Biobanks and Personalized Medicine: The FHRB Biobank Concept

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University of Helsinki
Population-based vs. disease/research-oriented biobanks

- Population-based biobanks
  - Large patient numbers, “simple” sample collection
  - Pathology samples as the backbone

- Focused, disease-specific biobanks
  - Smaller patient numbers
  - Extensive sample collection/patient
  - Multiple collection instances (diagnosis, remission, relapses)

- Need for both, not competitive, complementary
Some challenges for (cancer) biobanks

- Site and temporal variation inherent to cancer
  - Repeated sampling, multisite sampling

- Clinical annotation of biological/biobank samples
  - No uniform, national/international quality registers for clinical care and research
  - Annotation of drug response phenotypes
Example: selecting biobank samples for drug research from clinical annotation data

• Commonly, new drugs are tested in a population of patients resistant to conventional therapy

• **Search criteria**: pick biobank samples from those patients who have first responded to conventional therapy, but then lost the response (secondary resistance)
  
  • Current patient records/charts (FIN): not possible
  • Other hospital databases (lab, pathology etc): not possible/difficult, indirect
  • Only feasible with structured, disease-specific evaluation of therapy outcome
Finnish Hematology Registry and Biobank

Example of a disease-specific population-based biobank

Clinics

Biobank

Profiling and drug testing technologies

Research
FHRB – owners and partners

• Finnish Red Cross Blood Service
• FIMM – Institute for Molecular Medicine Finland
• Finnish Association of Hematology

• Contractual partners: all hospital districts treating hematological patients (n=21)
Finnish Hematology Registry and Biobank - FHRB

- National registry and biobanking effort that aims to include all university and regional medical centers by 2015 in a population-based manner.
- High-quality samples from all patients with a hematological disease diagnosis.
- Bone marrow aspirates and blood samples collected at diagnosis, remission and relapse(s). Skin biopsy taken at first sampling.
- Samples processed and coded by the Finnish Red Cross Blood Service and then stored at FIMM as viable frozen mononuclear cells, frozen plasma and DNA.
- Researchers (incl. pharma) apply to the FHRB board for use of samples.
- Data returned to FHRB database
Flow chart

Patient consent

Sample, data collection
- Hospitals

Sample processing
- Finnish Red Cross Blood Service (centralized)

Biobanking
- FIMM

FAH

Clinical registry

Experiments, discovery
- Research groups, pharma

Clinical applications
- Diagnostics
- Imaging
- Targeted therapies

- 30 mL blood, 30 mL bone marrow, skin biopsy (opt.)
- at diagnosis, remission (opt.), relapses
- all university and central hospitals (n=21)
FHRB = Clinical registry + Biobank
# FHRB clinical quality register & sample inventory: real-time data entry

### Table 1: Hematological Data

| Case 
<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnosis &amp; Procedure</strong></td>
</tr>
<tr>
<td><strong>4.4.2013</strong></td>
</tr>
<tr>
<td>25.5.2013</td>
</tr>
</tbody>
</table>

### Table 2: Laboratory Results

<table>
<thead>
<tr>
<th>Date</th>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.4.2013</td>
<td>Blood/EDTA</td>
<td>1</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>MNC/PROEN</td>
<td>7</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>MNP/PHEL</td>
<td>1</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>Plasma/EDTA</td>
<td>10</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>Plasma/Heparin</td>
<td>10</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>SERUM</td>
<td>7</td>
</tr>
<tr>
<td>8.4.2013</td>
<td>TISSUE/S</td>
<td>1</td>
</tr>
<tr>
<td>25.5.2013</td>
<td>Blood/EDTA</td>
<td>1</td>
</tr>
<tr>
<td>25.5.2013</td>
<td>MNC/PROEN</td>
<td>7</td>
</tr>
<tr>
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<td>Plasma/Heparin</td>
<td>10</td>
</tr>
<tr>
<td>25.5.2013</td>
<td>SERUM</td>
<td>10</td>
</tr>
</tbody>
</table>

---

**Note:** The data above is a screenshot of the FHRB clinical quality register & sample inventory, showing real-time data entry for hematological and laboratory results.
### Taudin status

Vastearvon yhteydessä pyritään tautikan arvioon ja taudin tilanteen lokitelemana oikean taulukon mukaisesti (SHR-muutuja: status-al).

Taulukosta valitaan arvokentat, jokialla oleva paras vaste.

<table>
<thead>
<tr>
<th>Koodi</th>
<th>Lyhenne</th>
<th>Nimike</th>
<th>Määritelmä</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>CR-MRDneg</td>
<td>Remissio, jäännöstävä negatiivinen</td>
<td>Morfolогinen remissio (CR tai CR1) ja molekyyliseen ennetys- tai immunoenfotyyppi-markeerien negatiivinen (jos 2 menetelmää, molemmat negatiivisia)</td>
</tr>
<tr>
<td>2</td>
<td>CR-MRDpos</td>
<td>Remissio, jäännöstävä positiivinen</td>
<td>Morfolогinen remissio (CR tai CR1) ja molekyyliseen ennetys- tai immunoenfotyyppi-markeerien positiivinen (jos 2 menetelmää, molemmat/toinen positiivinen)</td>
</tr>
<tr>
<td>3</td>
<td>CCR</td>
<td>Sytogeneettinen remissio</td>
<td>Morfolогinen remissio (CR tai CR1) ja G-raft/HIS-markeerien negatiivinen; vähintään 20 metafasea tutkittu Ex MRD-markeerilla (molgen/IFT) kytettävissä</td>
</tr>
<tr>
<td>4</td>
<td>CR</td>
<td>Morfolогinen remissio</td>
<td>Normo- tai hypercellulaarin luundy, blastisa &lt; 5%</td>
</tr>
<tr>
<td>5</td>
<td>CR1</td>
<td>Remissio, puutteinen luuttimen tolpuminen</td>
<td>Muuten CR, mutta B-neut &lt;1,0 tai b-trom &lt;100</td>
</tr>
<tr>
<td>6</td>
<td>PR</td>
<td>Osittainen remissio</td>
<td>Luuttimessa blastisa 5-10%</td>
</tr>
<tr>
<td>7</td>
<td>Hypoplasia</td>
<td>Dysplasia/hypoplasia</td>
<td>Luuttimella morfologia hypoplasia/dysplasia, blastisa ≤10%</td>
</tr>
<tr>
<td>8</td>
<td>RD</td>
<td>Refraktäärin tauti (RD)</td>
<td>Luuttimessa blastisa &gt;5% ja aiemmin tehtyesti negatiivinen jäännöstävämarkeeri positiivinen ja lisääntävää vähintä kahdeksassa remissiössä, jossa terapeuttinen toiminto tai</td>
</tr>
</tbody>
</table>
FHRB: Samples biobanked/patient

- 10 ml serum tube → Centrifuge → 10 serum aliquots (300 μl)
- 10 ml heparin tube → Centrifuge → 10 heparin aliquots (300 μl)
- 10 ml EDTA tube → Centrifuge → 10 EDTA plasma aliquots (300 μl)
- 30 ml bone marrow → Ficoll → Cell pellets, Alive cells
- 10 ml EDTA tube, skin biopsies → DNA extraction → Germ-line DNA, diseased DNA

50 sample tubes/patient/sampling: all samples stored in LN₂
FIMM: centralized LN$_2$ based sample storage system

- Current capacity >1M samples (2 ml cryotubes); expandable
- Samples stored in LN$_2$ vapour phase (-180°C)
- Trained personnel, audited processes
- IT-systems
  - Sample management
  - Storage conditions surveillance
  - Safety procedures
FHRB-project: biobanking timeline

• Started in Dec 5, 2011; currently all newly-diagnosed and relapse patients sampled (acute leukemias, MDS, myeloma, MPN, CLL, CML)

• All University hospitals: Helsinki, Turku, Tampere, Oulu, Kuopio collecting samples

• All other hospitals treating hematological patients by the end of 2015

• 300-500 patients/year (50 000 samples/year)
FHRB is part of routine patient care

- **A biobank lab request**
  - 453 €/sample (+157 € for Fridays)
    - Data registry
    - Sampling, transport
    - Sample processing
    - Biobanking (6-7 yrs)

- **Costs included in the daily care of the patients (laboratory budget)**
  - ”good care of a hematological patient”
  - Cost of one PCR test
  - <3% of university clinic lab budget
Continuous quality control: cell viability

Cell viability - 25% CM

Normalized luminescence

Day 0 - CM  Day 3 - CM  Day 5 - CM

FHRB operational group quality control round September 2013
# FHRB sample status

<table>
<thead>
<tr>
<th>All samples / Status / Diagnosis</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>All samples</td>
<td>729</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>660</td>
</tr>
<tr>
<td>Remission</td>
<td>126</td>
</tr>
<tr>
<td>Relapse</td>
<td>87</td>
</tr>
</tbody>
</table>

### Patients

<table>
<thead>
<tr>
<th>Diagnosis / Status / ICD-O</th>
<th>Patients</th>
<th>MNC (viably frozen)</th>
<th>MNC Serum (EDTA)</th>
<th>Plasma (EDTA)</th>
<th>Blood Li-EDTA (skin)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Akuutti lymfaattinen leukemia</td>
<td>60</td>
<td>773</td>
<td>435</td>
<td>798</td>
<td>814</td>
</tr>
<tr>
<td>Akuutti myelocinen leukemia</td>
<td>190</td>
<td>2786</td>
<td>1453</td>
<td>2572</td>
<td>2616</td>
</tr>
</tbody>
</table>

### Diagnosis

- **Blastinen plasmasytoidinen dendritisolheumia**
  - 9727
    - MNC: 2
    - MNC Serum: 0
    - Plasma: 20
    - Blood Li: 2
    - Biopsy: 0

- **Akuutti erilaistumaton leukemia**
  - 9801
    - MNC: 0
    - MNC Serum: 0
    - Plasma: 0
    - Blood Li: 0
    - Biopsy: 0

- **9808 AL, sekafenotyppi, B/myelocinen, muutoin spesiifimaton**
  - 1
    - MNC: 20
    - MNC Serum: 20
    - Plasma: 10
    - Blood Li: 10
    - Biopsy: 1

- **9809 AL, sekafenotyppi, T/myelocinen, muutoin spesiifimaton**
  - 0
    - MNC: 0
    - MNC Serum: 0
    - Plasma: 0
    - Blood Li: 0
    - Biopsy: 0

- **9840 AML, akuutti erytroleumia (FAB M6)**
  - 1
    - MNC: 0
    - MNC Serum: 10
    - Plasma: 10
    - Blood Li: 10
    - Biopsy: 1

- **9861 AML ja mutaatioitunut CEBPA (ehdollinen)**
  - 1
    - MNC: 20
    - MNC Serum: 20
    - Plasma: 10
    - Blood Li: 10
    - Biopsy: 1

- **9861 AML ja mutaatioitunut NPM1 (ehdollinen)**
  - 2
    - MNC: 40
    - MNC Serum: 28
    - Plasma: 20
    - Blood Li: 20
    - Biopsy: 2

- **9866 AML ja t(6;9)(p23;q34); DEK-NUP214**
  - 0
    - MNC: 0
    - MNC Serum: 0
    - Plasma: 0
    - Blood Li: 0
    - Biopsy: 0

- **9866 APL ja t(15;17)(q22;q12); PML-RARA* (FAB M3)**
  - 5
    - MNC: 22
    - MNC Serum: 3
    - Plasma: 50
    - Blood Li: 48
    - Biopsy: 5

- **9867 AML, myelomonosyyttileukemia (FAB M4)**
  - 3
    - MNC: 33
    - MNC Serum: 6
    - Plasma: 29
    - Blood Li: 30
    - Biopsy: 3

- **9871 AML ja inv(16)(p13.1q22) tai t(16;16)(p13.1;q22); CBF-B-MYH11**
  - 2
    - MNC: 25
    - MNC Serum: 2
    - Plasma: 20
    - Blood Li: 20
    - Biopsy: 2

- **9872 AML ja minimaalinen erilaistuminen (FAB M0)**
  - 5
    - MNC: 44
    - MNC Serum: 6
    - Plasma: 49
    - Blood Li: 50
    - Biopsy: 5

- **9873 AML ilman kyvyymistä (FAB M0)**
  - 6
    - MNC: 113
    - MNC Serum: 82
    - Plasma: 59
    - Blood Li: 56
    - Biopsy: 6
FHRB – a new biobank

- Finnish biobank act September 1, 2013
- TUKIJA (National Committee on Medical Research Ethics) approval November 2013
- VALVIRA (National Supervisory Authority for Welfare and Health) approval July 14, 2014
- Transfer of FHRB project samples to FHRB biobank (October 2014).
- First national, disease-focused biobank in Finland
- Accepting applications for samples autumn 2015
  - Academic research groups
  - Pharma
FHRB - organization

Administrative and legal responsibility

- Board of owners
- Steering group
- Executive officer

Operative responsibility

- Scientific advisory board
- Operative unit

Sample applications

Hematology Research Unit Helsinki
Infrastructure and collaborative environment required to practice individualized systems medicine in acute myeloid leukemia (AML)

Why AML?
Sampling over the course of disease
Cancer always accessible
Ex-vivo functional drug testing easier
AML: survival from relapse by age

Genomic and molecular landscape in AML

Number of patient samples

23 significantly mutated genes in 200 AML patient samples

The Cancer Genome Atlas (TCGA) dataset

Chromatin modifiers (30.5%) MLL fusions, MLL PTD, NUP98-NSD1, ASXL1, EZH2, KDM6A, other modifiers

Transcription factor fusions (18%) PML-RARA, MYH11-CBF, RUNX1-RUNX1T1, PICALM-MLLT10

Myeloid transcription factors (22%) RUNX1, CEBPA, other myeloid transcription factors

Tumor suppressors (16.5%) TP53, WT1, PHF6

Cohesin complex (13%)

DNA methylation (46%) TET1, TET2, IDH1, IDH2, DNMT3B, DNMT1, DNMT3A

Activated signaling (59%) FLT3, KIT, KRAS, NRAS, PTPs, Ser/Thr kinases, other Tyr kinases

Panoramic view of AML, Chen and Chen; Nature Genetics, June 2013

Genomic and Epigenomic Landscapes of Adult De Novo Acute Myeloid Leukemia, New England Journal of Medicine 2013
Molecular diversity: a challenge for drug development

100 newly diagnosed AML patients

AML genotype 1
AML genotype 2
AML genotype 3
AML genotype 5
AML genotype 6
AML genotype 7
AML genotype 98
AML genotype 99
AML genotype 100

New drug 1: Phase I-II
(n=10-40)

Response rate 2/100 = 2%

Study fail
End development
Personalized Medicine
Accurate diagnosis, prognosis and treatment can save your life.

Genomic Medicine

STRATIFIED MEDICINE
Building Hope With Individualized Medicine

Concepts in Pharmacogenomics

WHAT IS PRECISION MEDICINE?

COMPANION DIAGNOSTICS
THE FUTURE OF MEDICINE
Individualized Systems Medicine Overview

Systems medicine approach:
- Data integration
- Repeated sampling
- Feedback to clinic
- Learning system

Drug sensitivity and resistance testing (DSRT)

FHRB biobank

Molecular profiling
Genome Transcriptome Signalome

Leukemia sample work flow

Sample collection
- Bone marrow aspirate
- Peripheral blood
- Skin biopsy

Sample processing
- Mononuclear cell separation
- Protein lysates
- DNA extraction
- RNA extraction

Sample analysis
- Drug screening
- Phospho-protein analysis
- Whole genome/exome sequencing
- RNA sequencing
Augmenting molecular drug discovery/repurposing

- Genome/transcriptome-driven
  - Molecular profiling of leukemic cells
  - Disease-associated genomic variants
  - Rarely directly clinically actionable
  - Molecularly targeted therapies
    - 3-10+ years

- Drug-response phenotype-driven
  - Drug response profiling of leukemic cells
  - Disease-associated drug responses
    - Often directly clinically actionable
    - Unbiased
    - Drug repurposing
  - Molecularly dissection of drug response
  - Molecularly targeted therapies
    - 1-5+ years
Drug sensitivity and resistance testing

- Leukemic patient cells
- Oncology collection 5 conc. 10,000-fold conc. range
- Assay & detection reagents (CellTiter-Glo for viability)
  - 37°C, 72 h
- 300 dose response curves
- Calculate activity
  - Compare to controls
- Novel biological understanding of the disease
- 4 day turnaround
- Find responding cells/patients. Repositioning
- Translate directly to patients
  - 4 day turnaround
  - Calculate activity

Drug sensitivity and resistance testing involves testing the responsiveness of cancer cells to various drugs. This process is crucial for identifying effective treatments and improving patient care. The protocol involves collecting patient samples, growing cells under controlled conditions, and testing their response to a wide range of drugs. By analyzing the dose response curves, researchers can determine which drugs are most effective and tailor treatments for individual patients.

The process begins with the collection of patient cells. These cells are then cultured under conditions that simulate the body, allowing researchers to observe how they respond to different medications. The cell viability is measured using reagents such as CellTiter-Glo, which provides a quantitative assessment of cell health.

The collected data is then analyzed to identify patterns and trends. This information is crucial for understanding the biological mechanisms of the disease and for developing new treatment strategies. The ultimate goal is to translate these findings directly to patients, improving outcomes and advancing the field of oncology.

In summary, drug sensitivity and resistance testing is a critical component of personalized medicine. By identifying which drugs are most effective for an individual patient, we can tailor treatments to maximize efficacy and minimize side effects, ultimately leading to better patient outcomes and improved overall health care.
Assembling a concise and comprehensive functional screening collection

• **Currently 306 oncology-focused active substances/drugs**
  – Approved small molecule oncology substances
  – Oncology-related approved substances
  – Major investigational oncology compounds
  – Probe compounds with unique and cancer-relevant activities

• 135 approved
• 171 investigational
• 65 conventional chemotherapeutics
• 20 hormone therapy drugs
• 119 kinase inhibitors
• 33 epigenetic/differentiating drugs
• 55 other targeted drugs
• 10 immunosuppressants
Unsupervised hierarchical clustering creates functional taxonomy of patients

Controls and patient samples

AML patient samples can be divided into five broad groups

Disease stage
Navitoclax
Ruxolitinib
Dexamethasone
MEKi
PI3K/mTORi
Quizzartinib
Sunitinib
Dasatinib
Topoisomerase IIi

TP53
RUNX1
DNMT3A
NRAS/KRAS
MLL-X fusions
WT1
ETV6-NTRK3
Kit
NUP98-NSD1
FLT3-ITD
NPM1
IDH1/IDH2
PTPN11

Adverse karyotype
FAB subtypes

Challenge with big data management, rapid integration and interpretation to give feedback to the clinic.

Drug sensitivity testing

Exome sequencing

Integration & Interpretation & Feedback to clinic (4 days to 2 weeks)

Gene expression

Phosphoproteomics

Fusion genes
In personalized medicine, the patient (person) is at the center.
Example I

DRUG REPURPOSING FOR CHEMOREFRACTORY AML
Patient case I: FHRB.600

• 50-year-old male, previously healthy
• AML FAB M5, normal karyotype, FLT3-ITD
• Failed 3 consecutive induction therapies
• Febrile, pancytopenia, WHO 3

• Molecular profiling: exome and transcriptome sequencing and targeted phospho-proteomics of serial samples
• DSRT: Selection of an off-label drug-combination as per compassionate use based on ex vivo drug screening results
FHRB.600: NUP98-NSD1 fusion

NUP98 (chr 11)  
NSD1 (chr 5)

Ex vivo DSRT results

DSS: Drug sensitivity score = (AUC/Total Area) \times 100/\log(100-\min)

- control
- AML

% viability

Conc (log M)

Treatment response

Bone marrow blast cell %
Neutrophil count

Days from start of therapy

Bone marrow blasts + promonocyte count (%)

Neutrophil count (10⁹/L)

Sensitive sample
Exome sequencing
CNA
RNAseq
Expression
Phosphoproteomics

Resistant sample
Exome sequencing
CNA
RNAseq
Expression
Phosphoproteomics

Dasatinib
Sunitinib
Temsirolimus

Infection
Loss of *ex vivo* drug sensitivity
Clonal evolution

⇒ Novel candidate combination therapy for NUP98-NSD1-driven AML

Functional investigation of NUP98-NSD1/FLT3 AML

**Biobanked patient material:**
- 3 patients

**Cell models:**
- **Ba/F3** (murine, C3H, pro-B-cell, BM)
  - Empty vector
  - NUP98-NSD1 variant 1
  - NUP98-NSD1 variant 2
- **32D** (murine, C3H, myeloblast-like, BM)
  - Empty vector
  - NUP98-NSD1 variant 1
  - NUP98-NSD1 variant 2
- **Balb/c BM Lin (-)**
  - Empty vector
  - NUP98-NSD1 (pre)
  - NUP98-NSD1/FLT3-ITD (pre)
- NUP98-NSD1/FLT3-ITD (M3)
- **BL/6 BM Lin (-)**
  - Empty vector
  - NUP98-NSD1/FLT3-ITD (M1)

**Transgenic model**
- In collaboration with Prof. Juerg Schwaller (Basel)
Example II

DRUG REPURPOSING – T315I MUTATED CML
Patient case: FHRB.1408 (CML BC T315I+)

- **February 2012**: male aged 35 diagnosed of CML in lymphatic blast phase
  - Imatinib 600 mg monotherapy, hematologic response

- **April 2012**: hematologic relapse, switch to dasatinib 140 mg x1, no response. HD chemorherapy => partial response
  - BCR-ABL1 KD sequencing: \textcolor{red}{BCR-ABL1_{T315I}} 100%

- **August 2012**: AlloHSCT

- **Current status (May 2014)**: BM in CR

- Exome sequencing (100x): missense mutations in RUNX1, ABL1, NRK, ABCA13
20 most selective drugs

- Dexamethasone
- Methylprednisolone
- Ponatinib
- AZD8055
- Prednisolone
- FK–866 HCl
- Axitinib
- OSI–027
- Vincristine
- Mocetotinostat
- Plicamycin
- GDC–0980
- Prima–1 Met
- Neratinib
- PF–04691502
- Danusertib
- Trametinib
- TAK–733
- Entinostat
- Clofarabine

Multikinase inhibitor
PI3K/mTOR KI
Namt inhibitor
VEGFR-1/2/3 TKI
mTORC2/mTORC1 inhibitor
PI3K/mTOR KI
p53-activating drug
EGFR/HER-2 inhibitor
PI3K/mTOR KI
Aurora A/B/C KI
MEK-inhibitor
MEK-inhibitor
HDAC-inhibitor
Clinical translation

⇒ Confirmation of results from similar samples in the FHRB biobank
⇒ Formal Phase I/II study; combination therapy; MOA – in partnership with Pfizer
⇒ New, already approved drug for T315I-mutated leukemia in 2-3 years

Multiple myeloma sample analysis

DSRT
ex vivo drug sensitivity

Methyl sequence epigenomic profile

Exome sequence SNVs, CNVs

RNA sequence
gene expression profile, expressed fusion genes,
small RNAs, lncRNAs
Bone marrow aspirate

CD138+ cell selection

Clinical data
Biobanked samples

DNA
(CD138+ cells and skin biopsy)

RNA
(CD138+ cells)

Drug sensitivity testing

Next generation DNA and RNA sequencing

Biobanked samples

Finnish Hematology Registry and Biobank

Hematology Research Unit Helsinki
Conclusions

• Disease-specific deep biobanks are key components in the development of personalized cancer care

• ISM strategy is one way to implement personalized cancer therapy
  • Novel drug candidates for difficult to treat cancers
  • Drug repurposing: patient selection for formal Phase I-II studies. Patenting opportunities

• Formal Phase II clinical study necessary to validate clinical usefulness (in collaboration with pharmaceutical companies and research consortia)
FHRB

Steering group

Kari Aranko
Tiina Wahlfors
Henna Jalovaara
Outi Huoponen
Eeva Mainio

Tiina Vesterinen
Kimmo Pitkänen
Caroline Heckman
Janna Saarela
Kyösti Sutinen
Olli Kallioniemi

Aimo Strömberg
Mika Peltovaara
Timo Laukkio

http://www.hematology.fi/fhrb
Leukemia Network Helsinki

FIMM

**Personalized Cancer Medicine**
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- Samuli Eldfors
- Riikka Karjalainen
- Jarno Kivioja
- Ashwini Kumar
- Heikki Kuusanmäki
- Muntasir Mamun Majumder
- Alun Parsons
- Minna Suvela

**Chemical Systems Biology**
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- Tea Pemovska
- Arjan van Adrichem

**Computational Systems Biology**
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- Petteri Hintsanen
- Agnieszka Szwajda
- Bhagwan Yadav

**Individualized Systems Medicine**
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- Taija af Hällström
- Henrik Edgren
- Poojitha Kota Venkata
- Disha Malani
- John Patrick Mpindi
- Astrid Murumägi
- Päivi Östling
- Maija Wolf

**Technology Center**
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- Evgeny Kulesskiy
- Laura Turunen
- Anna Lehto
- Ida Lindenschmidt
- Pekka Ellonen
- Maija Lepistö
- Sonja Lagström
- Sari Hannula
- Pirkko Mattila
- Aino Palva

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