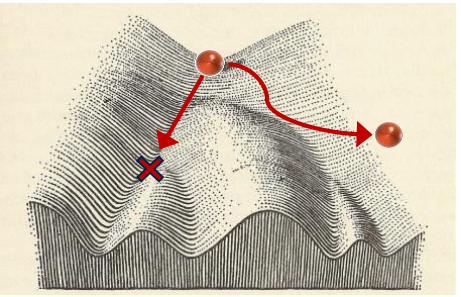

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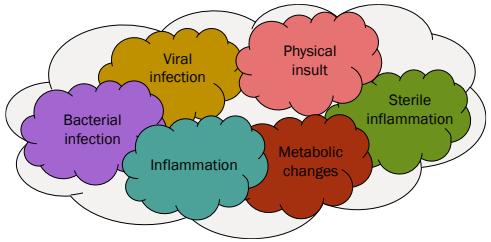

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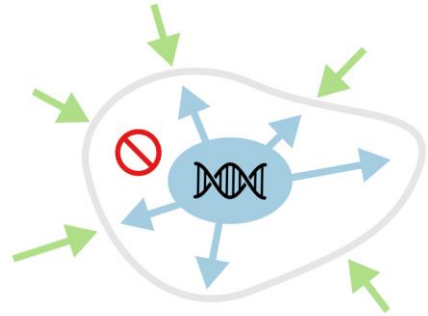

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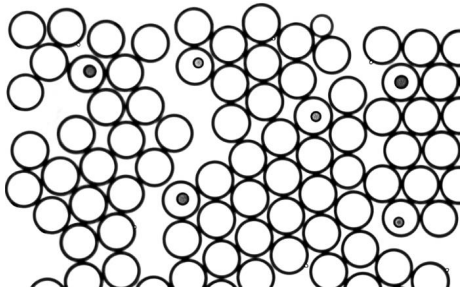
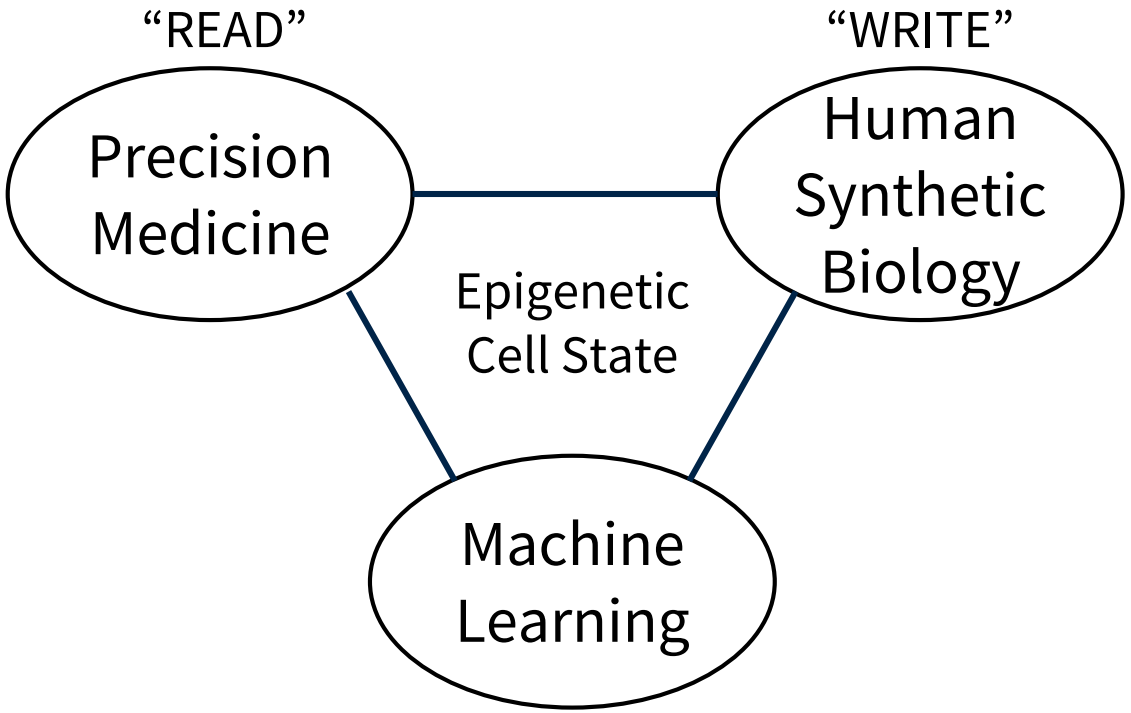
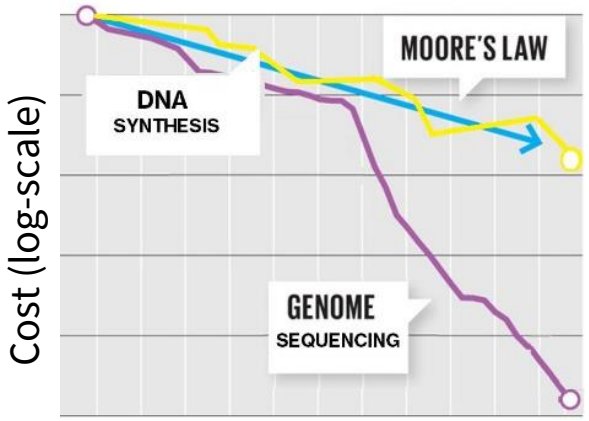


Our goal: Programming cells for biological discovery and therapeutic applications

Lead technology: Next Generation Sequencing

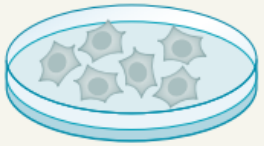
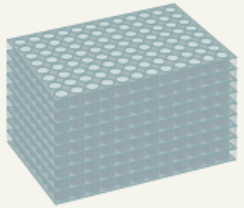
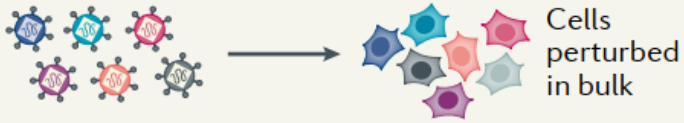
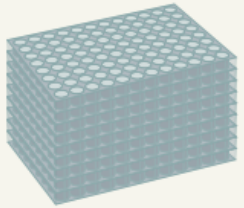
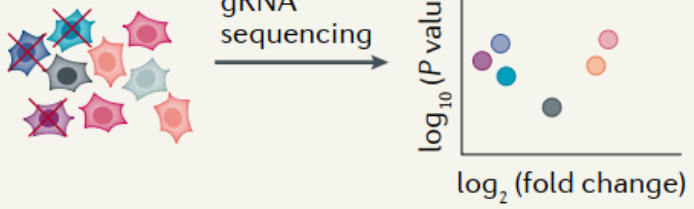
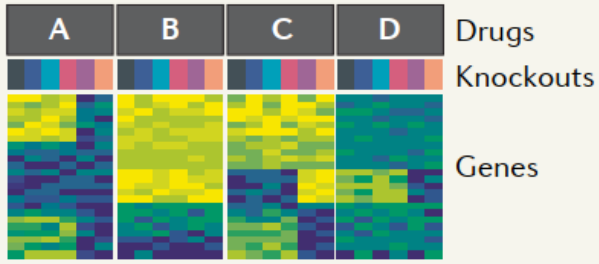




Lead technology: Massive-scale DNA synthesis

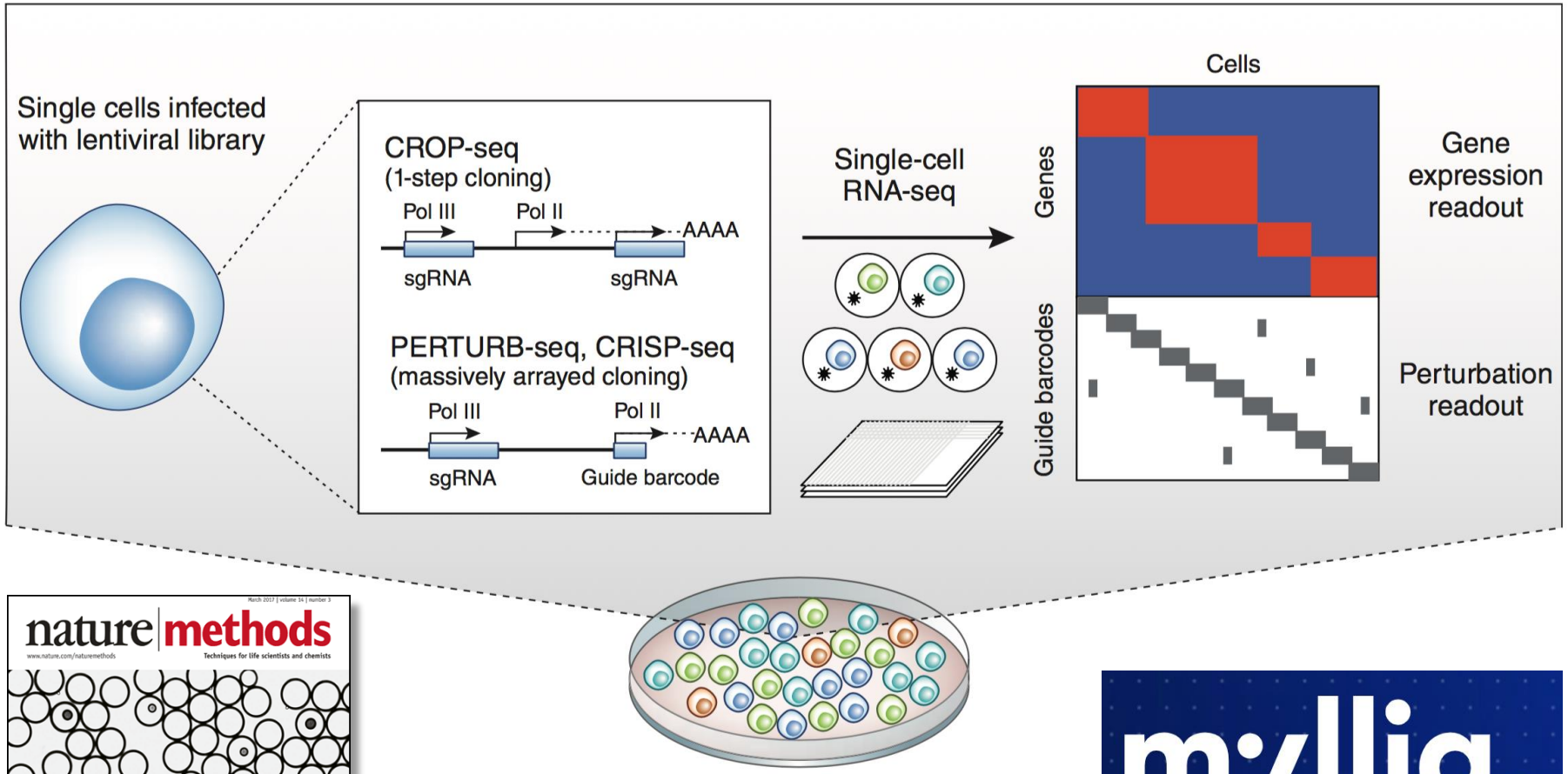
Lead technology: Interpretable machine learning

High-content CRISPR screening

	Pooled CRISPR screen	Arrayed CRISPR screen
① **Model** Cell lines, primary cells, organoids, model organisms (in vivo screens)	Cas9-expressing cells in bulk 	Cas9-expressing cells in separate wells → 
② **Perturbation** CRISPR knockout, interference, activation, base editing, prime editing	CRISPR gRNA library  Cells perturbed in bulk	Cells perturbed in separate wells 
③ **Challenge** Cell survival and proliferation, drug treatment and resistance, virus/pathogen infection, metabolic challenges	 Cells challenged in bulk (e.g. cytotoxic drug)	Cells challenged in separate wells (e.g. cytotoxic drugs) A B C D → 
④ **Readout** Sequencing-based counting of gRNA frequencies, single-cell RNA-seq, multi-omics profiling, imaging	 gRNA sequencing → gRNA enrichment/ depletion $\log_{10}$ (*P* value) $\log_2$ (fold change)	Molecular phenotyping  A B C D Drugs Knockouts Genes

Bock et al. (2022) *High-content CRISPR Screening*. Nature Reviews Disease Primers (http://dx.doi.org/10.1038/s43586-021-00093-4)

CROP-seq enables CRISPR screening with very complex phenotypes



myllia.

"CROP-seq as a service" for biotech/pharma (CoI)

Formerly: Aelian Biotechnology

Related methods

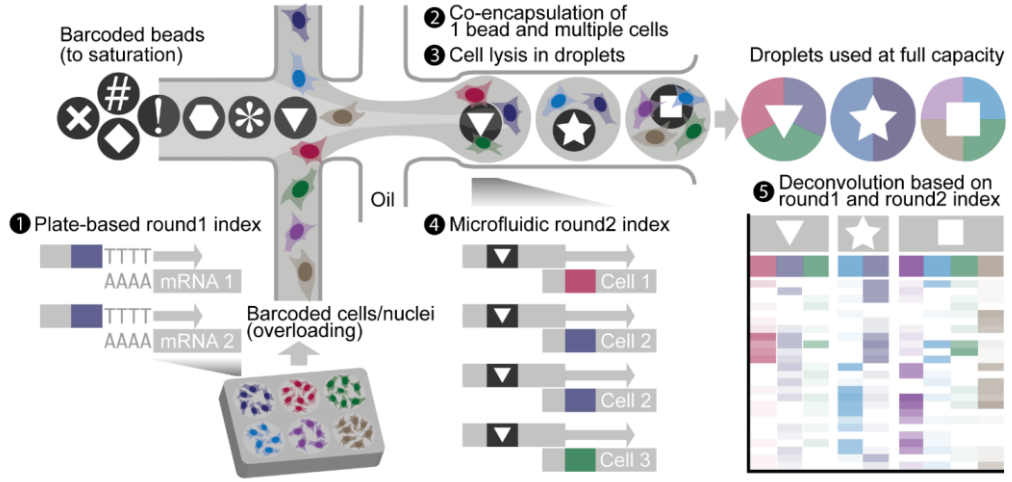
Perturb-seq – A. Regev & J. Weissman labs (Dixit et al, 2016; Adamson et al, 2016)
CRISP-seq – I. Amit lab (Jaitin et al, 2016)
Mosaic-seq – G. Hon lab (Xie et al, 2017)

‘Hacking’ droplet technology for million-scale single-cell RNA-seq

scifi-RNA-seq integrates combinatorial indexing with fluidic indexing

- Droplet-based single-cell RNA-seq (e.g. 10x Genomics) is highly inefficient due to stochastic droplet loading (→ most droplets remain empty)
- We use single cells/nuclei as our reaction compartment and pre-index all RNA molecules on 384-well plates (as in combinatorial indexing)
- Massive overloading of the 10x Genomics machine puts 5-10 cells into each droplet, yielding >1 million cells per chip (instead of ~50k)

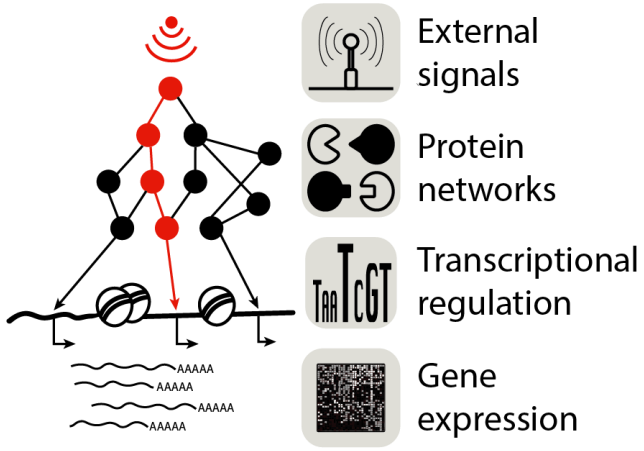
scifi-RNA-seq assay design



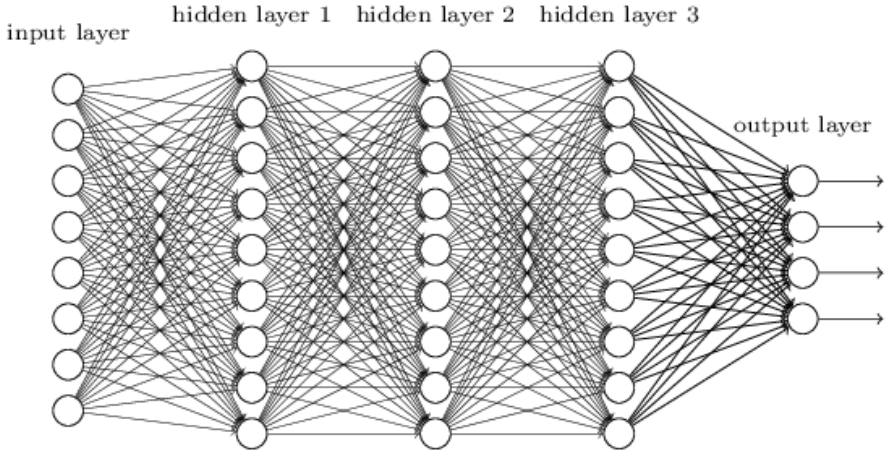
151,788 cells in one 10x Genomics channel

Interpretable deep learning for causal inference in biological networks

Biological networks are very different from deep learning networks

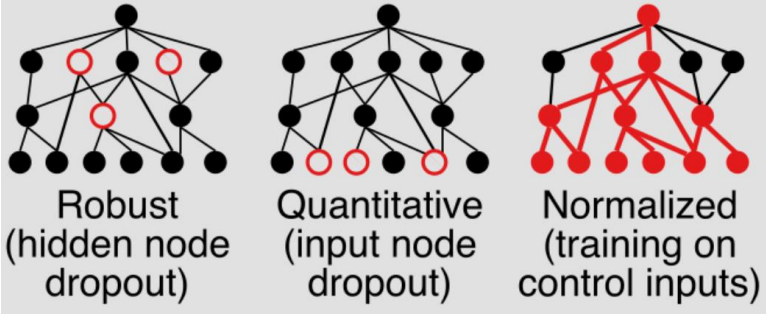


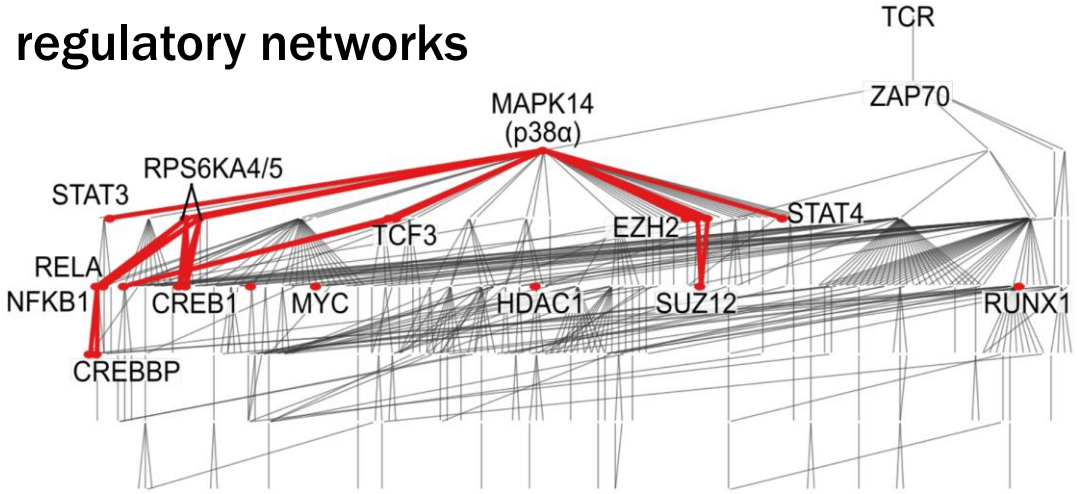
Gene-regulatory network



Fully connected artificial neural network

We developed interpretable deep learning on regulatory networks





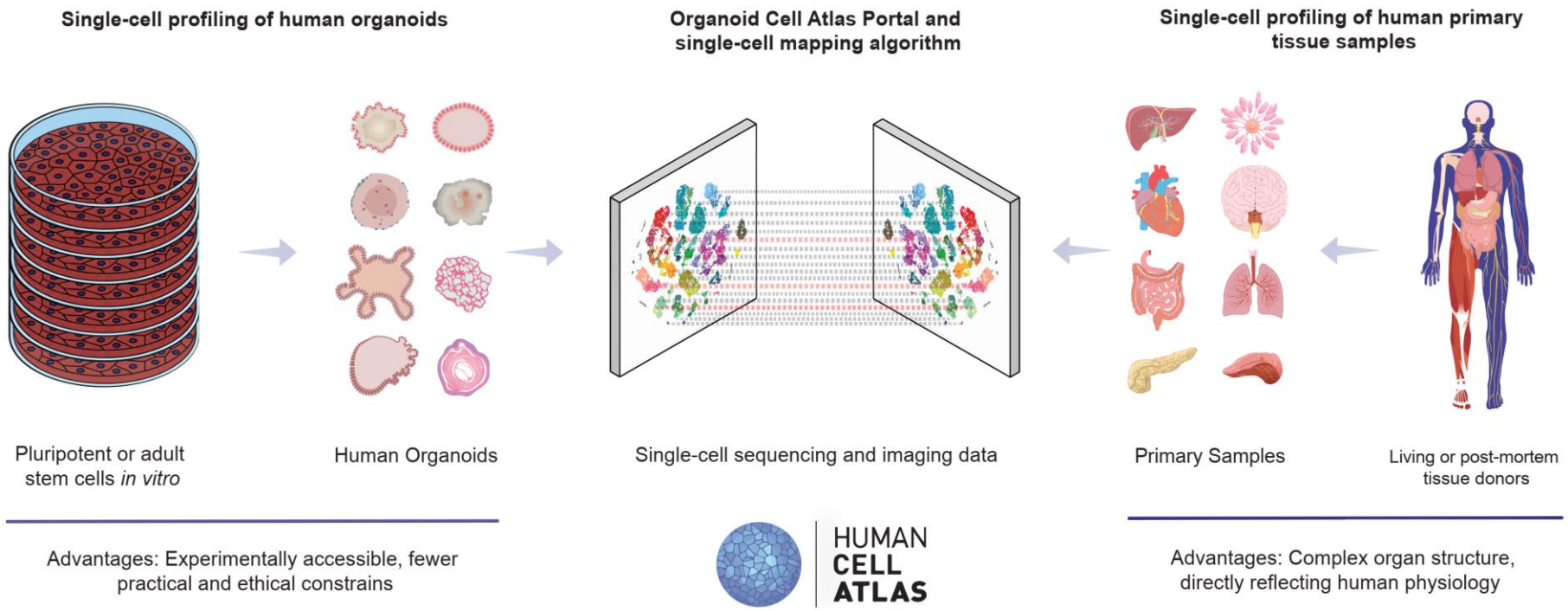
Nikolaus Fortelny & Christoph Bock (2020) Genome Biology (https://doi.org/10.1186/s13059-020-02100-5)

Organoids provide an ideal platform for mechanistic biology at scale

Advantages of organoids:

- Faithfully recapitulate human biology (much better than immortalized cell lines)
- High-throughput perturbation experiments with molecular & phenotypic readout

Pilot project to establish an Organoid Cell Atlas



HCA|Organoid

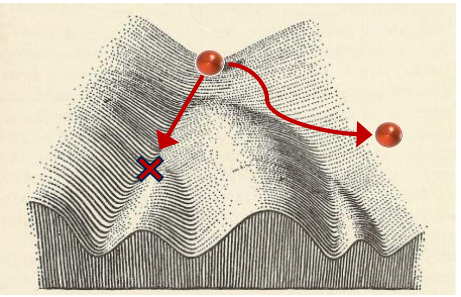
Website: https://hca-organoid.eu/

Bock et al. (2021) Nature Biotechnology (https://nature.com/articles/s41587-020-00762-x)

Presentation outline

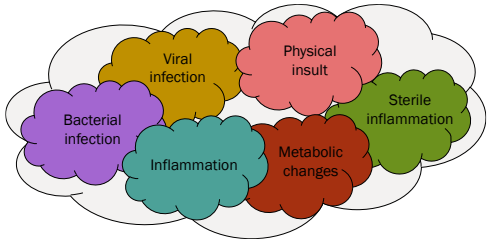
1. Developmental history and epigenetic cell states in immune diseases

Immune cells “remember” their differentiation history and re-use regulatory processes of normal development in immune diseases



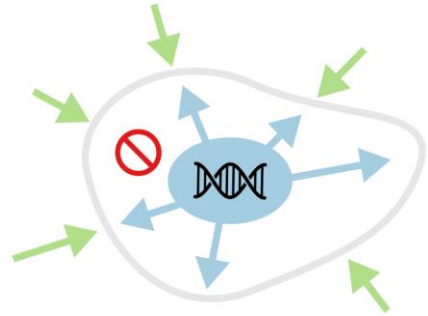
2. Epigenetic potential for rapid immune gene activation

Hematopoietic and non-hematopoietic structural cells implement an epigenetic potential for rapid immune gene activation upon challenge



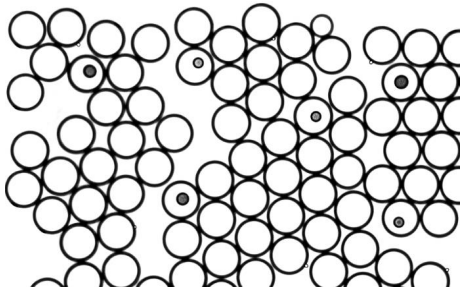
3. Epigenetic cell states connecting the past and future of cancer cells

Epigenetic and transcription-regulatory profiles identify cancer cells-of-origin, detect disease progression, and prioritize potential therapies



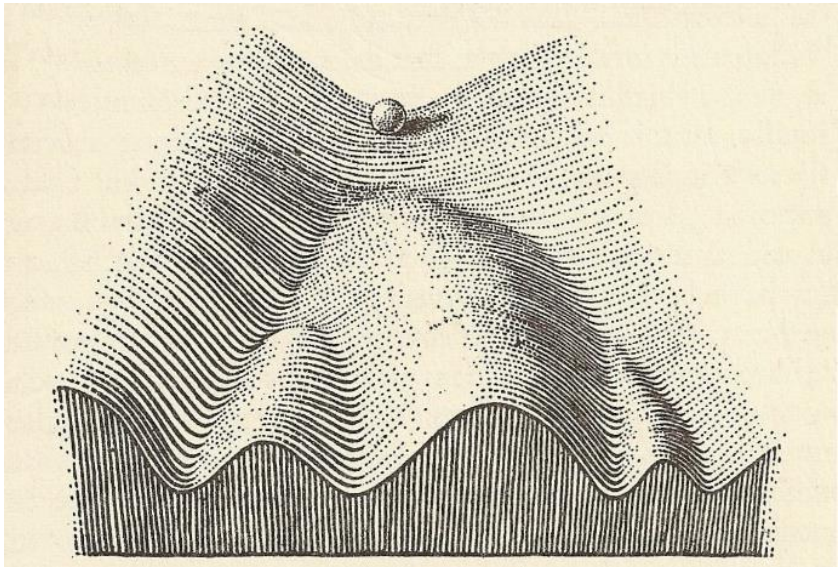
4. Rational programming of human cells for biomedical applications

Interpretable deep learning, CRISPR single-cell sequencing, and patient-derived organoids facilitate mechanistic biology at scale

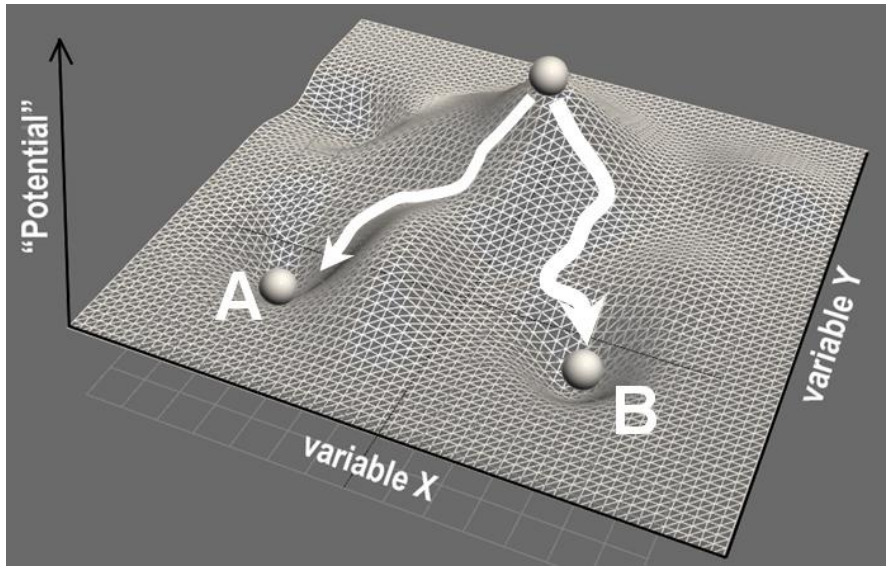


Summary & outlook: Epigenetics connects the cells' past and future

Cells retain an epigenetic record of their developmental history

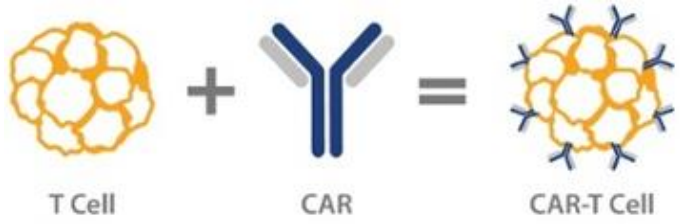


Epigenetic cell states capture a cell's future potential to respond to stimuli



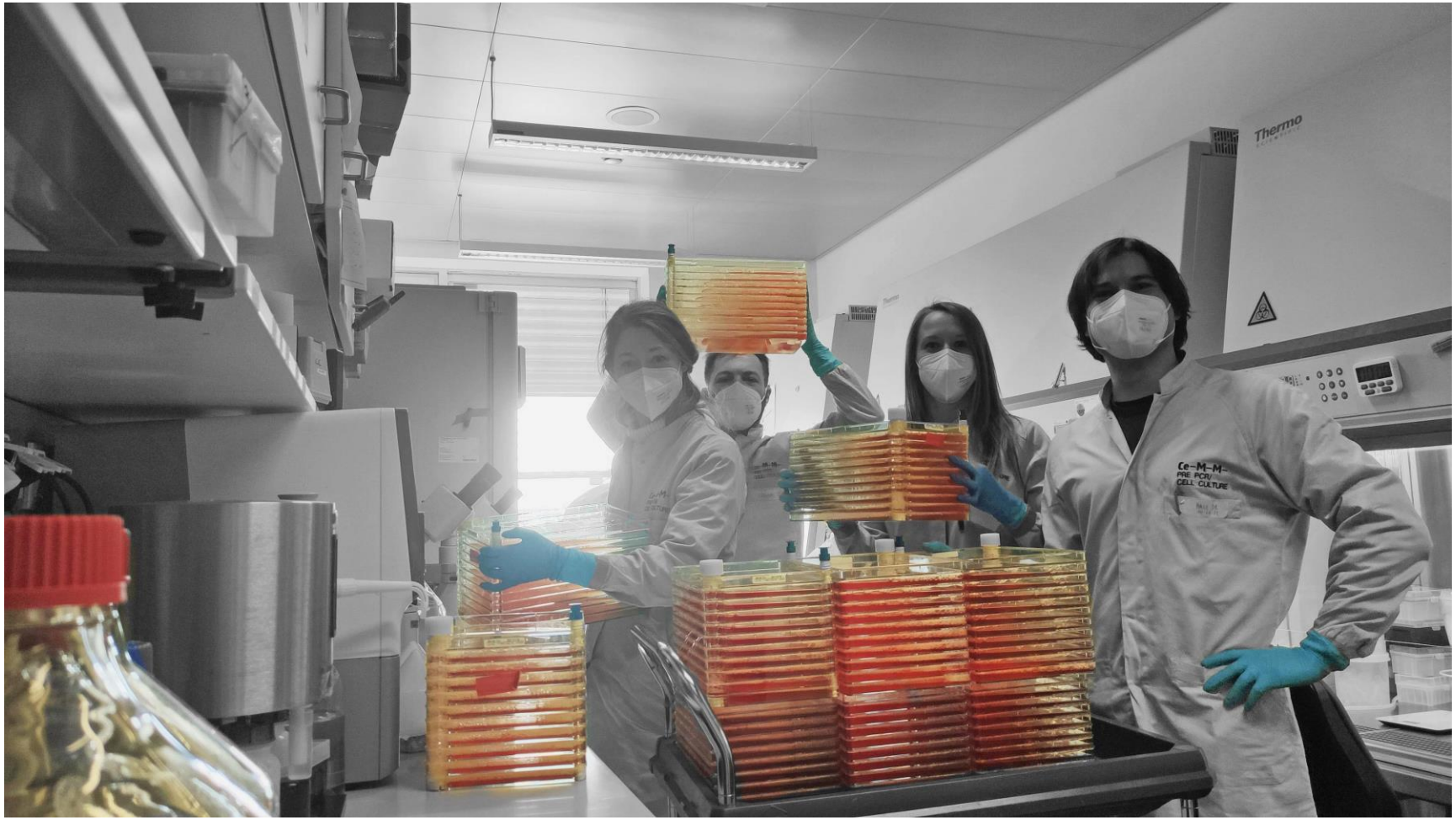
Can we engineer epigenetic cell states for better cell-based therapies?

- This is a topic of a major research project in our lab (ERC Consolidator Grant 2021-2026)
- We try to epigenetically "supercharge" CAR T cells to work effectively in solid tumors (and autoimmune diseases?)

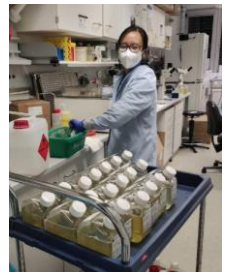


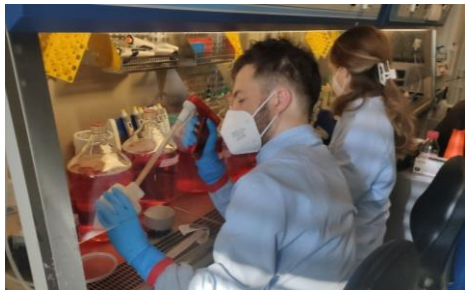
Example: Genome-wide high-content screen in CAR T cells

3 donors x 8 readouts x 77,000 gRNAs x 1000x coverage → >12 billion CAR T cells



Tumor cells vs. CAR T cells





Acknowledgements

Bock Lab: Cosmas Arnold, Raphael Bednarsky, Cecilia Georges, Mustapha Jaiteh, Viktoriia Kartysh, Thomas Krausgruber, Sabrina Ladstätter, Wentao Li, Jenny Lin, Amelie Nemc, Adele Nicolas, Anne-Christine Orts, Eugenia Pankevich, Francesco Piras, Stephan Reichl, Daria Romanovskaia, Moritz Schäfer, Varun Sharma, Peter Stepper, Rob ter Horst, Fangwen Zhao

Lab Alumni: Nathan Sheffield (now: University of Virginia), Christian Schmidl (now: RCI / Uni Regensburg), Florian Halbritter (now: CCRI Vienna), Matthias Farlik (now: MedUni Vienna), Johanna Klughammer (now: LMU Munich Gene Center), André Rendeiro (now: PI at CeMM), Nikolaus Fortelny (now: University of Salzburg), Peter Traxler (now: MedUni Vienna), Lukas Folkman (now: Griffith University), Paul Datlinger (now: Illumina AI Lab, San Francisco)

CeMM & MedUni Wien: Andreas Bergthaler, Christoph Binder, Kaan Boztug, Ulrich Jäger, Stefan Kubicek, Georg Stary, Giulio Superti-Furga, Georg Winter & many more

Collaborations: Mihai Netea, Eleni Tomazou, Human Cell Atlas, ELLIS & many more

Funding

MedUni Vienna & Austrian Academy of Sciences
Austrian Science Fund (2x FWF SFB)
Ludwig Boltzmann Society (LBI-RUD)
EU Horizon 2020 & Horizon Europe
ERC Starting & Consolidator grants

European Commission | Horizon 2020 European Union funding for Research & Innovation

https://bocklab.org
https://twitter.com/BockLab
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https://cemm.at

BSF | Biomedical Sequencing Facility

Alberto Alises, Michelle Chan, Lina Dobnikar, Diana Drobná, Veronika Mancikova, Michael Schuster, Carina Suete, Benjamen White

We are looking for ambitious students & postdocs!