Data science og big data kan bidra til bedre helse

Arnoldo Frigessi

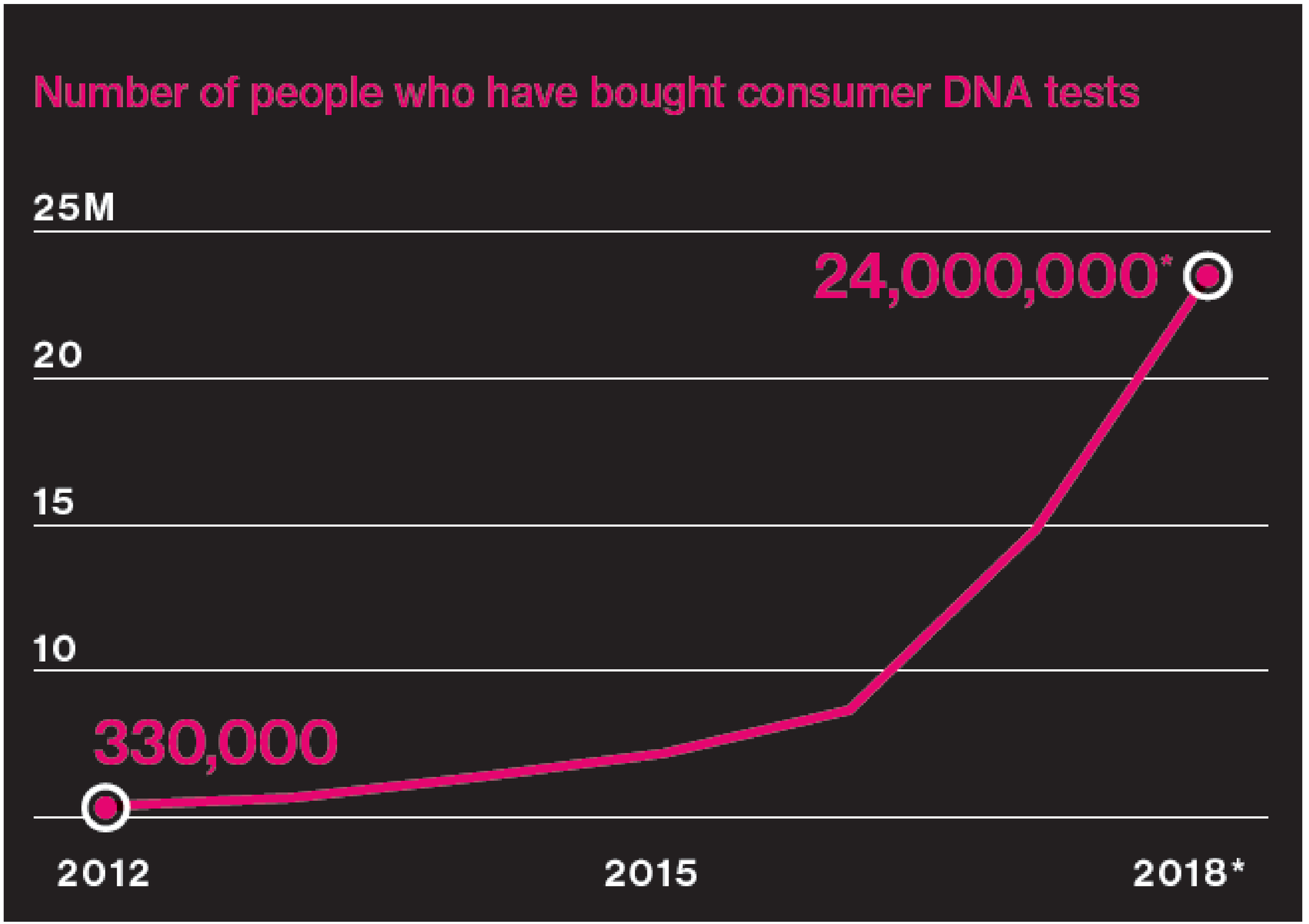
OCBE Oslo Centre for Biostatistics and Epidemiology
University of Oslo
Oslo University Hospital

frigessi@medisin.uio.no

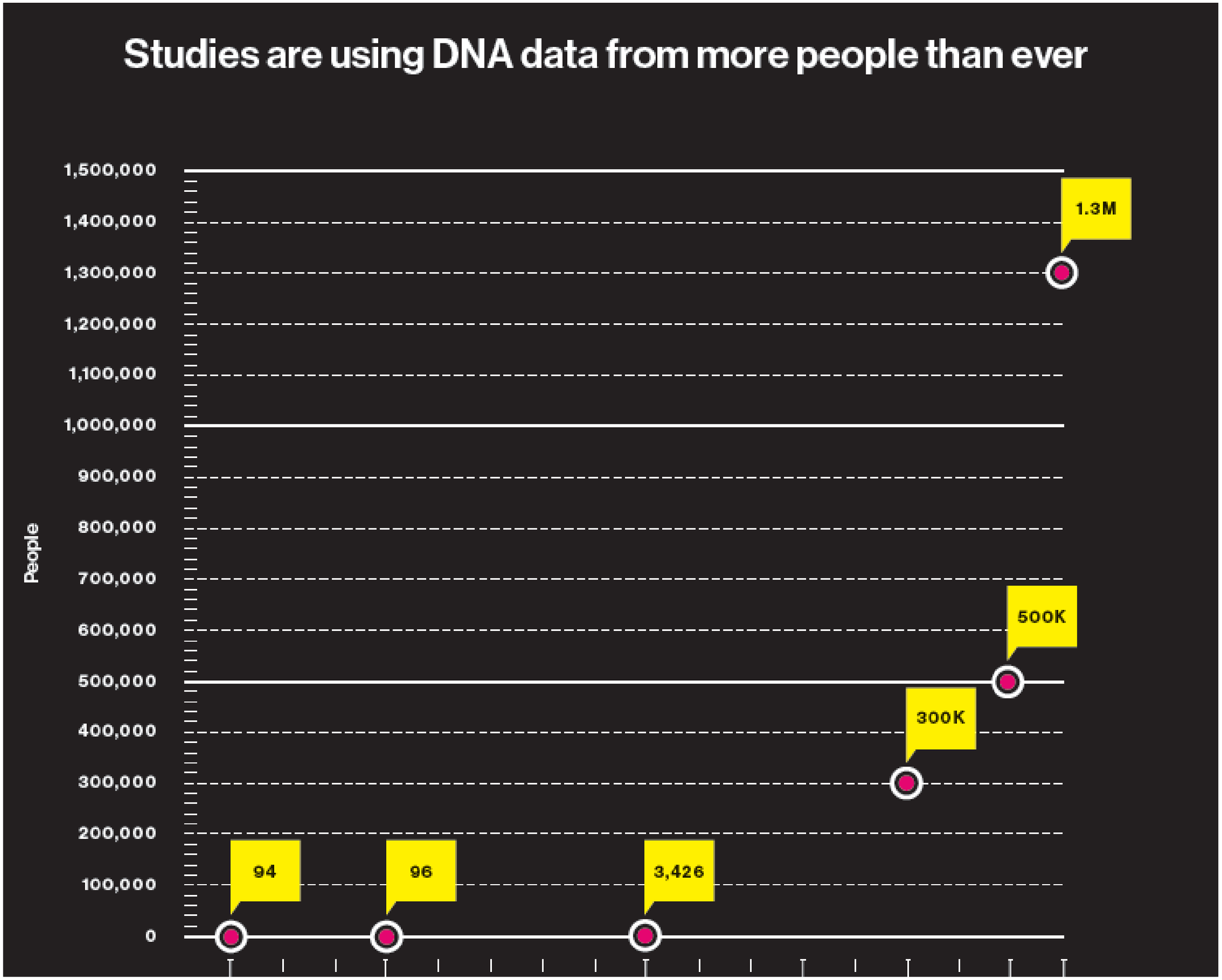
Big Data in Health

1. Genetics and genomics
2. Electronic health records, incl. images, videos, text
3. Biomedical sensors, incl. wearables, apps

Number of people who have bought consumer DNA tests



Studies are using DNA data from more people than ever



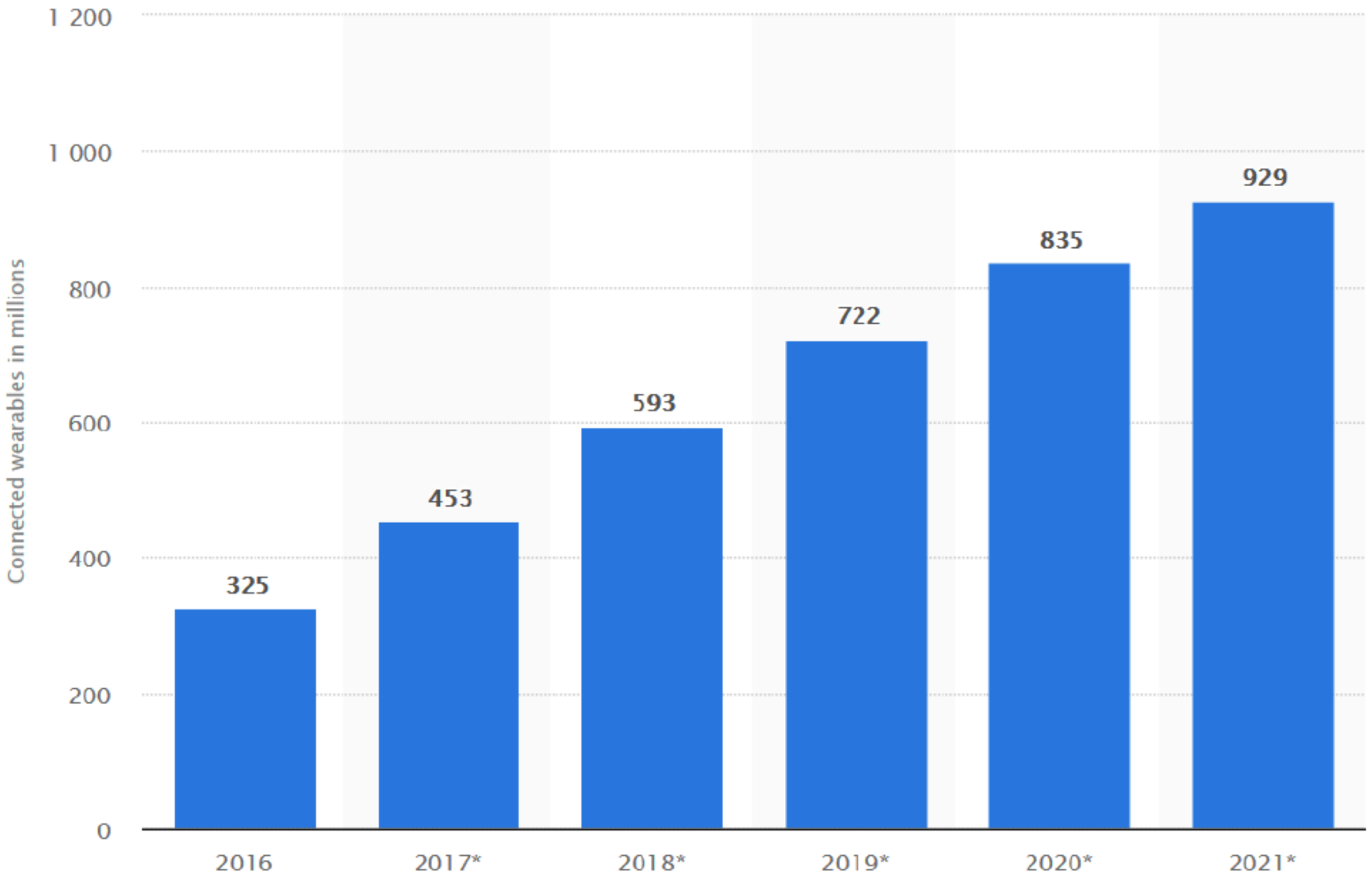
Electronic patient record for 4,3 million people

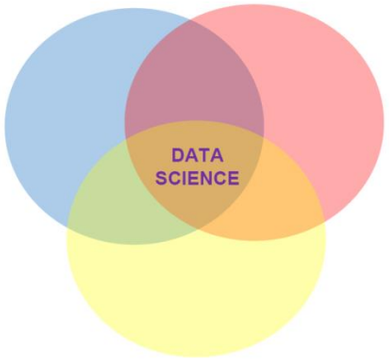
DIPS ASA is the leading supplier of eHealth systems to Norwegian hospitals. Our eHealth solutions spans from EHR and hospital information systems to laboratory systems and an advanced CPOE solution that can be plugged in to any 3rd party system. DIPS Arena is a fully integrated patient record system including closed loop medication, charting, booking and planning, electronic document workflow, CPOE, multimedia and reporting.

Over 80.000 users

DIPS ASA has contracts with three of Norway's four regional health trusts, including five of the six university hospitals in Norway. Our solutions has more than 80,000 professional daily users and is thus one of Norway's

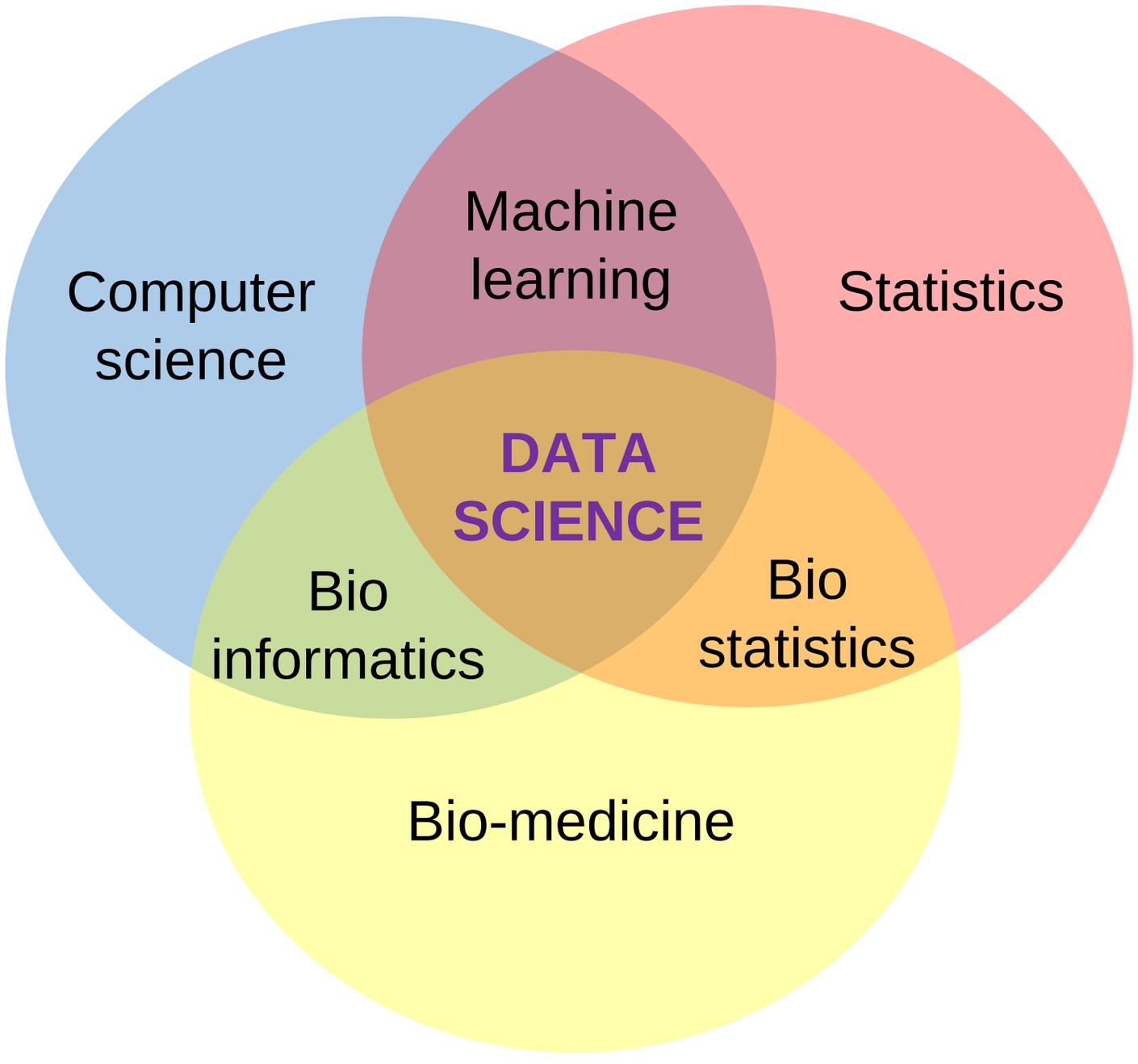
Millions of connected wearables worldwide



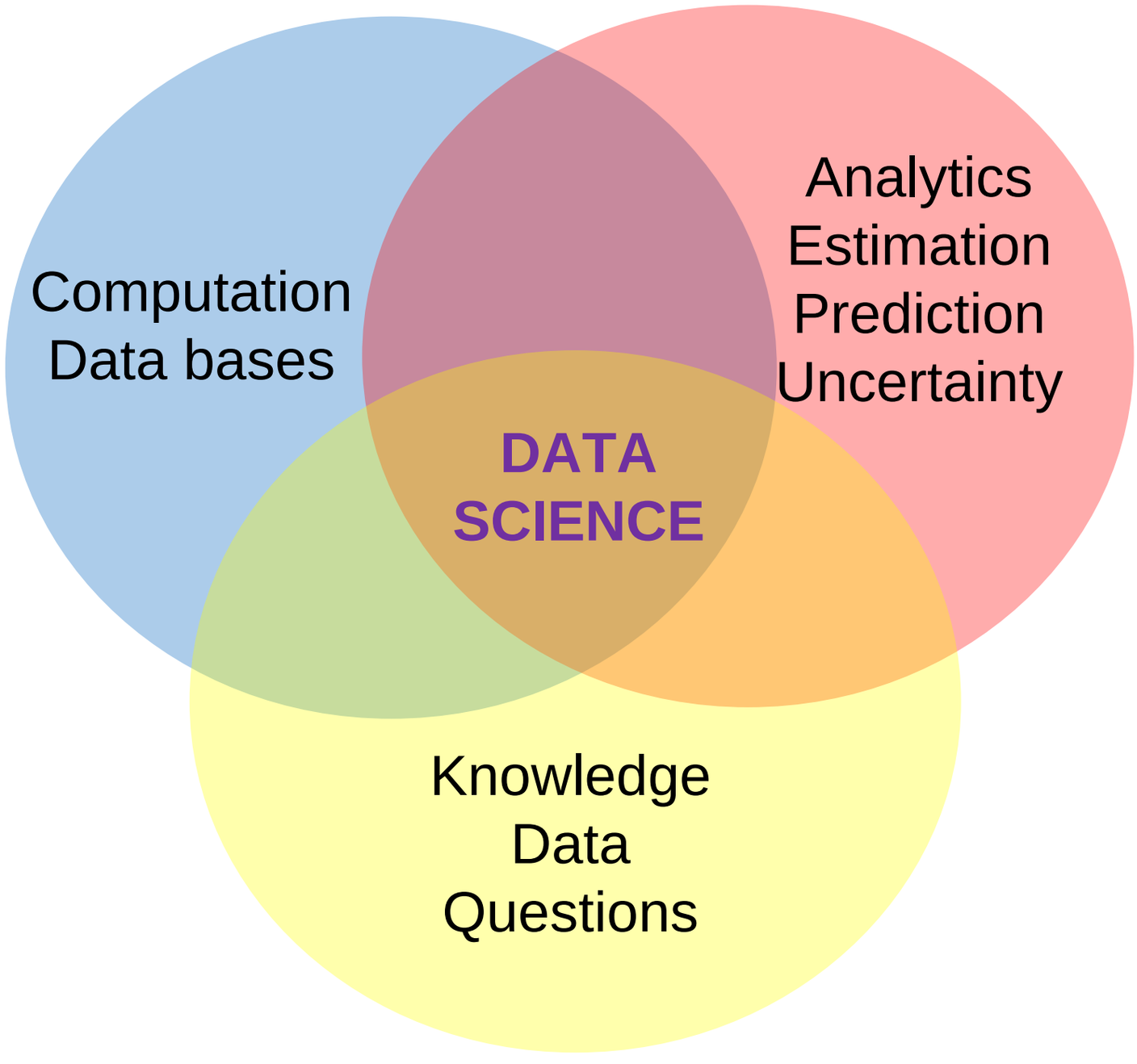


1. Geography

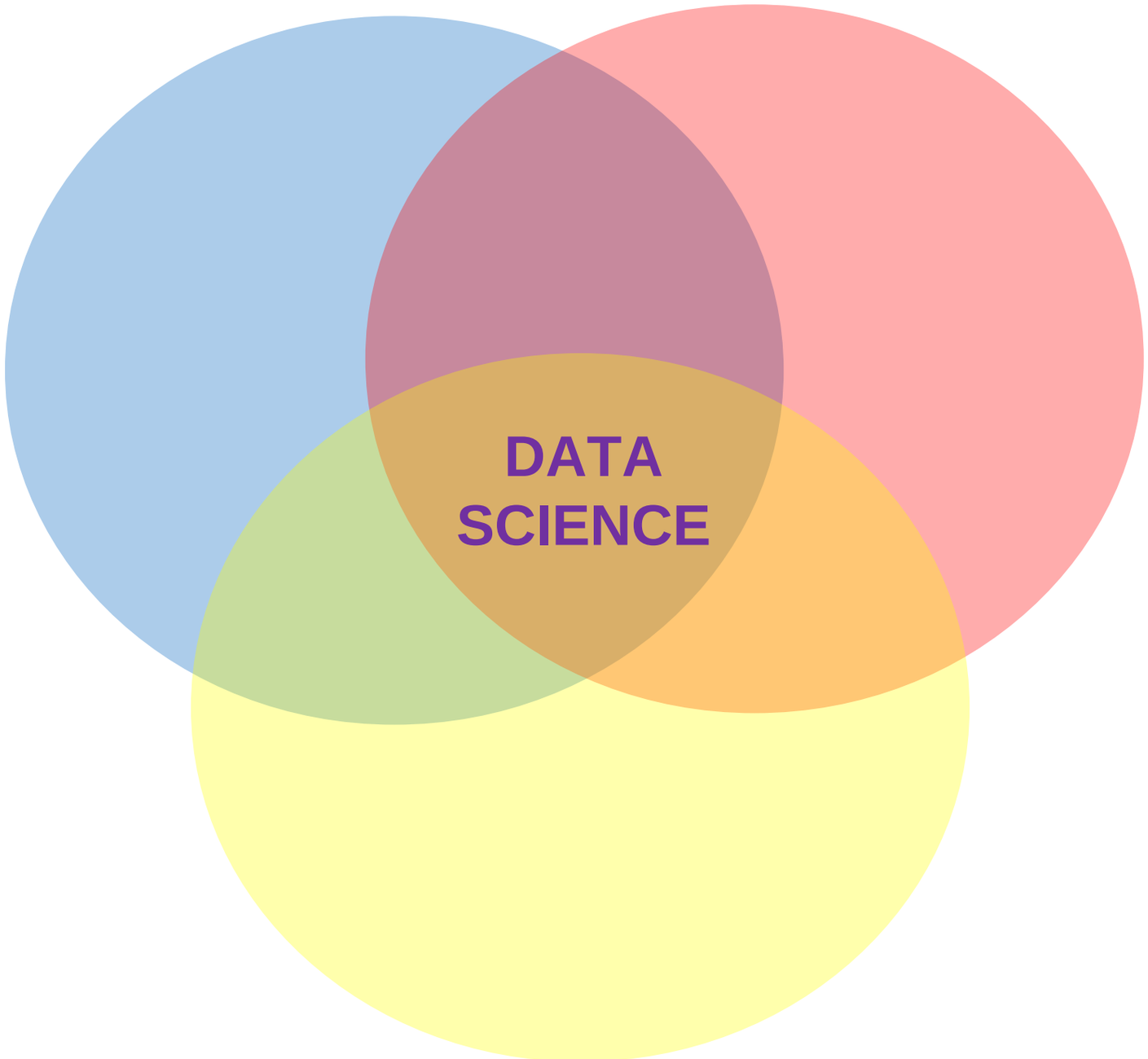
Data Science in Health



Data Science in Health

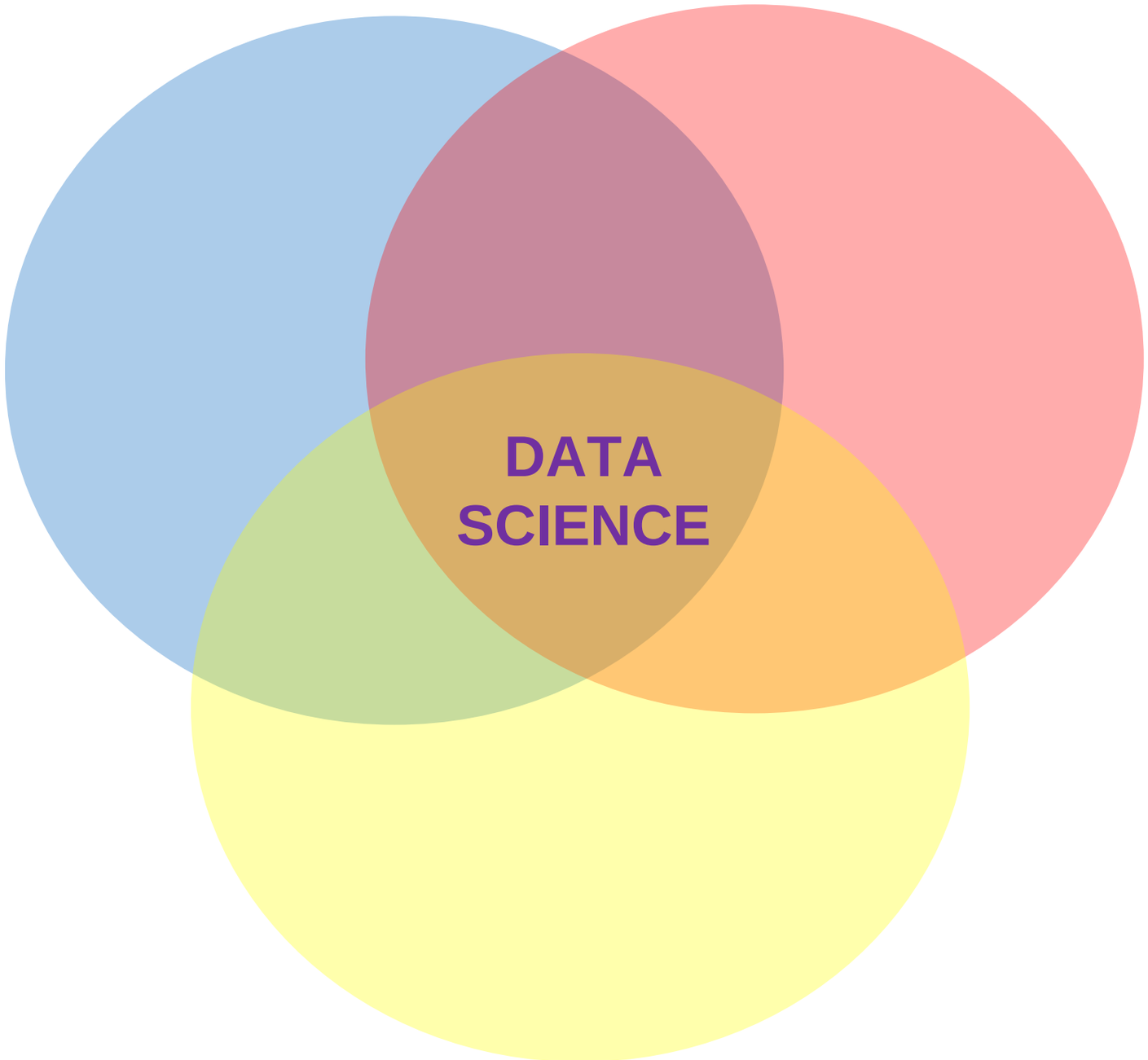


Data Science in Health



- Algorithms
- Models = digital twins
- Decision support
- What-if machines
- Process automation
- Robots
- …

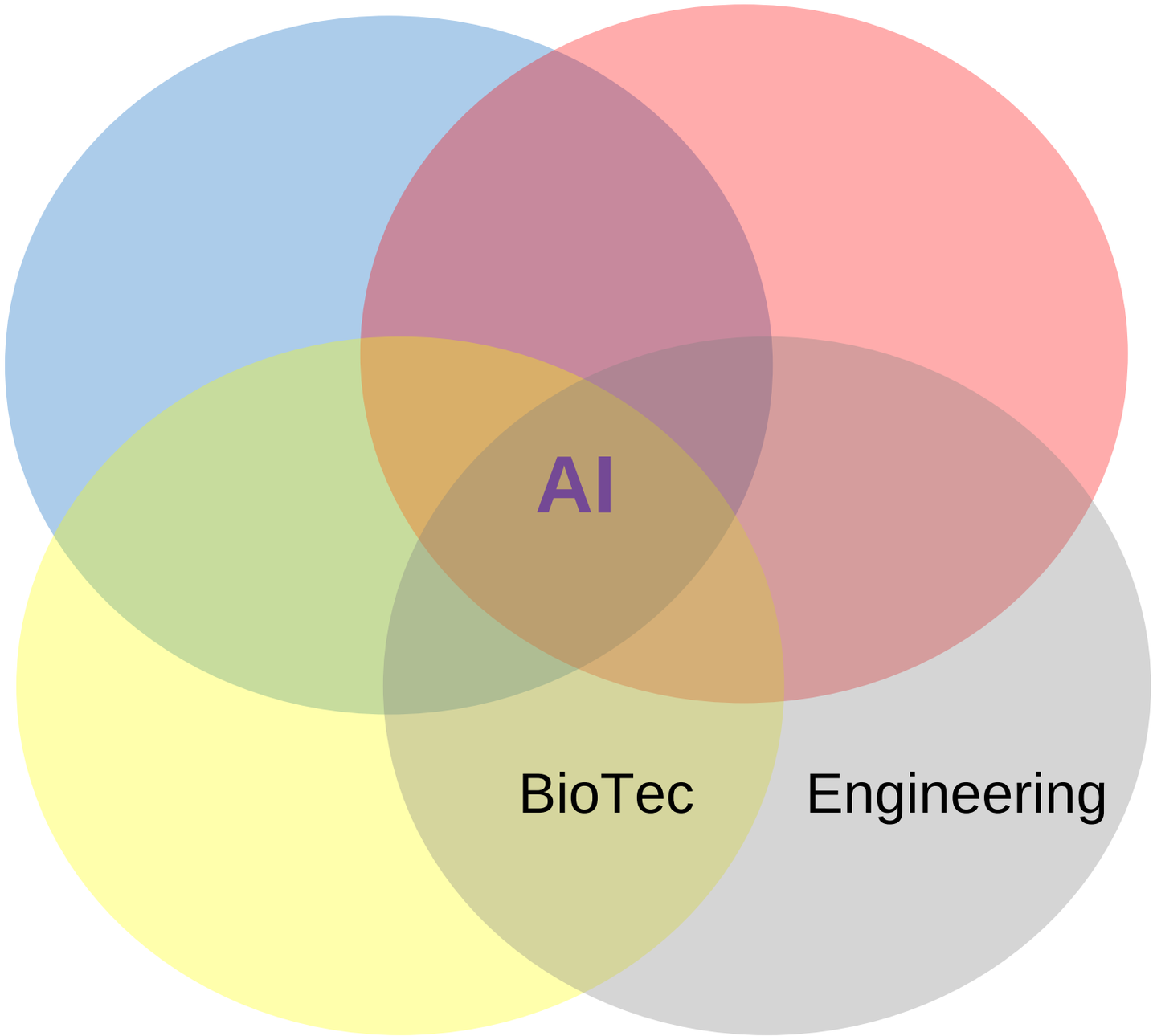
Data Science in Health

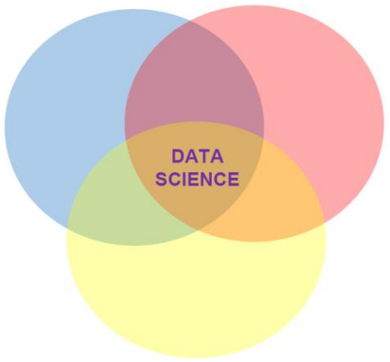


- Prevent
- Diagnose
- Treat
- Prognose
- Aftercare

- Organise
- Lean
- Patient safety

AI in Health

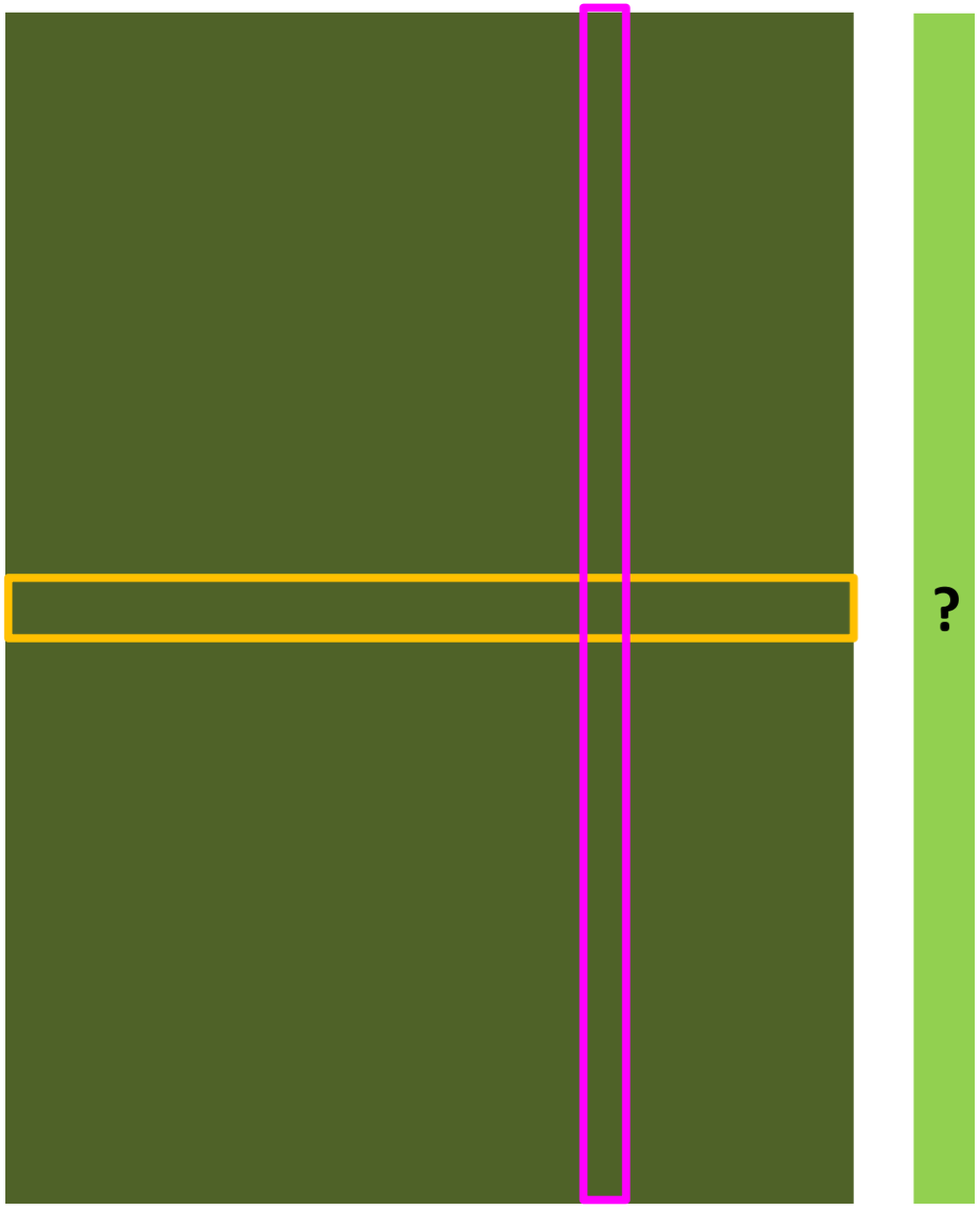




2. Methods

Factors (genes, clinics,…)

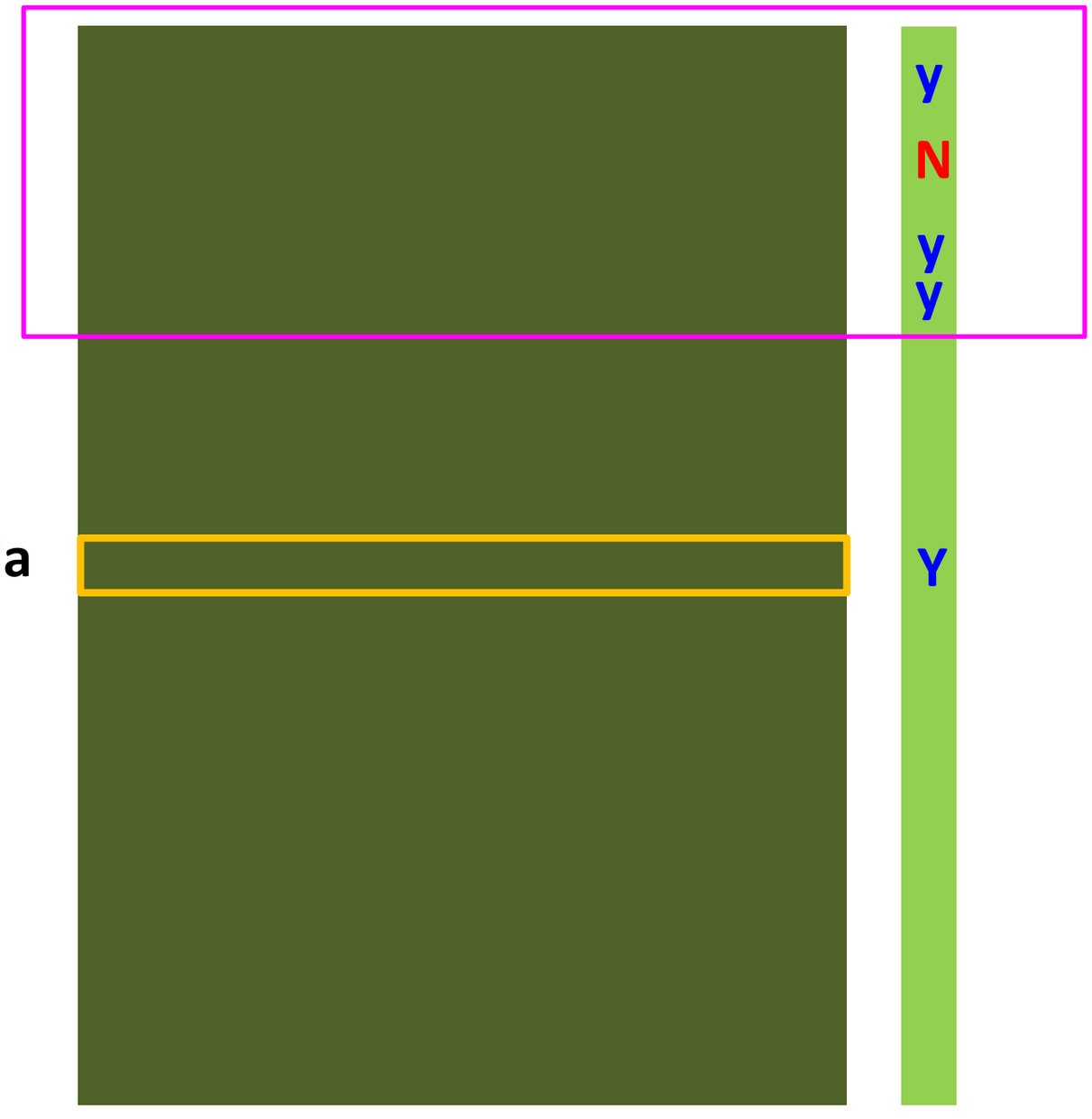
Therapy works ?



Angela

?

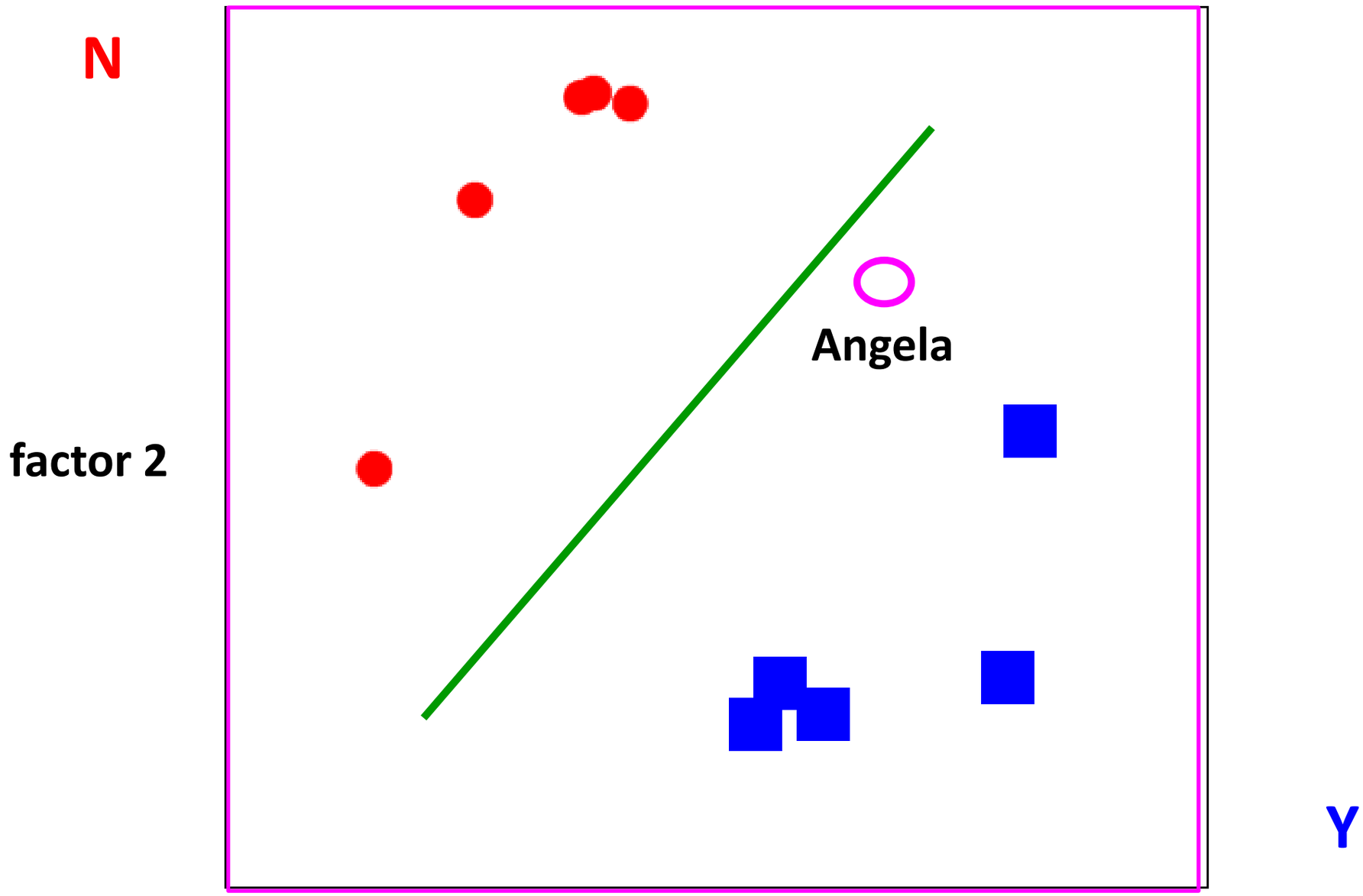
Therapy works ?



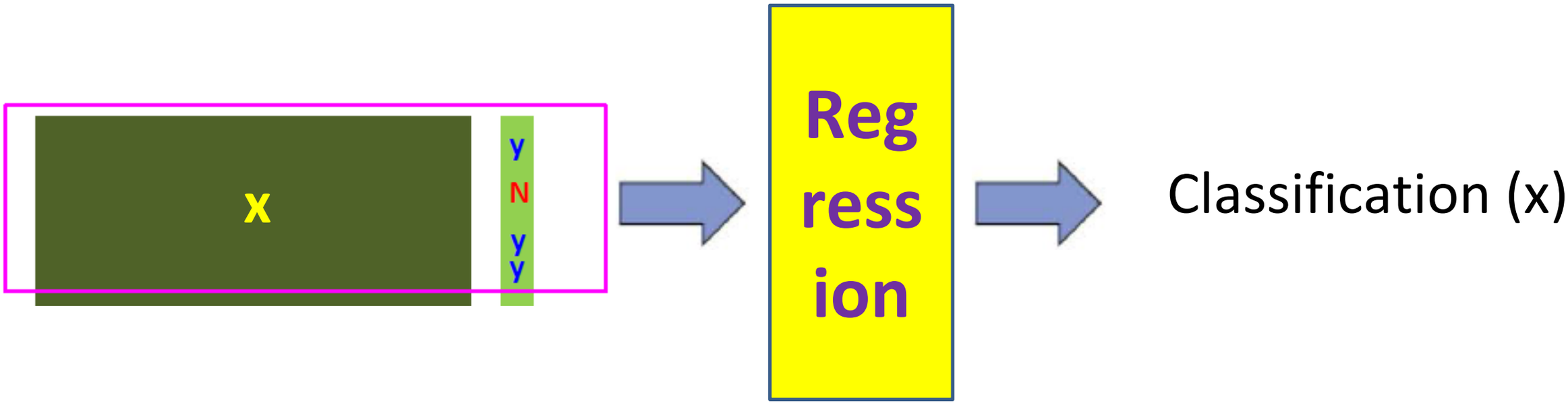
We LEARN a RULE from complete data

which we apply to ANGELA

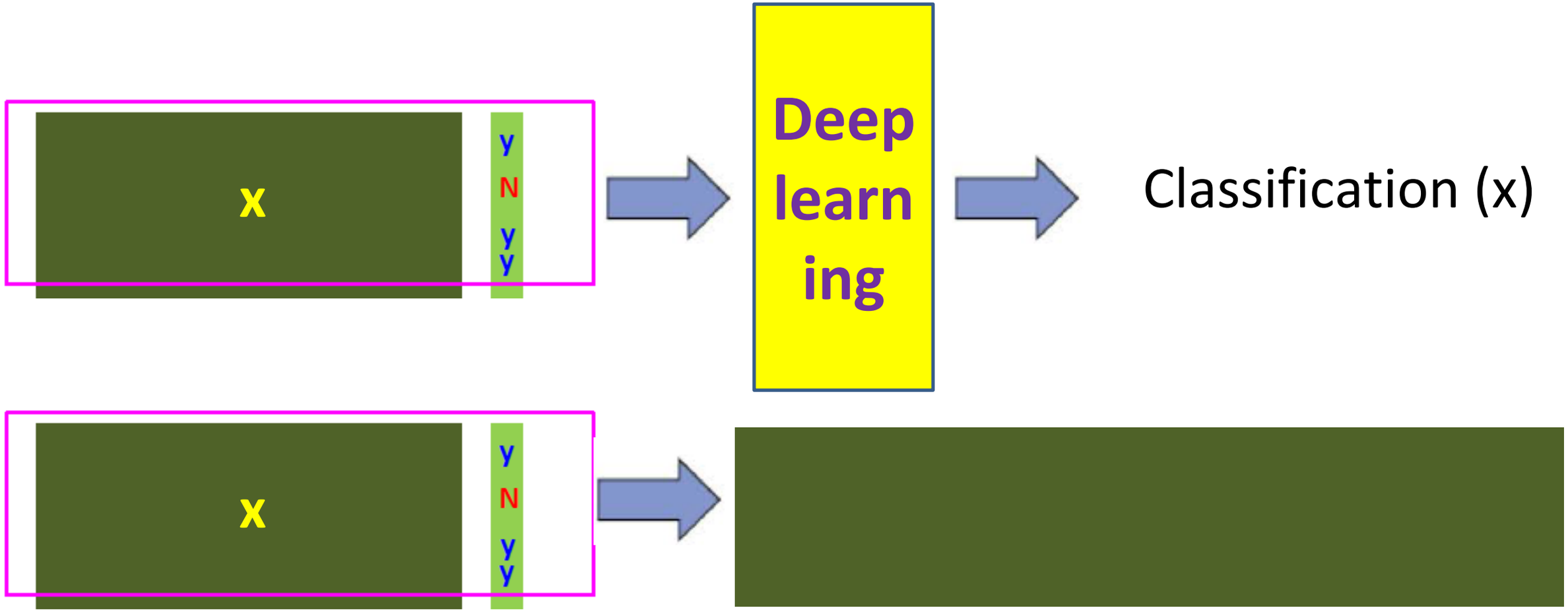
- **Supervised: a Training data set with patients with known outcome.**



- **Supervised: People with outcome known.**
- **Classification rule**

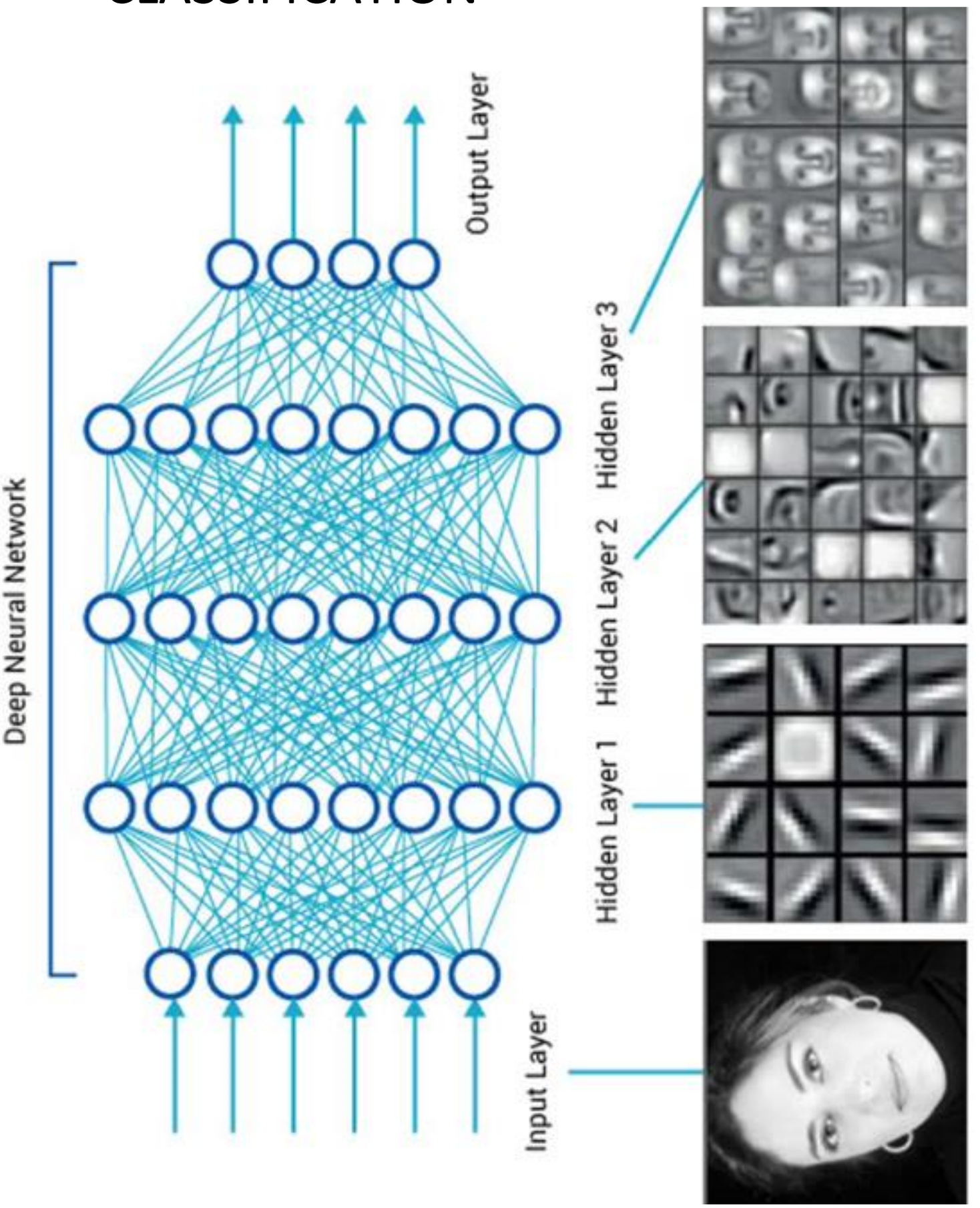


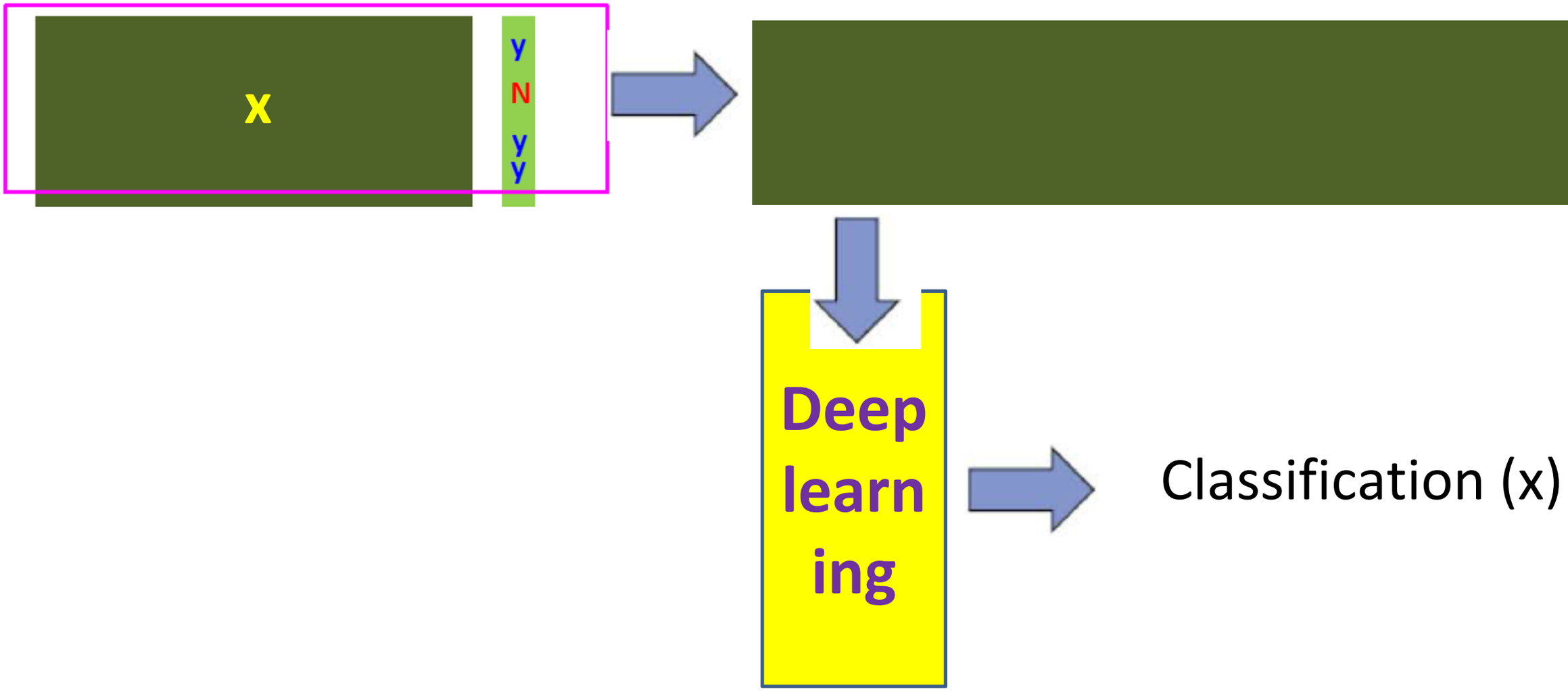
- Regression!
- Using all factors x.
- Producing a level of uncertainty in the classification:
  Angela: Therapy works with probability 73%
  Lukas: Therapy works with probability 53%
- Model based methods
- Can be understood: how the classification depends on x.
- Maybe some x can be intervened on, to improve the classification?



- Deep learning!
- Using all factors x.
- Creates automatically many combinations of x, and among these finds the ones that give best classification

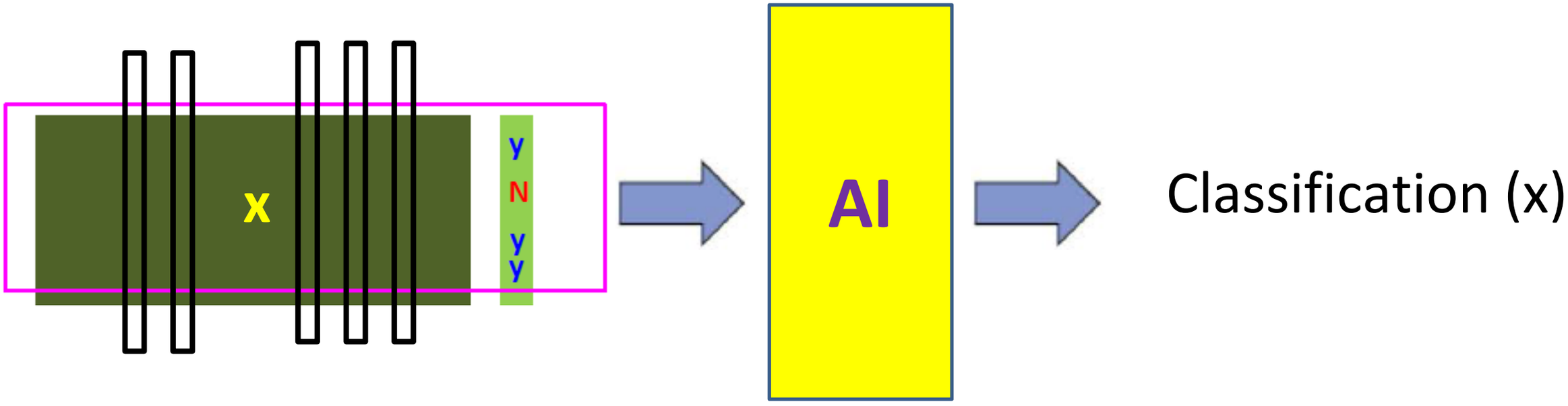
CLASSIFICATION





- Black box model
- Cannot be understood: how the classification depends on x.
- No intervention possible.
- Producing no level of uncertainty in the classification

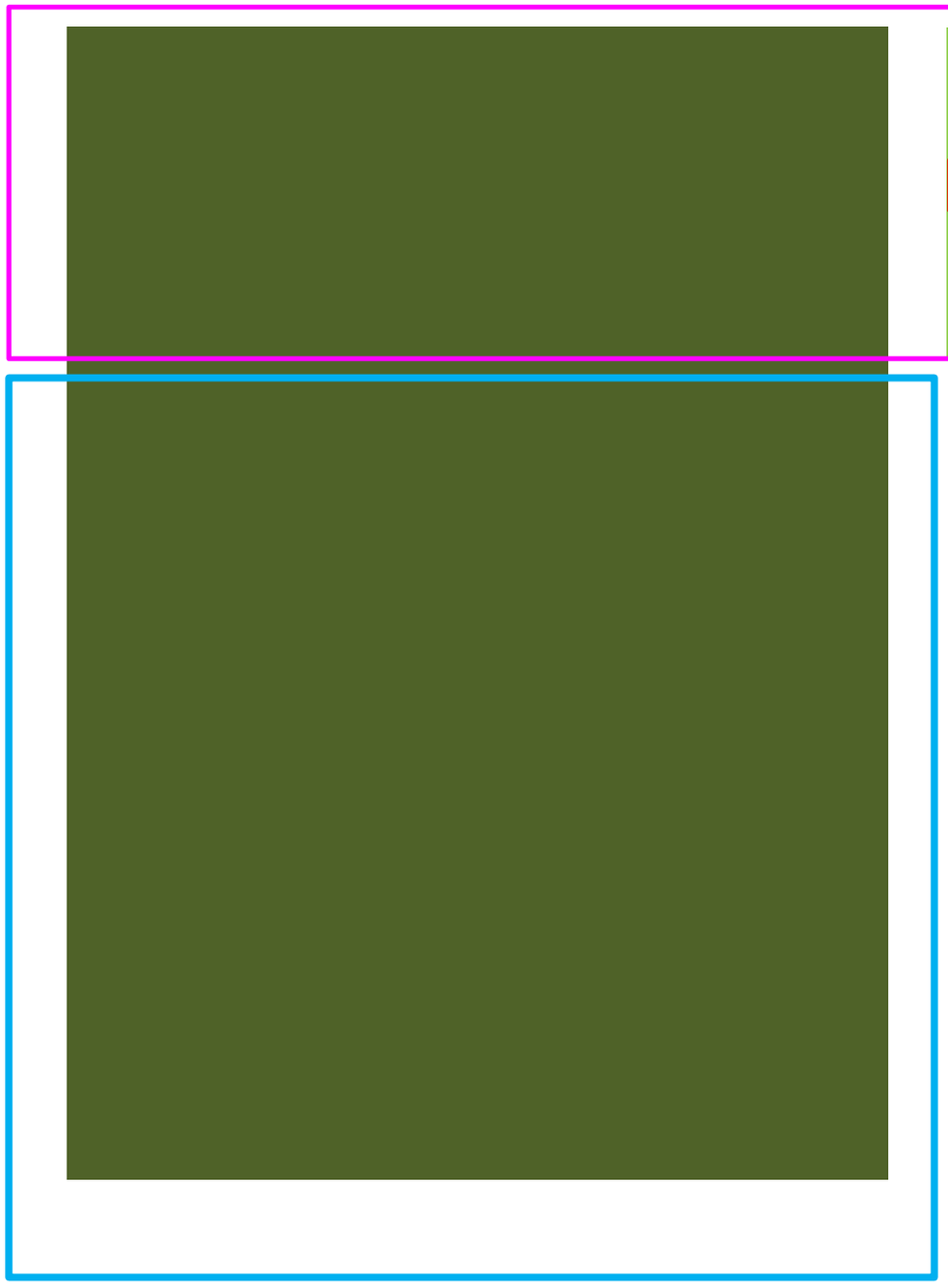
Select the factors that really matter!



Angela 

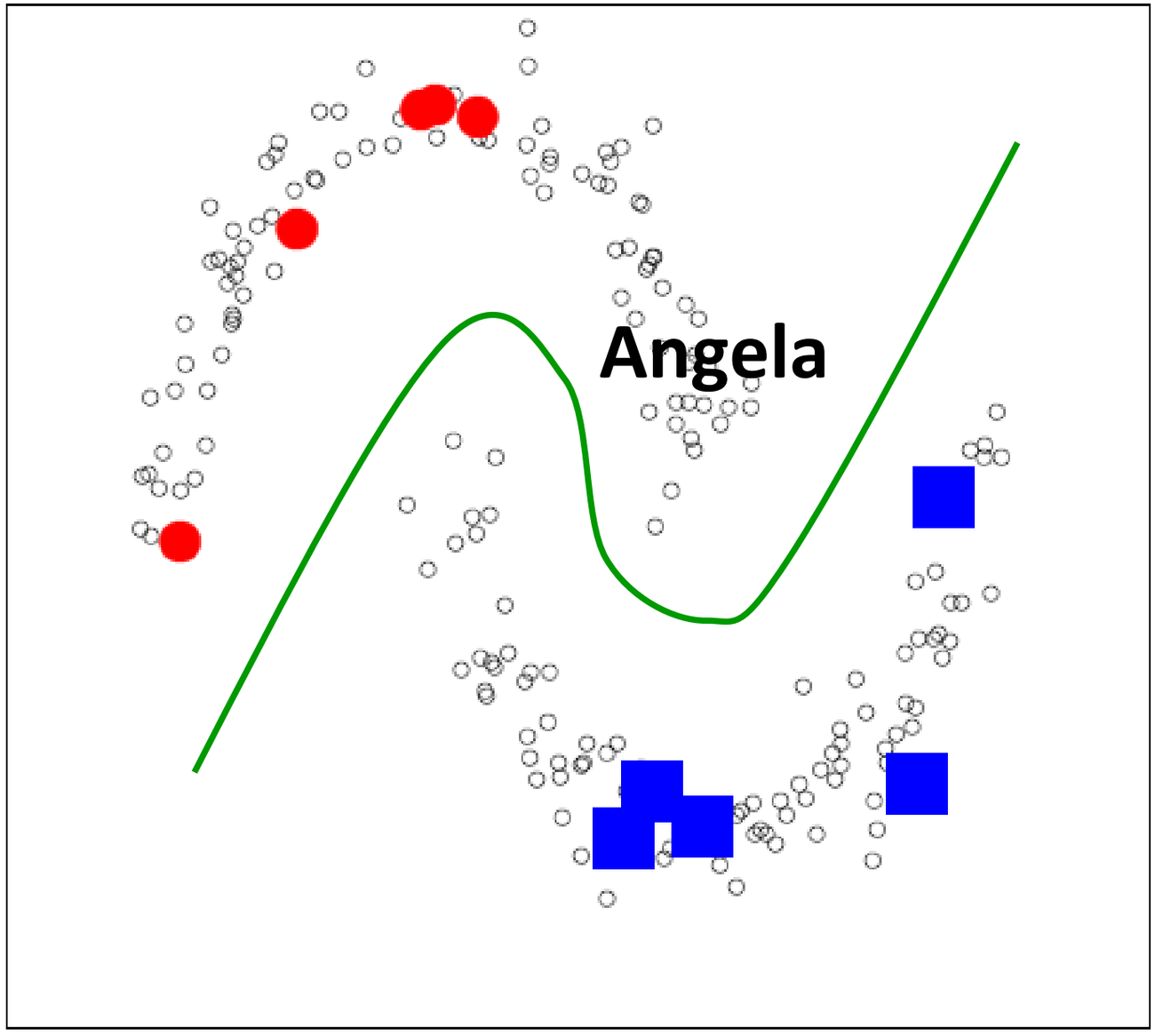
Classification ( ) = Y

Therapy works ?



No outcome known:
useless to design the rule

We can use all the patients, also the ones not treated, to estimate the decision rule much better.



Use all data!

JAMA The Journal of the American Medical Association

VIEWPOINT

Questions for Artificial Intelligence in Health Care

Thomas M. Maddox, MD, MSc
Healthcare Innovation Lab, BJC Healthcare/ Washington University School of Medicine, St Louis, Missouri; and Division of Cardiology, Washington University School of Medicine.

Artificial intelligence (AI) is gaining high visibility in the realm of health care innovation. Broadly defined, AI is a field of computer science that aims to mimic human intelligence with computer systems.[1] This mimicry is accomplished through iterative, complex pattern matching, generally at a speed and scale that exceed human capability. Proponents suggest, often enthusiastically, that AI will revolutionize health care for

What Are the Right Data for AI?

AI is most likely to succeed when used with high-quality data sources on which to "learn" and classify data in relation to outcomes. However, most clinical data, whether from electronic health records (EHRs) or medical billing claims, remain ill-defined and largely insufficient for effective exploitation by AI techniques. For example, EHR data on demographics, clinical condi-

Published online December 10, 2018

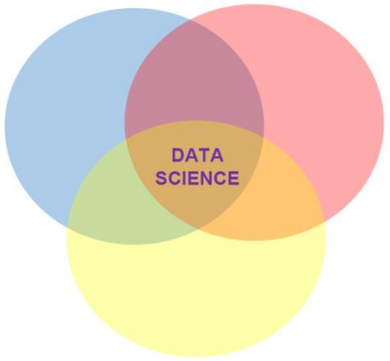
Answers

- Deep learning can exploit subtle and complex associations that are not visible otherwise (say a series of mutations appearing together)
- Image-rich specialities: Radiology, pathology, ophtalmology, cardiolgy.
- For some diseases, it is not clear how the training data should be: heart failure

- Uncertainty is necessary, no decision is 100% safe.
- Difficult to accept a classification which we cannot understand and discuss. (lack of explainability: why will the therapy fail?)

- How to assess evidence of efficacy? RCT with AI arm vs non-AI arm.

- Legal issues related to responsibility when failing.



3. Data science for precision medicine
4. Data science for medical care

The New York Times

Sept. 11, 2018

Are We Being Misled About Precision Medicine?

By Liz Szabo

- Slow effects
- Not enough personalised
- Diseases are polygenic: difficult to invent one drug that attacks, repairs all
- Mostly genetic risk signatures for prevention and prognoses.
- Very costly: towards a more unequal health system.

Vinay Prasad

@VPplenarysesh

The skeptic: What precision medicine revolution?

The benefits of genomic drugs are exaggerated, hurting patients and the practice of medicine, says one high-profile oncologist.

By Stephen S. Hall
Portraits by John Clark

- «Promises of personalised medicine have largely not materialised
- Impact of personalised medicine has been exaggerated
- No evidence (yet) that precision oncology is taking off.
- (as expected, breakthroughs are rare!)»

Research

September 11, 2017.

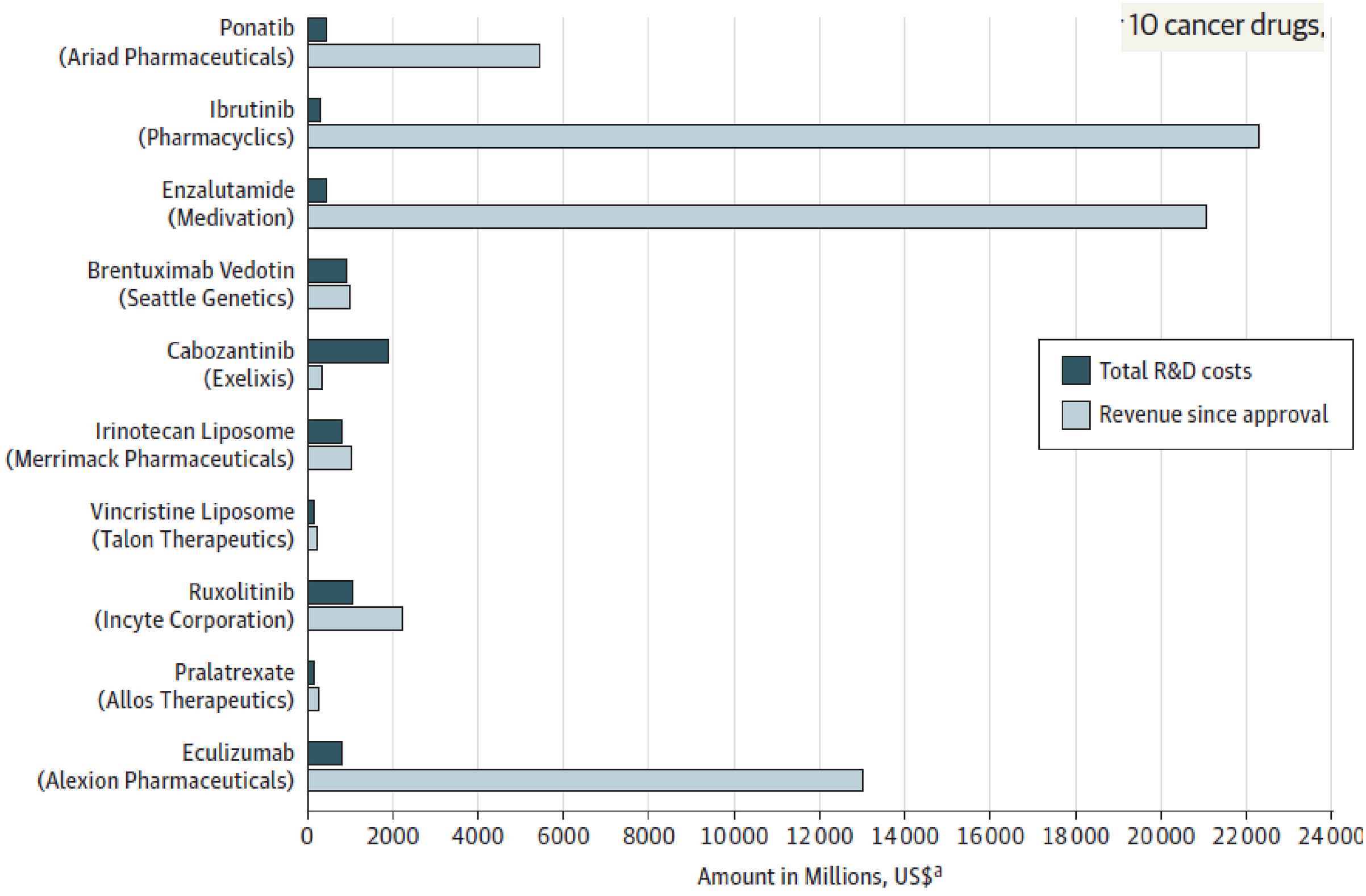
JAMA Internal Medicine | Original Investigation

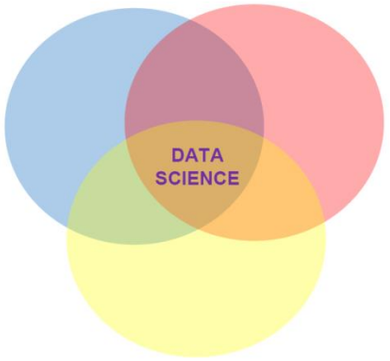
Research and Development Spending to Bring a Single Cancer Drug to Market and Revenues After Approval

Vinay Prasad, MD, MPH; Sham Mailankody, MBBS

- Estimate R&D cost to pring a drug to market:
  800 million USD
- Industry's own figure: **2600** million USD.

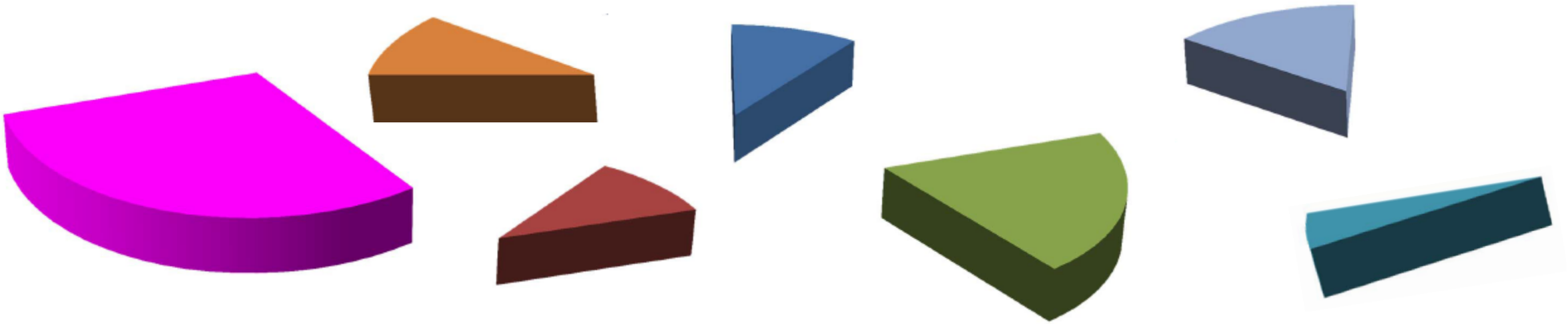
Figure. Comparison of Drug Development Costs With Revenue Earned After Approval



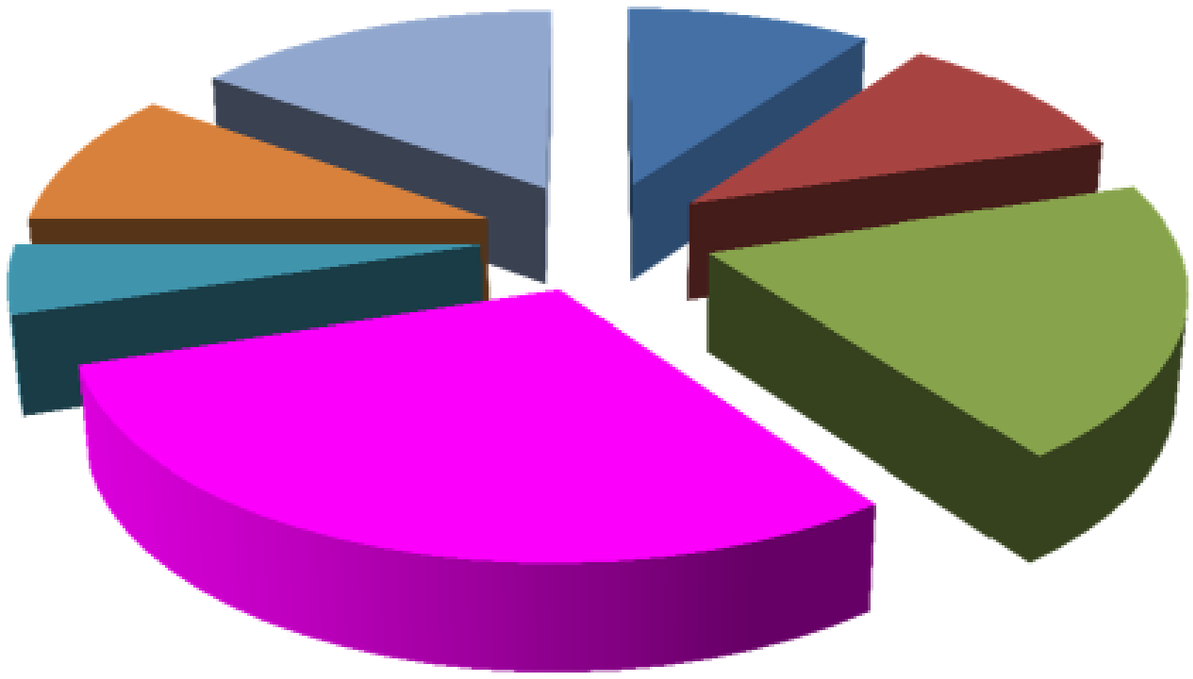


Personalised cancer therapy

Breast cancer is many diseases.



Oncologist
Radiologist
Molecular biologist
Biostatistician
Bioinformatician
Pathologist



Randomised clinical trial

vs.

CURRENT BEST DRUG

- Significantly better
- BUT efficient only for 35% of patients
- Looking for a genomic biomarker of the cancer

Randomised clinical trial

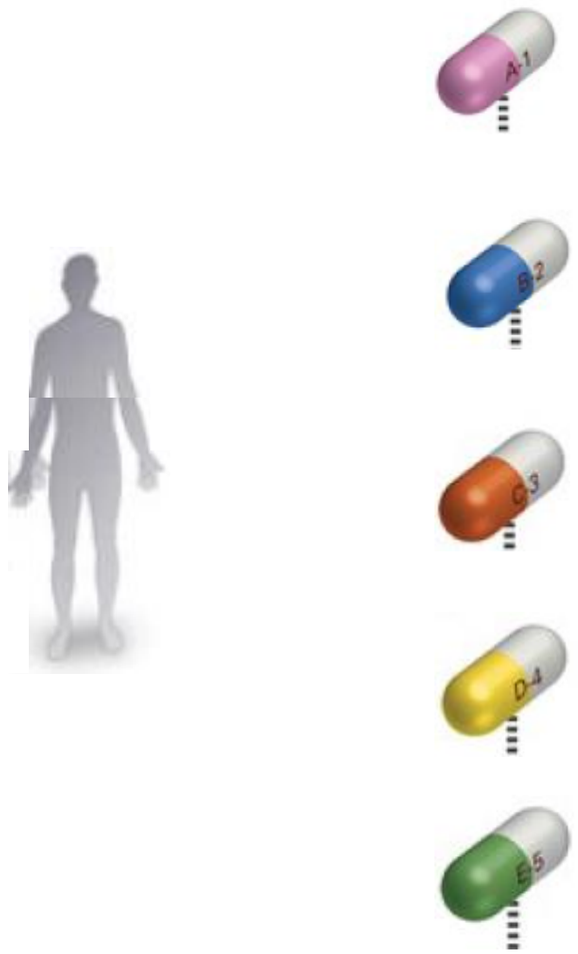
vs.

CURRENT BEST DRUG

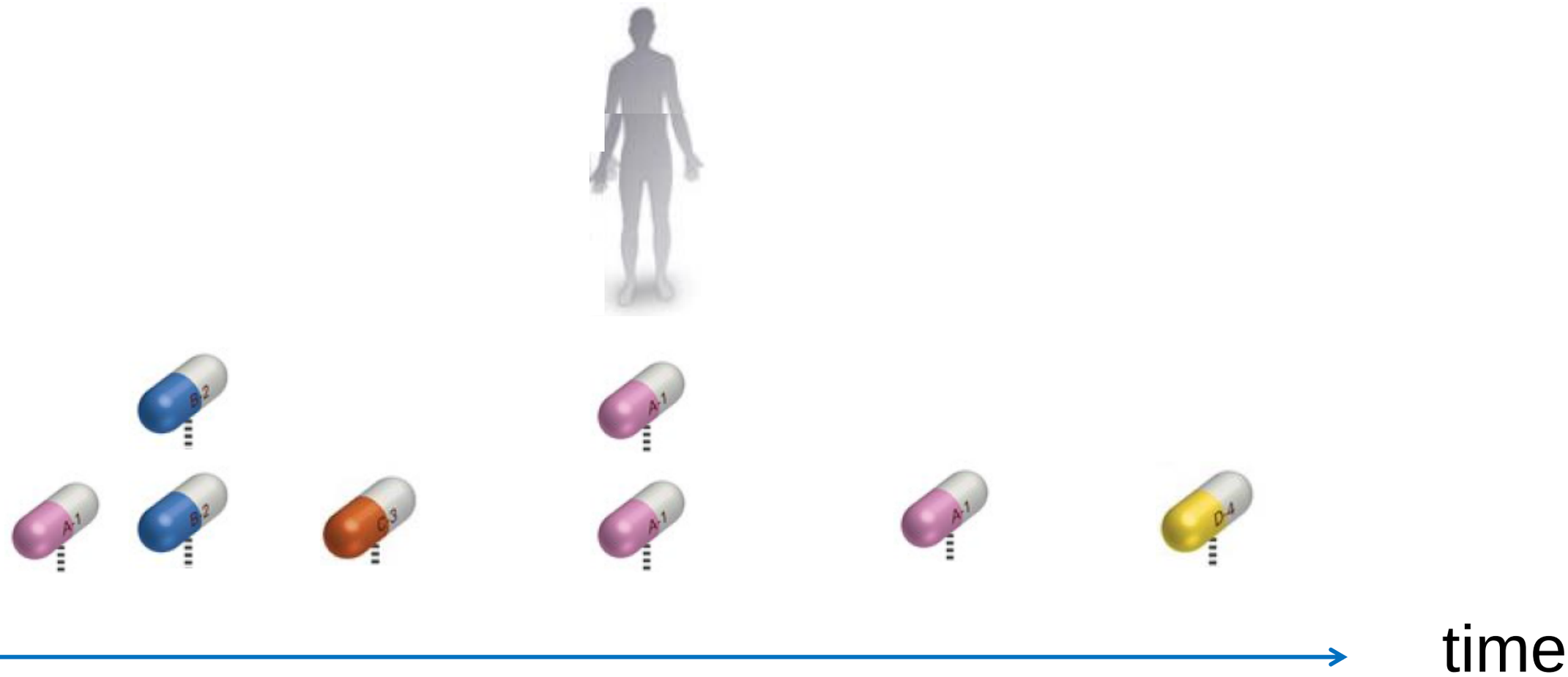
RCT more and more difficult

1 to n

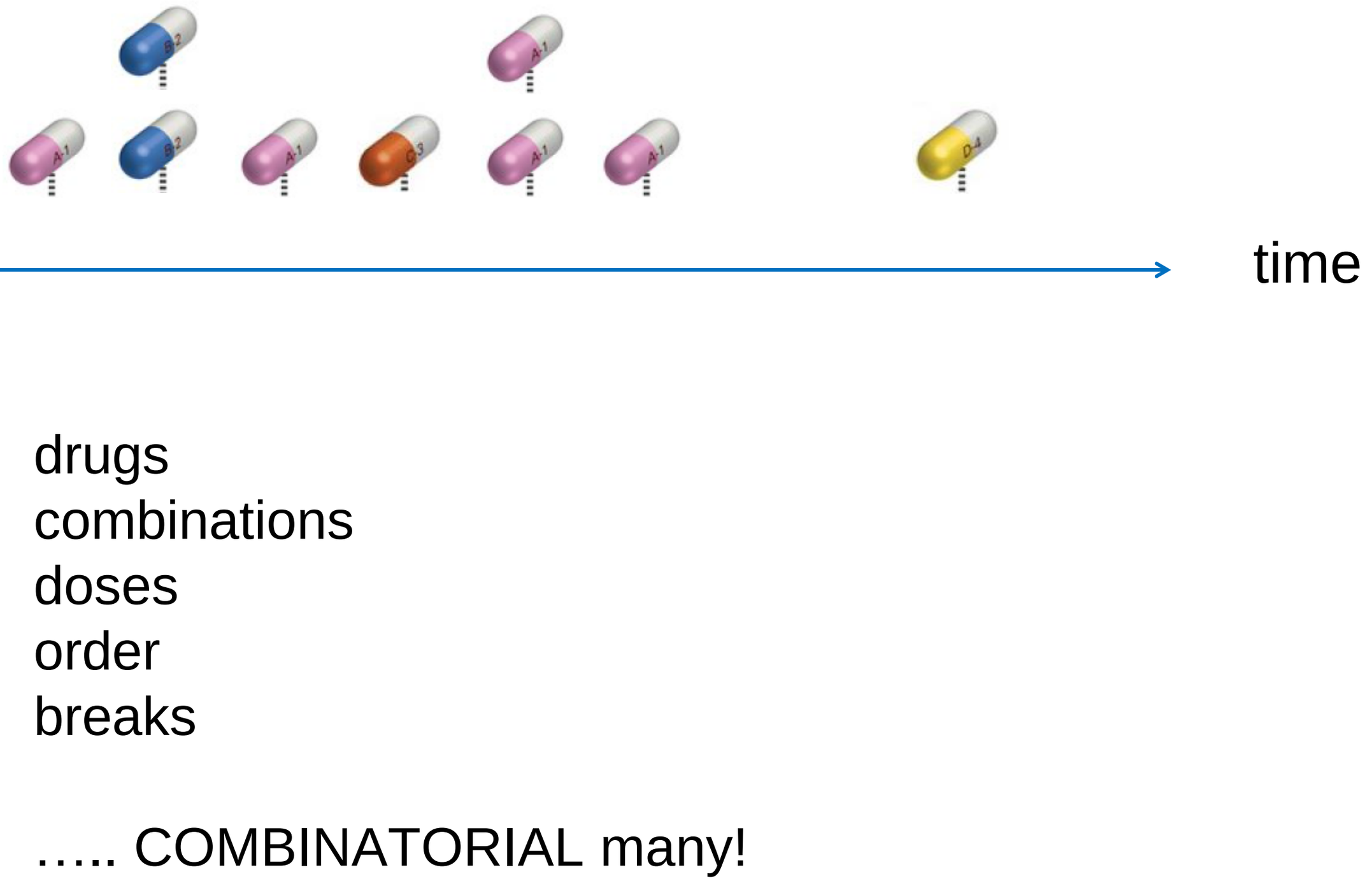
Personalised cancer therapy
Assigning the best therapy to each patient



There are maaaaaany options!



There are maaaaaany options!

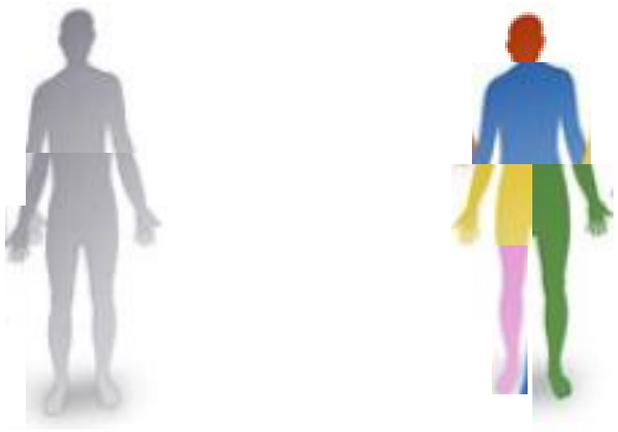


- drugs
- combinations
- doses
- order
- breaks

- ….. COMBINATORIAL many!

Strategy 1 (despite all…) exploit similarities

Strategy 1 (despite all…) exploit similarities



- Find who patient is a similar with, at least in part.
- Merge this information
- Which therapies worked?

IBM Watson for Genomics

A Quick Study

Watson can read 40 million documents in 15 seconds.

- Systematic search in “all” data bases and literature to find “everything” known about similar cases and therapies.
- Text mining, cognitive natural language processing

“ 230 healthcare organizations worldwide use Watson technology “

Cognitive Computing, Paired with Genomics, May Benefit Cancer Treatment

Cancer March 1, 2018

Cognitive computing has the ability to scour large volumes of data from scientific studies and databases to identify potentially relevant clinical trials or therapies for patients with cancer based on the genetics of their tumors, according to a recently published study.[1]

Researchers from the UNC Lineberger Comprehensive Cancer Center at the University of North Carolina at Chapel Hill say their findings, which are preliminary, could help physicians stay up to date regarding expanding amounts of available scientific literature and information concerning cancer genetics. The study's first authors, Nirali Patel, MD, of UNC Lineberger Comprehensive Cancer Center, and Vanessa Michelini , BS of IBM Watson Health in Boca Raton, Florida, used IBM Watson for Genomics cognitive computing to assess whether the system was more effective than a panel of cancer experts in identifying therapeutic options for patients with tumors with specific genetic abnormalities.

In a retrospective analysis of 1018 cancer cases, the UNC Lineberger molecular tumor board of cancer experts identified actionable genetic alterations in 703 cases. Concurrently, Watson for Genomics identified additional potential therapeutic options in 323 patients, or approximately one-third of the cases reviewed, that the molecular tumor board had not identified. Of the 323 cases of Watson-identified actionable alterations, only 8 genes had not been considered actionable by the molecular tumor board, according to William Kim, MD, the study's corresponding author and an associate professor of medicine and genetics at the University of North Carolina at Chapel Hill School of Medicine.

In the majority of those additional cases, IBM Watson identified a new clinical trial, one of which opened within 1 week of the Watson analysis.

The study's main finding was that cognitive computing augmented the tumor board's process for the interpretation and collection of information regarding a patient's genomic profile, Dr. Kim notes. He adds that the study was not designed to determine whether cognitive computing actually improves patient outcomes in terms of survival or response to treatment.

Although the findings were not relevant for the majority of patients in the study, either because they did not have active cancer or had died by the time of the analysis, they did provide additional options for 47 patients with active disease, the authors say.

Reference

1. Patel NM, Michelini VV, Snell JM, et al. Enhancing next-generation sequencing-guided cancer care through cognitive computing [published online ahead of print November 20, 2017]. *Oncologist*. doi:10.1634/theoncologist.2017-0170.

DOI: 10.1002/cncr.31276

THE WALL STREET JOURNAL.

IBM Has a Watson Dilemma

Big Blue promised its AI platform would be a big step forward in treating cancer. But after pouring billions into the project, the diagnosis is gloomy.

By Daniela Hernandez and Ted Greenwald

Aug. 11, 2018 12:19 a.m. ET

Can Watson cure cancer?

The Boston Globe

A STAT INVESTIGATION

IBM pitched its Watson supercomputer as a revolution in cancer care. It's nowhere close

By CASEY ROSS @caseymross and IKE SWETLITZ @ikeswetlitz / SEPTEMBER 5, 2017

KUNSTIG INTELLIGENS

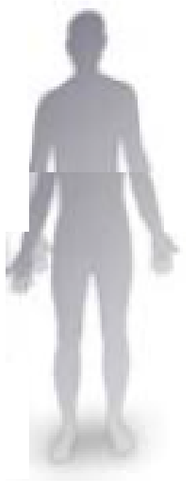
Det danske rikshospitalet stopper IBM Watson: Foreslo livsfarlig medisin

Flere kreftleger mener at IBMs kunstige intelligens, Watson Oncology, er altfor umoden. Et dansk forsøk på Rigshospitalet ble stoppet etter enorm feilrate.

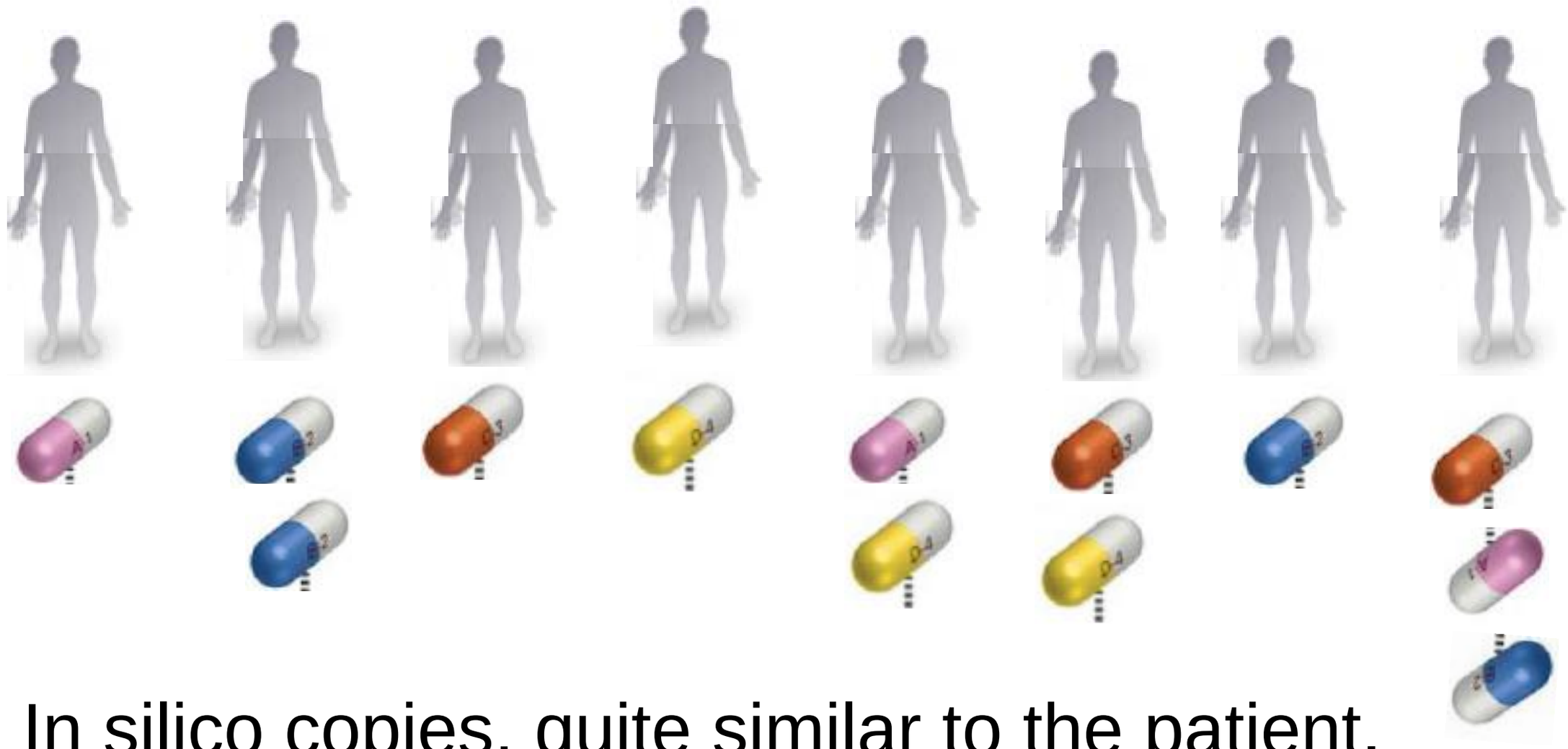
AV THOMAS DJURSING, ING.DK | HELSE-IT | PUBLISERT: 24. OKT. 2017 - 07:56

digi.no

Strategy 2 Copies of the patient



Strategy 2 Copies of the patient



- In silico copies, quite similar to the patient.
- Simulate therapies.
- Which worked?

Towards personalized computer simulation of breast cancer treatment: a multi-scale pharmacokinetic and pharmacodynamic model informed by multi-type patient data

Xiaoran Lai[1], Oliver M Geier[2], Thomas Fleischer[3], Øystein Garred[4], Elin Borgen[4], Simon Wolfgang Funke[5], Surendra Kumar[3], Marie Elisabeth Rognes[5], Therese Seierstad[6], Anne-Lise Børresen-Dale[3], Vessela N. Kristensen[3,7,8], Olav Engebraaten[9,10], Alvaro Köhn-Luque[1,*] and Arnoldo Frigessi[1,11,*]

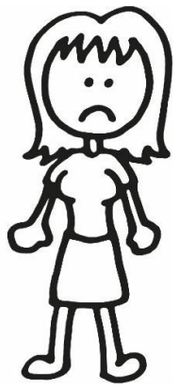
Revision Cancer Research 2018

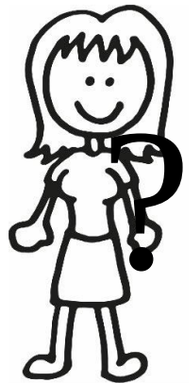
- Model based
- Personalised
- Based on computer simulation of therapy effect.

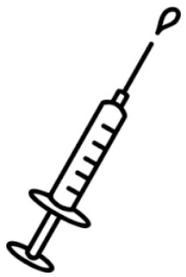
- UiO Life Science Convergence Environment PerCaThe
- Digital Life Norway NFR

Personalised computer simulation

cancer patient







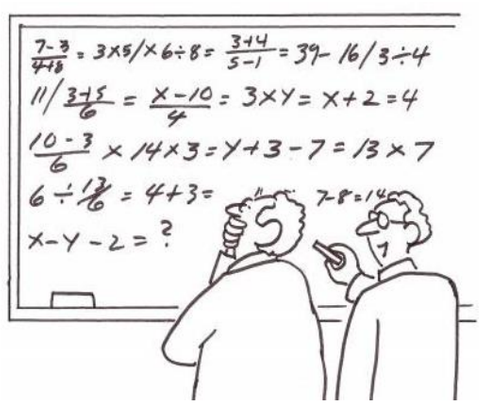
personal clinical data

MRI histology molecular

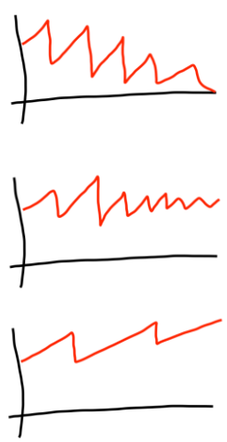
optimal treatment plan

oncologist

multi-scale mathematical model of cancer growth



personalised computer simulations



strategy 3

strategy 2

strategy 1

THREE DRUGS COMBINED:

- FLUOROURACIL
- EPIRUBICIN
- CYCLOPHOSPHAMIDE.

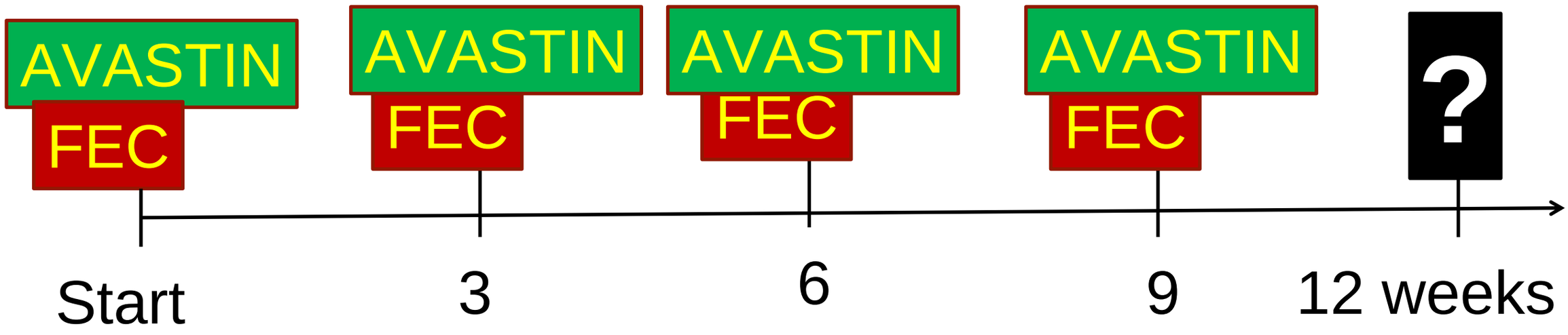
KILLS CELLS WHEN THEY DOUBLE

BEVACIZUMAB BLOCKS ANGIOGENESIS BY STOPPING VEGF

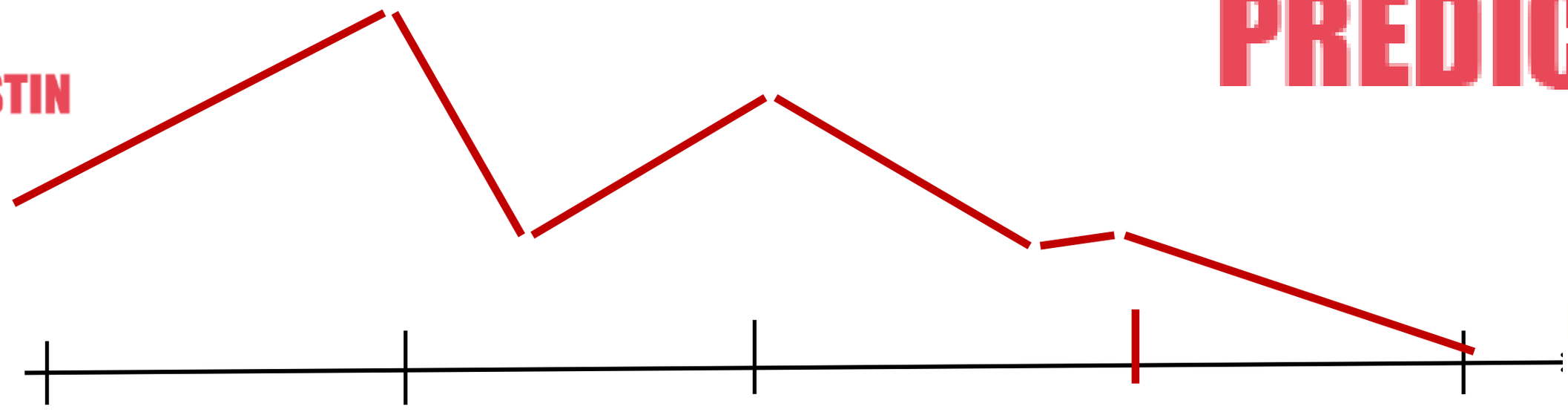
NO BLOOD = NO OXIGEN = DEATH

AVASTIN

FEC


AVASTIN
FEC
AVASTIN
FEC
AVASTIN
FEC
AVASTIN
FEC
?
Start
3
6
9
12 weeks

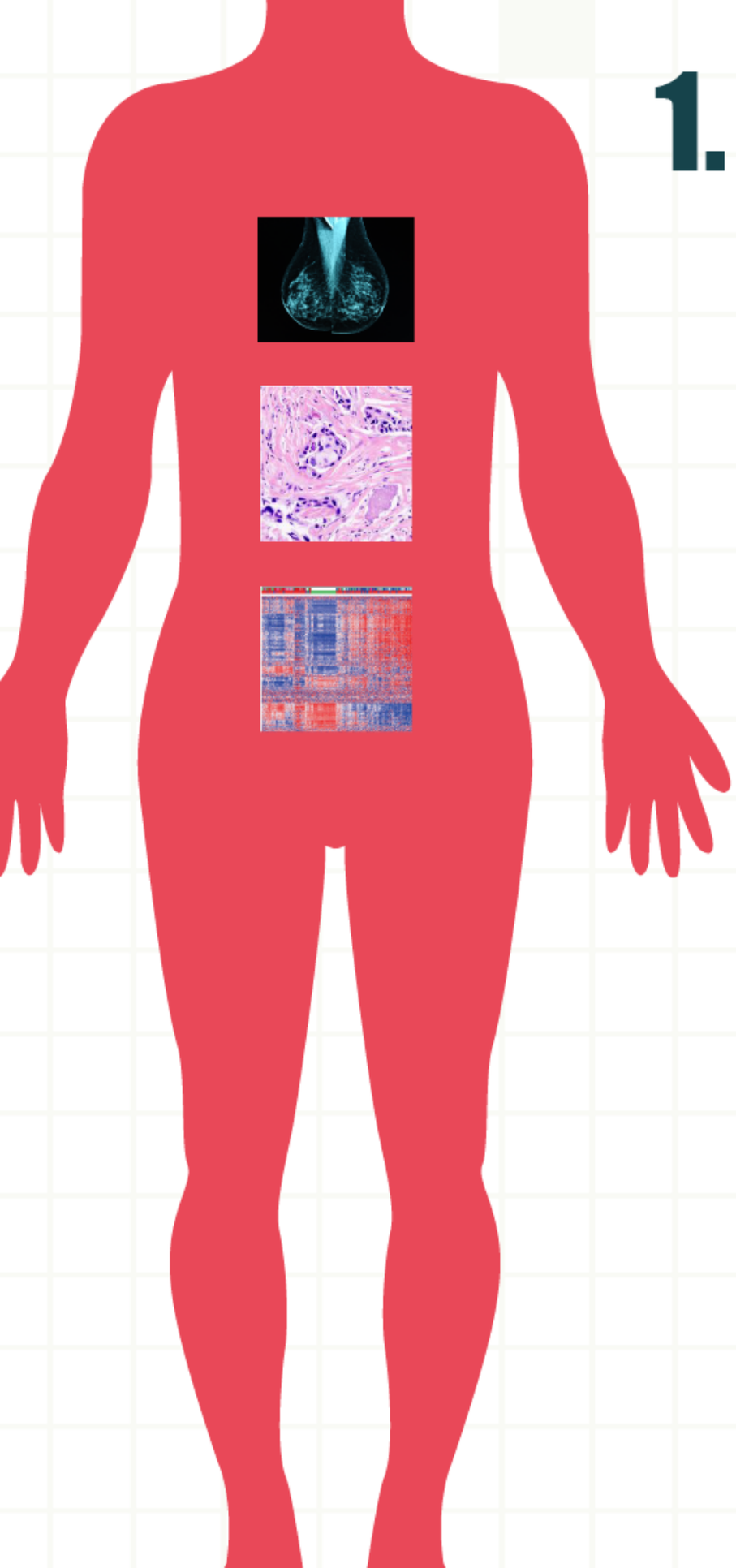
PREDICT



Start 3 6 9 12 weeks

Start 3 6 9 12 weeks

1. PERSONAL CLINICAL DATA



Patient Biopsy
H&E staining

1.3cm
approx.

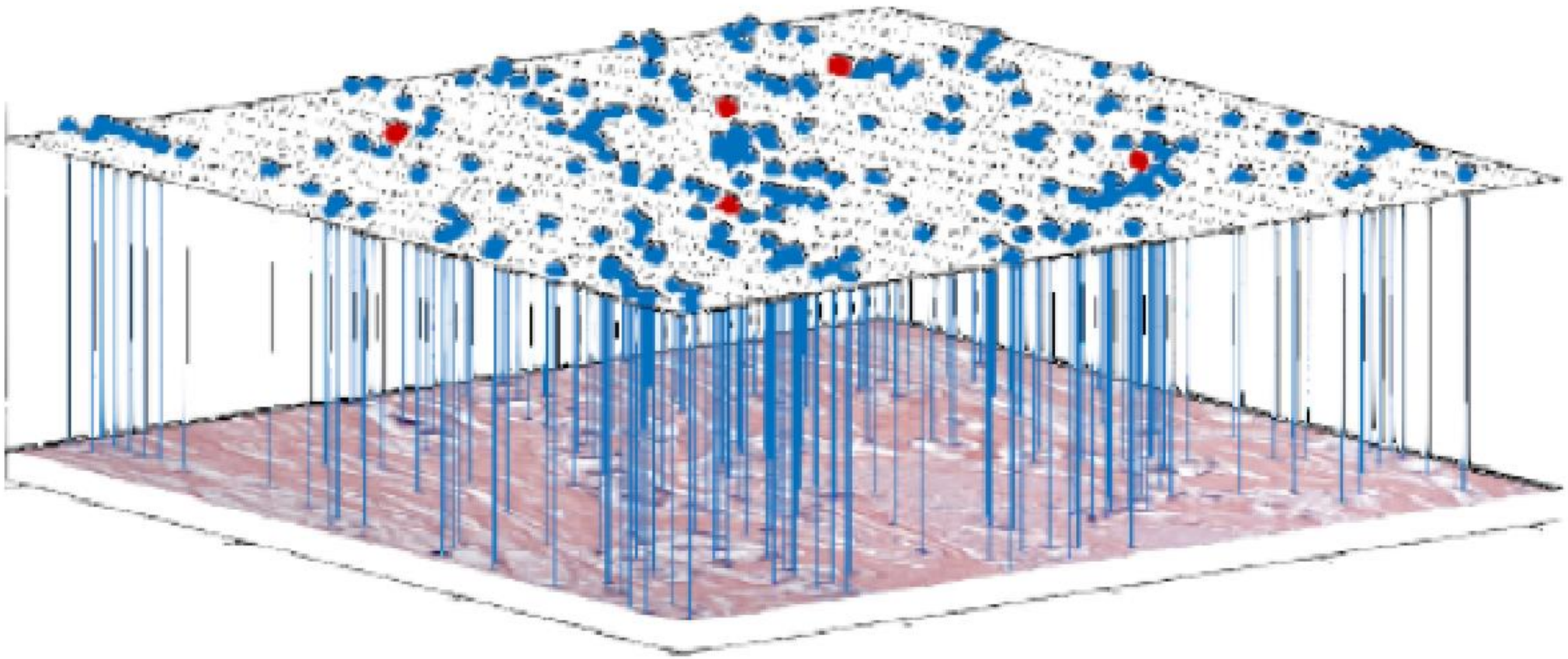
0.2 mm

0.3 mm

20 x 30
numerical grid

approx. 15 min
CPU time

COMPUTATIONAL GRID

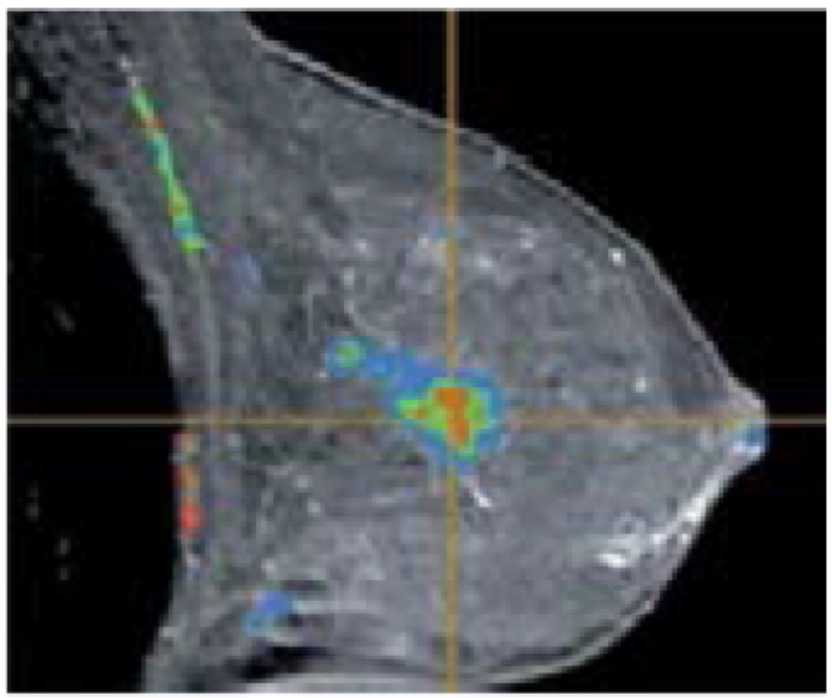


Normal cells

Cancer cells

Blood vessels

MR IMAGES

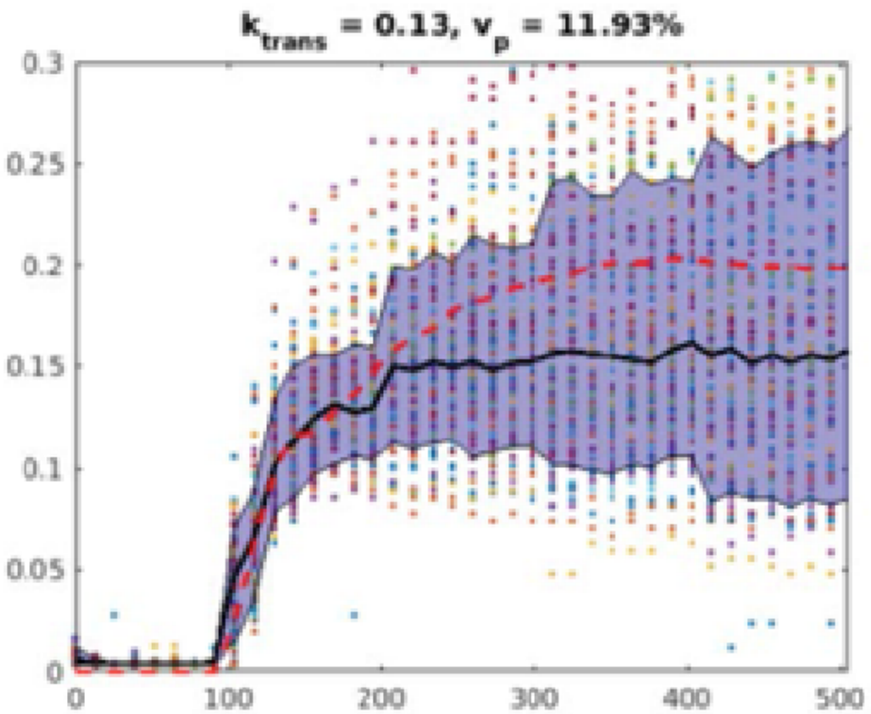


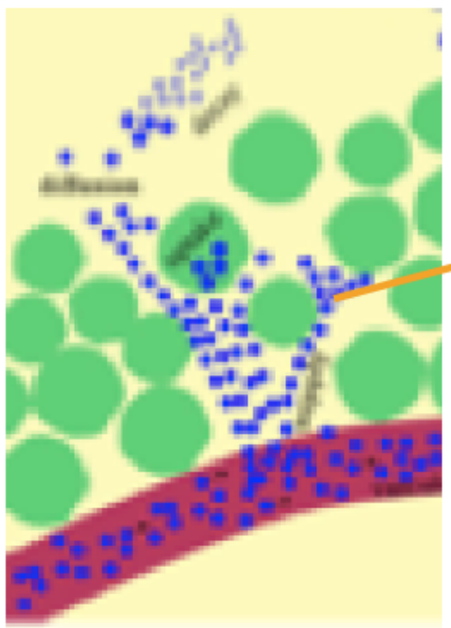
-Permeability of blood vessels unknown.

-Estimated with help of MRI: we see when the contrast agent diffuses in the tumor

-MRI data resolution: $1mm^3$ tumor voxel.

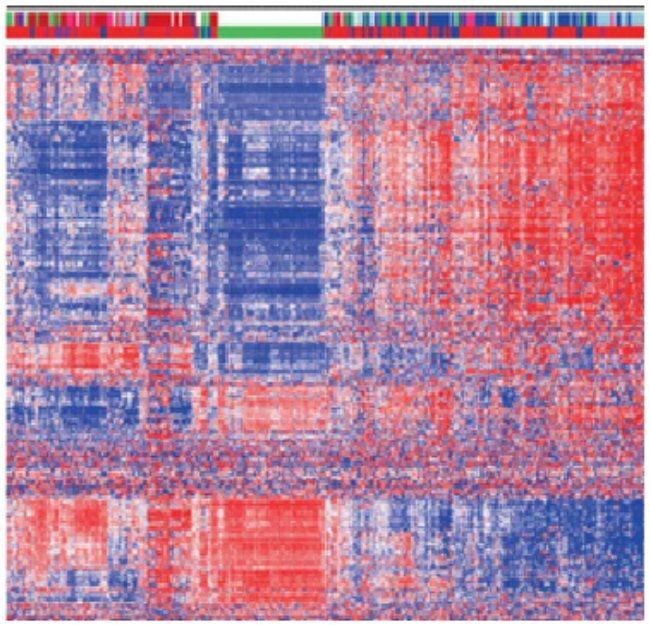
-Use this to estimate permeability at the smaller scale ($0.001mm^3$)





OXYGEN DRUGS

GENOMICS



A genetic portrait
of the tumor

Gene VEGF: induces growth and migration of blood vessels (angiogenesis). Blood vessels grow where there is a lot of VEGF

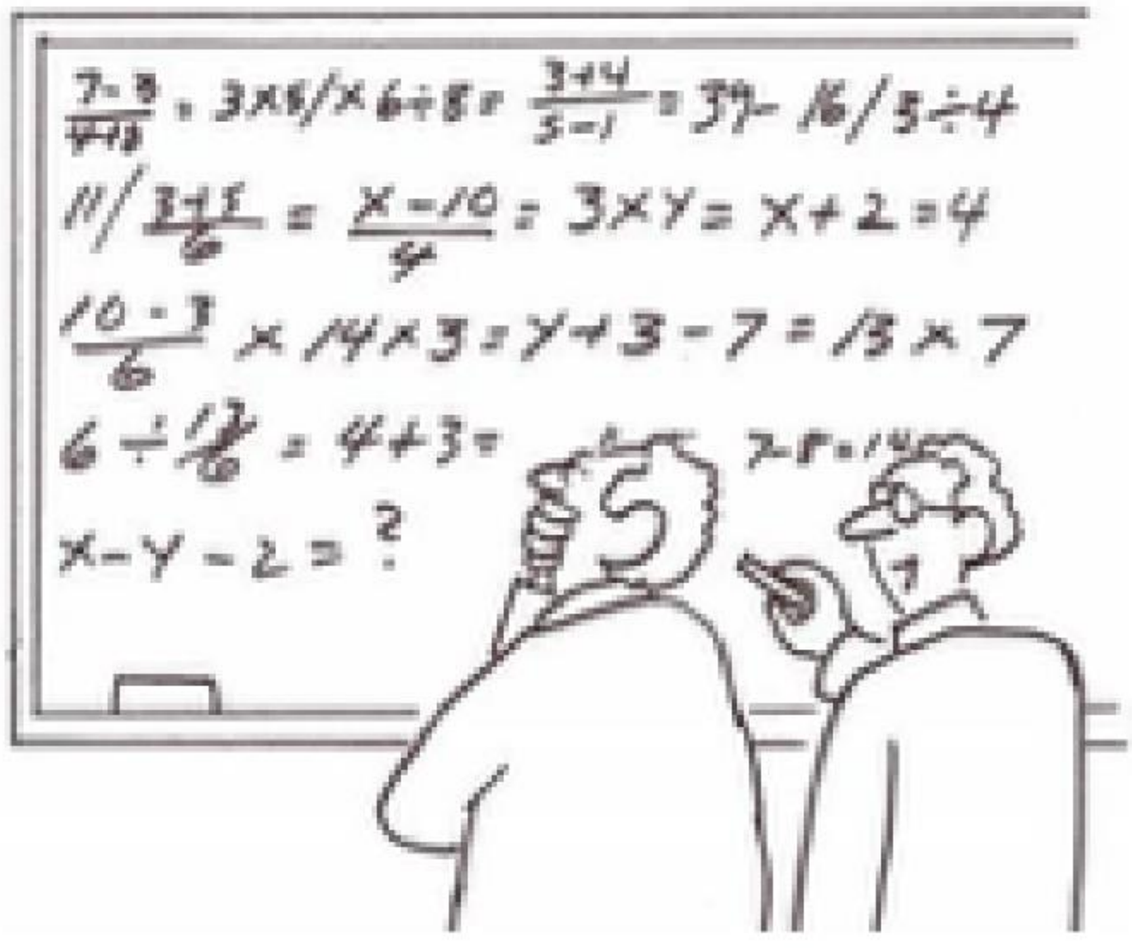
Gene TP53

Mutated TP53: VEGF production is increased.

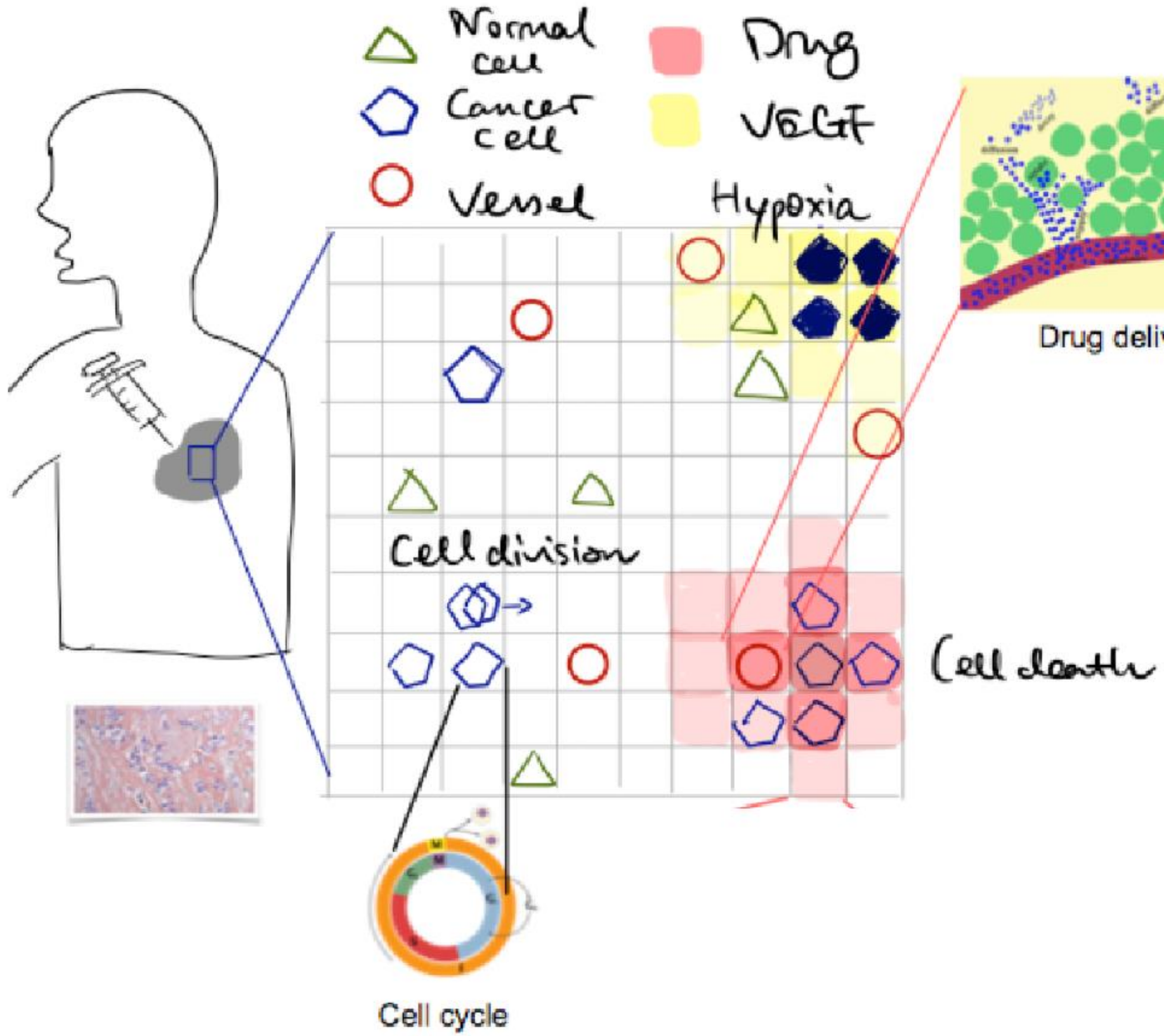
Non mutated TP53: VEGF production reduced

Oxygen, TP53 and VEGF are coupled. Hypoxia upregulates the production of TP53 and VEGF in cancer cells

2. MULTISCALE MATHEMATCAL MODEL OF CANCER GROWTH



MODELLING BIOLOGICAL MECHANISMS



$$\frac{d[\mathrm{TP53}]_{\boldsymbol{x}}}{dt} = k_7 - k_7' \frac{K(\boldsymbol{x},t)}{K_{\mathrm{TP53}} + K(\boldsymbol{x},t)} [\mathrm{TP53}]_{\boldsymbol{x}},$$

$$\frac{d[\mathrm{sVEGF}]_{\boldsymbol{x}}}{dt} = k_8 - k_8' \frac{K(\boldsymbol{x},t)}{K_{\mathrm{VEGF}} + K(\boldsymbol{x},t)} [\mathrm{sVEGF}]_{\boldsymbol{x}} + k_8'' \frac{[\mathrm{TP53}]_{\boldsymbol{x}}[\mathrm{sVEGF}]_{\boldsymbol{x}}}{J_5 + [\mathrm{sVEGF}]_{\boldsymbol{x}}}$$

$$s_K \frac{\partial K}{dt} - D_k \nabla^2 K = r_k (K_0 - K)\mathcal{G}(\boldsymbol{x},t) - \frac{\phi_k K}{K_1 + K}\delta(\boldsymbol{x},t)$$

$$s_V \cdot \frac{\partial V}{dt} - D_v \nabla^2 V = r_v([\mathrm{sVEGF}]_{\boldsymbol{x}})\delta(\boldsymbol{x},t) - k_d C - k_a A_e V - \psi_v V$$

$$s_A \cdot \frac{\partial A}{dt} - D_A \nabla^2 A = r_A (A_1(t) - A)\mathcal{G}(\boldsymbol{x},t) - k_d C - k_a A V$$

$$\frac{\partial G^j}{dt} - D_{G^j} \nabla^2 G^j = r_{G^j}(G_1^j(t) - G^j)\mathcal{G}(\boldsymbol{x},t) - \psi_{G^j} G^j$$

$$\frac{dG_1^2(t)}{dt} = -\frac{q_2}{w_1} G_2^2(t) - \frac{q_3}{w_1} G_3^2(t) - \frac{cl_2}{w_1} G_1^2(t) + \frac{q_3}{w_3} G_3^2(t) + \frac{q_2}{w_2} G_2^2(t)$$

$$\frac{dG_2^2(t)}{dt} = -\frac{q_2}{w_2} G_2^2(t) + \frac{q_2}{w_1} G_1^2(t)$$

$$\frac{dG_3^2(t)}{dt} = -\frac{q_3}{w_3} G_3^2(t) + \frac{q_3}{w_1} G_1^2(t)$$

$$\frac{dA_1(t)}{dt} = -\frac{q}{v_1} A_1(t) - \frac{cl}{v_1} A_1(t) + \frac{q}{v_2} A_2(t)$$

$$\frac{dA_2(t)}{dt} = -\frac{q}{v_2} A_2(t) + \frac{q}{v_1} A_1(t),$$

Oxygen $K(x,t)$

$$\frac{\partial K}{dt} - D_k \nabla^2 K = r_k(K_0 - K)\mathcal{G}(\boldsymbol{x},t) - \frac{\phi_k K}{K_1 + K}\delta(\boldsymbol{x},t)$$

diffusion

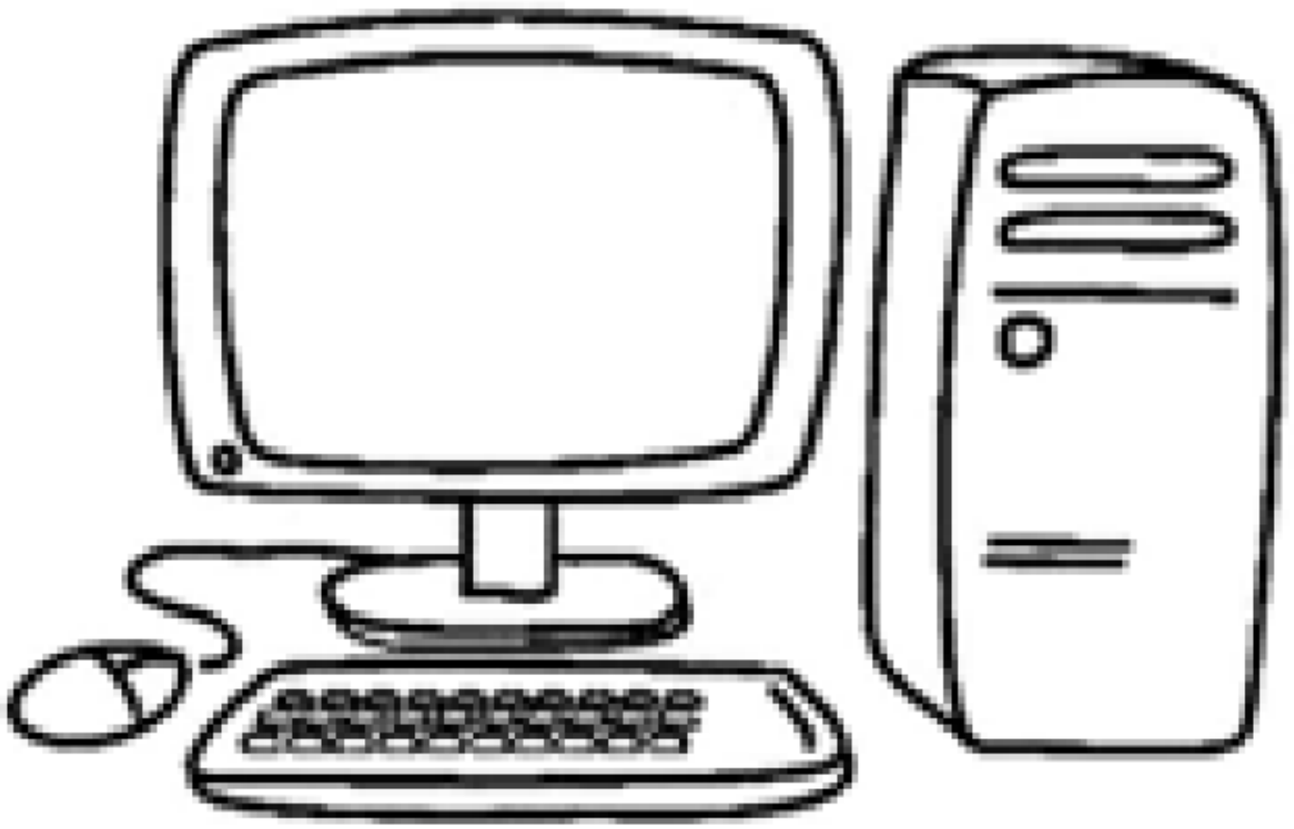
supply from vessels

consumption by cells

Parameter	Description	Value	Units
k_ϕ	Oxygen concentration at half-maximal cycle speed	1.4	mmHg
k_7	Degration rate of p53 by oxygen	0.01	min^{-1}
K_{TP53}	Oxygen concentration for half-maximal TP53 degredation	0.01	mmHg
k_8	Synthesis rate of VEGF	0.002	min^{-1}
k_8'	Reaction rate of p53 with VEGF	0.01	min^{-1}
J_5	sVEGF concentration for half-maximal sVEGF production	0.04	$\mu\text{g mL}^{-1}$
K_{VEGF}	Oxygen concentration for half-maximal VEGF degredation	0.01	mmHg
D_k	Oxygen diffusion coefficient	1.05×10^5	$\mu\text{m}^2\ \text{min}^{-1}$
r_k	Oxygen supply rate	1.88×10^4	min^{-1}
K_0	Oxygen concentration in the blood	20	mmHg
ϕ_k	Oxygen consumption rate	900	min^{-1}
K_1	Oxygen concentration for half-maximal consumption	2.5	mmHg
D_v	VEGF diffusion coefficient	3.52×10^3	$\mu\text{m}^2\ \text{min}^{-1}$
a	VEGF secretion slope	6.66×10^{-6}	min^{-1}
b	VEGF secretion intercept	-1.10×10^{-6}	$\mu\text{g mL}^{-1}\text{min}^{-1}$
k_a	VEGF association rate to Avastin	7.4×10^{-1}	$\mu\text{g}^{-1}\ \text{mL min}^{-1}$
k_d	VEGF dissotiation rate from Avastin	1.76×10^{-3}	min^{-1}
ψ_v	VEGF decay rate	1.0×10^{-2}	min^{-1}
D_A	Avastin diffusion coefficient	2.4×10^3	$\mu\text{m}^2\ \text{min}^{-1}$
ψ_C	Complex decay rate	1.0×10^{-2}	min^{-1}
D_{Gi}	Chemotherapies diffusion coefficient	9.6×10^3	$\mu\text{m}^2\ \text{min}^{-1}$
ψ_{Gi}	Chemotherapies decay rate	1.0×10^{-2}	min^{-1}
v_1	Avastin plasma compartment volume	2.66×10^3	mL
v_2	Avastin peripheral compartment volume	2.76×10^3	mL
q	Avastin intercompartmental clearance	0.412	mL min^{-1}
cl	Avastin elimination clearance	0.144	mL min^{-1}
v_{max}	Fluororacil maximal degradation rate	1.75	$\mu\text{g mL}^{-1}\ \text{min}^{-1}$
k_m	Fluororacil half-maximal concentration	27	$\mu\text{g mL}^{-1}$
w_1	Epirubicin plasma compartment volume	18×10^3	mL
w_2	Epirubicin peripheral compartment volume	957×10^3	mL
w_3	Epirubicin peripheral compartment volume	25×10^3	mL
q_2	Epirubicin intercompartmental clearance	0.918×10^3	mL min^{-1}
q_3	Epirubicin intercompartmental clearance	0.25×10^3	mL min^{-1}
cl_2	Epirubicin elimination clearance	0.983×10^3	mL min^{-1}
u	Cyclophosphamide plasma compartment volume	2430×10^3	mL
cl_3	Cyclophosphamide elimination clearance	3.93×10^3	mL min^{-1}
Δx	Space interval	10	μm
Δt	Time interval of cell cycle update	30	min
Δv	Vessel update interval	720	min
Low_V	Lower VEGF angiogenic threshold	10^{-6}	$\mu\text{g mL}^{-1}$
α	FEC dose-response shape	1	dimensionless

Parameter	Description (units)	Patient 1		Patient 2		Patient 3		Patient 4	
		Bio A	Bio B	Bio A	Bio B	Bio A	Bio B	Bio A	Bio B
n_c	Initial cancer cells (number)	146	218	386	516	315	333	188	273
n_s	Initial stroma cells (number)	45	72	140	53	149	101	339	148
$T_{\min}$	Minimum cell cycle duration (days)	14.69		3.74		3.74		3.74	
k_7	Basal p53 synthesis rate ($\min^{-1}$)	0.002		0.012		0.0004		0.002	
k_8''	Maximal p53 effect in VEGF production ($\min^{-1}$)	-0.002		-0.0037		-0.0002		0.002	
		Cond A	Cond B	Cond A	Cond B	Cond A	Cond B	Cond A	Cond B
n_v	Initial blood vessels (number)	27	45	27	45	4	35	14	9
r_{C^i}	Chemotherapies permeability ($\min^{-1}$)	8.17	8.17	8.80	8.80	0.42	13.5	0.42	6.96
r_A	Avastin permeability ($\min^{-1}$)	0.08	0.08	0.08	0.08	0.0042	0.135	0.0042	0.07
d_{C^1}	Fluorouracil dose (mg m^{-2})	600		600		600		600	
d_{C^1}	Epirubicin dose (mg m^{-2})	100		100		100		100	
d_{C^3}	Cyclophosphamide dose (mg m^{-2})	600		600		600		600	
d_A	Bevacizumab dose (mg kg^{-1})	15		15		0		0	

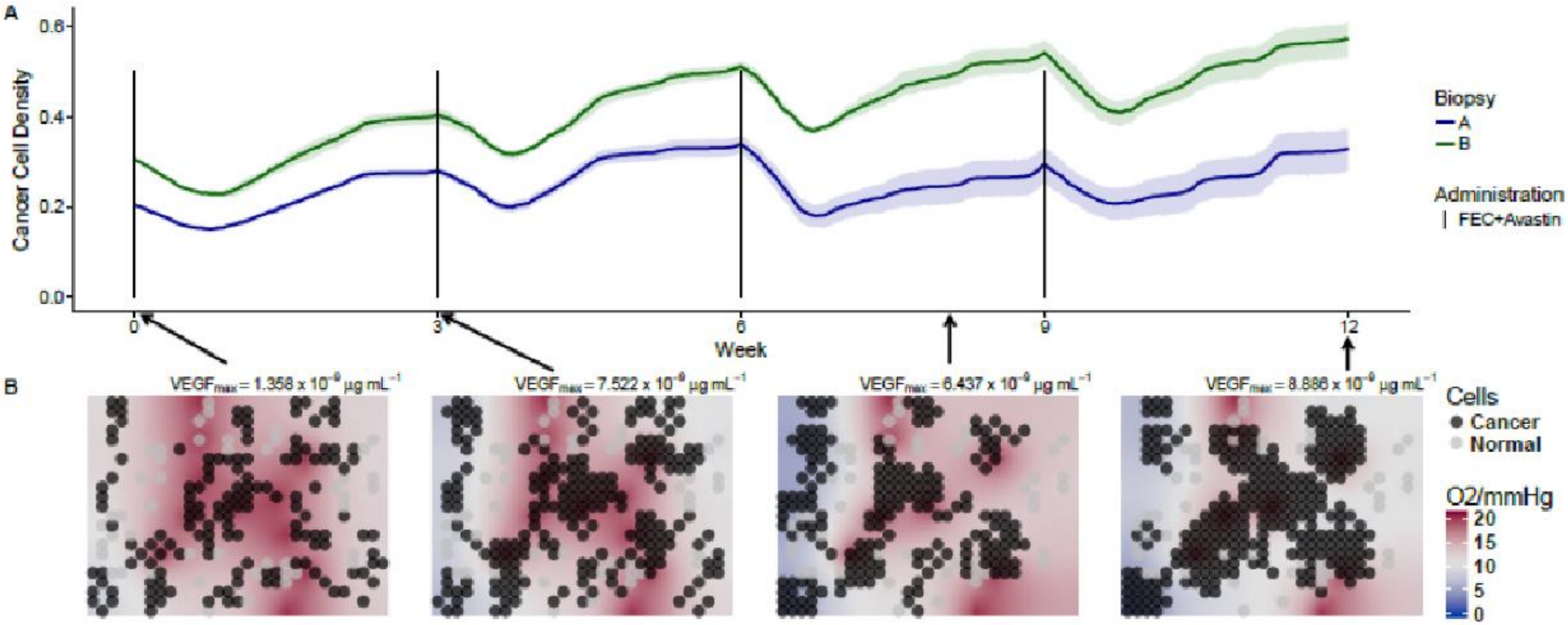
3. PERSONALIZED COMPUTER SIMULATION



Patient 1

-FEC and Avastin
-Truth: Non-responder, same tumour size
-Simulation

Uncertainty

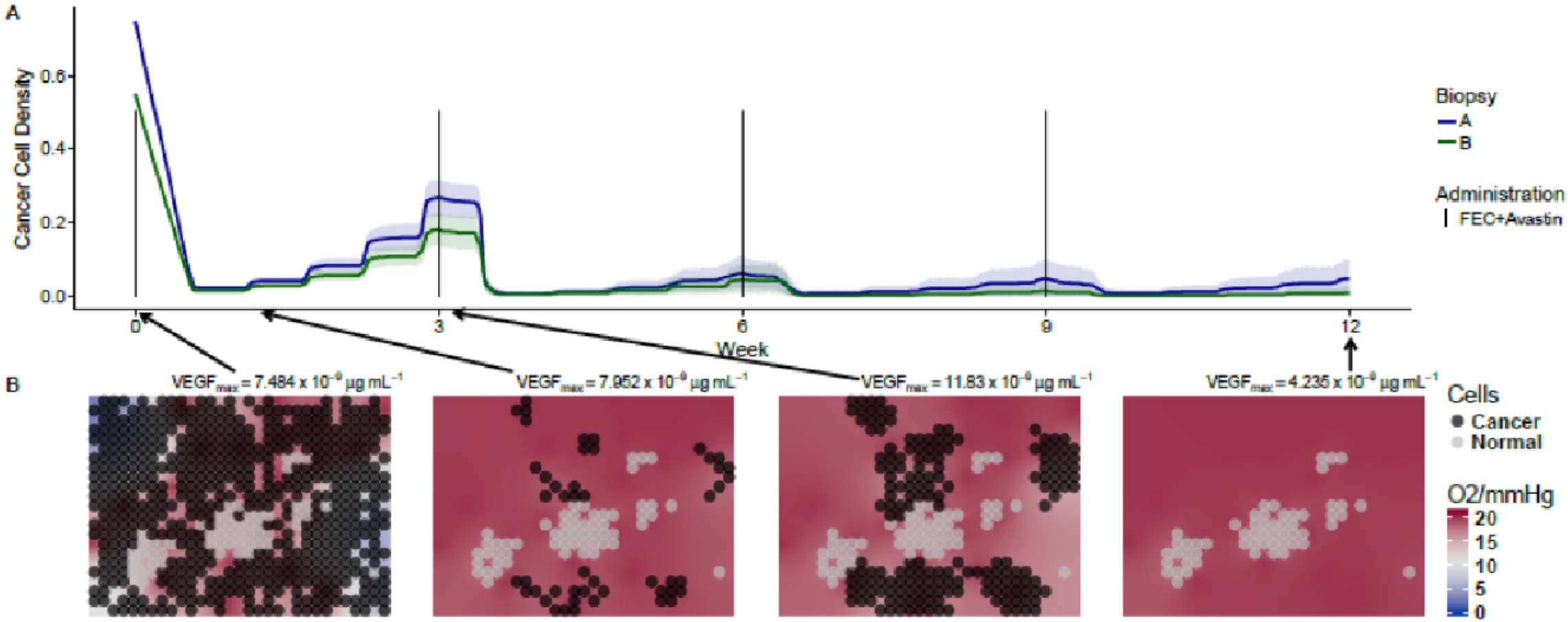


Patient 2

-FEC and Avastin

-Truth: Responder, tumour away

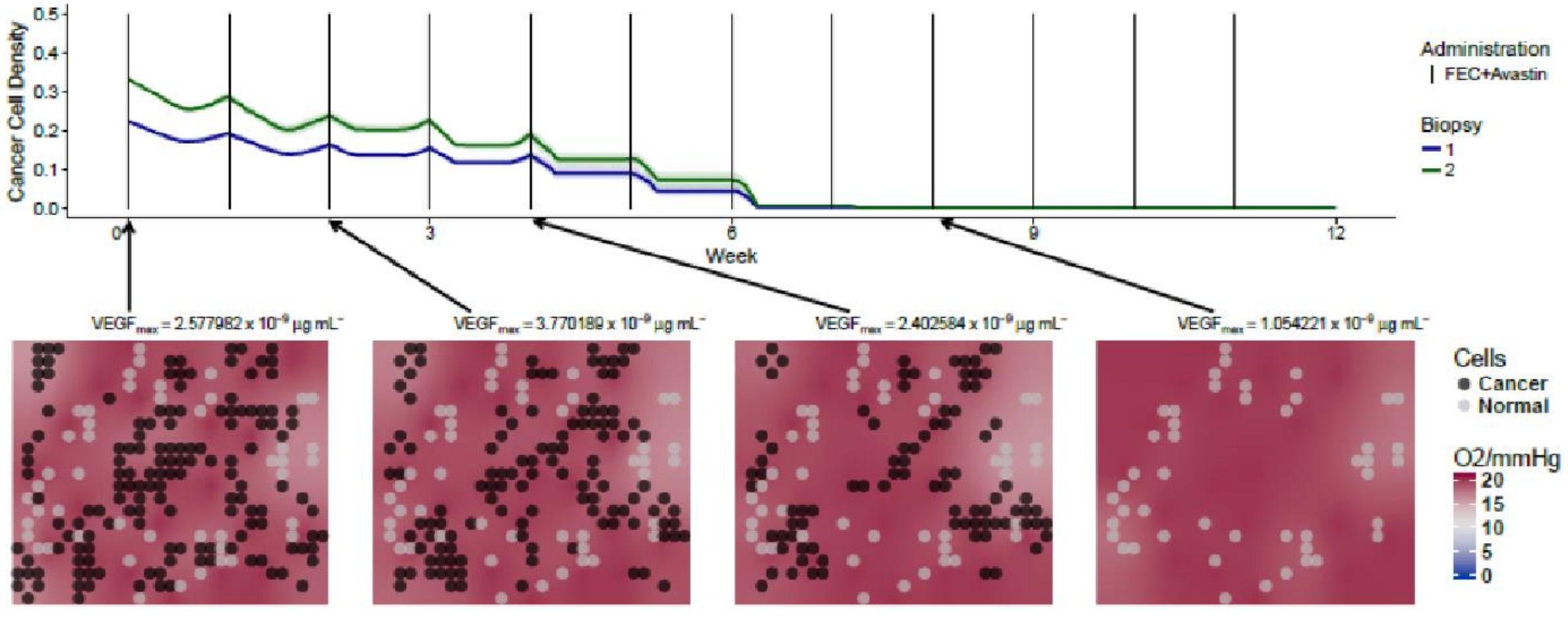
-Simulation

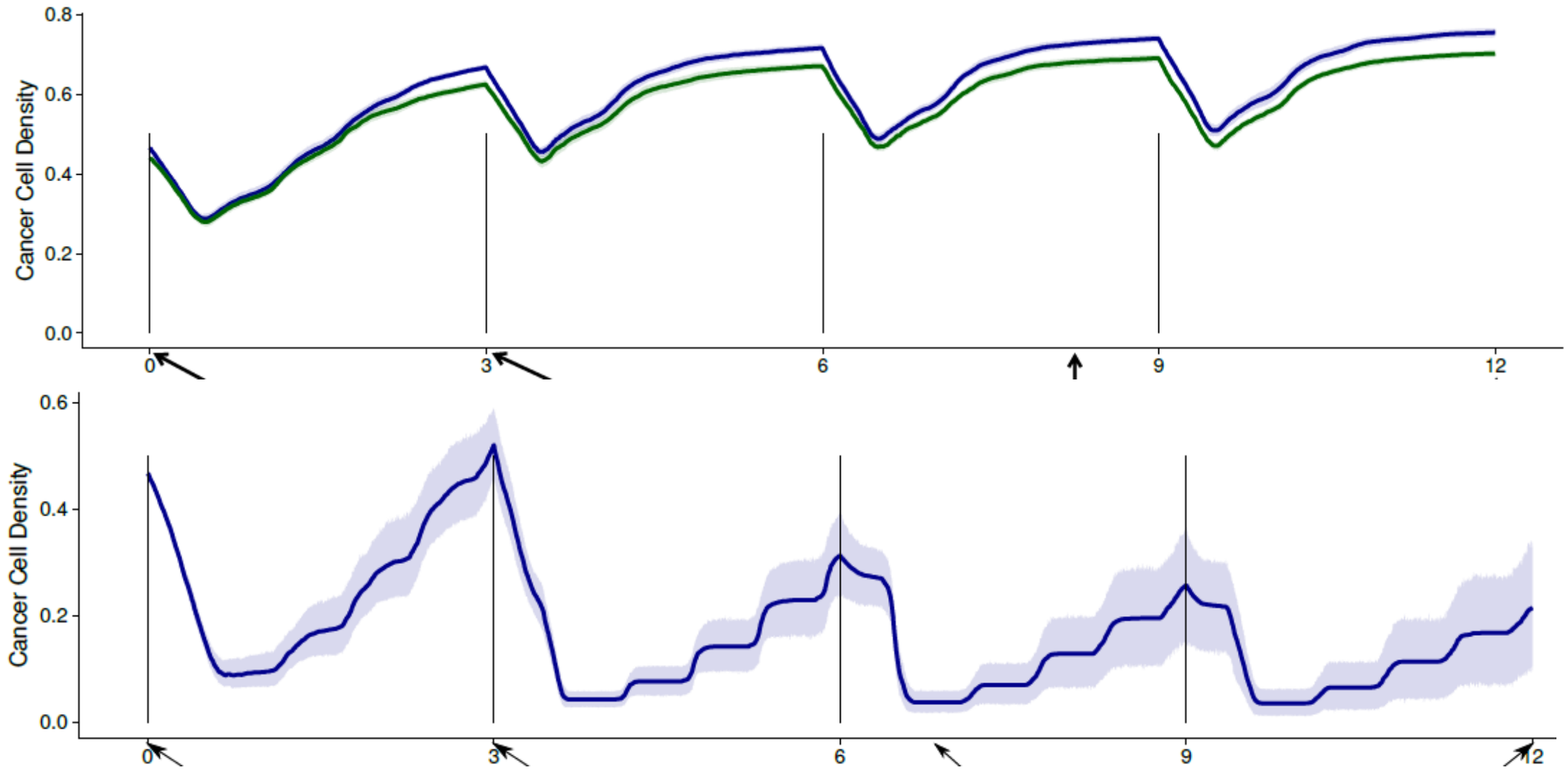


Patient 1 - Alternative therapy

-FEC and Avastin mab, more frequently, half dose

-Simulation: Responder

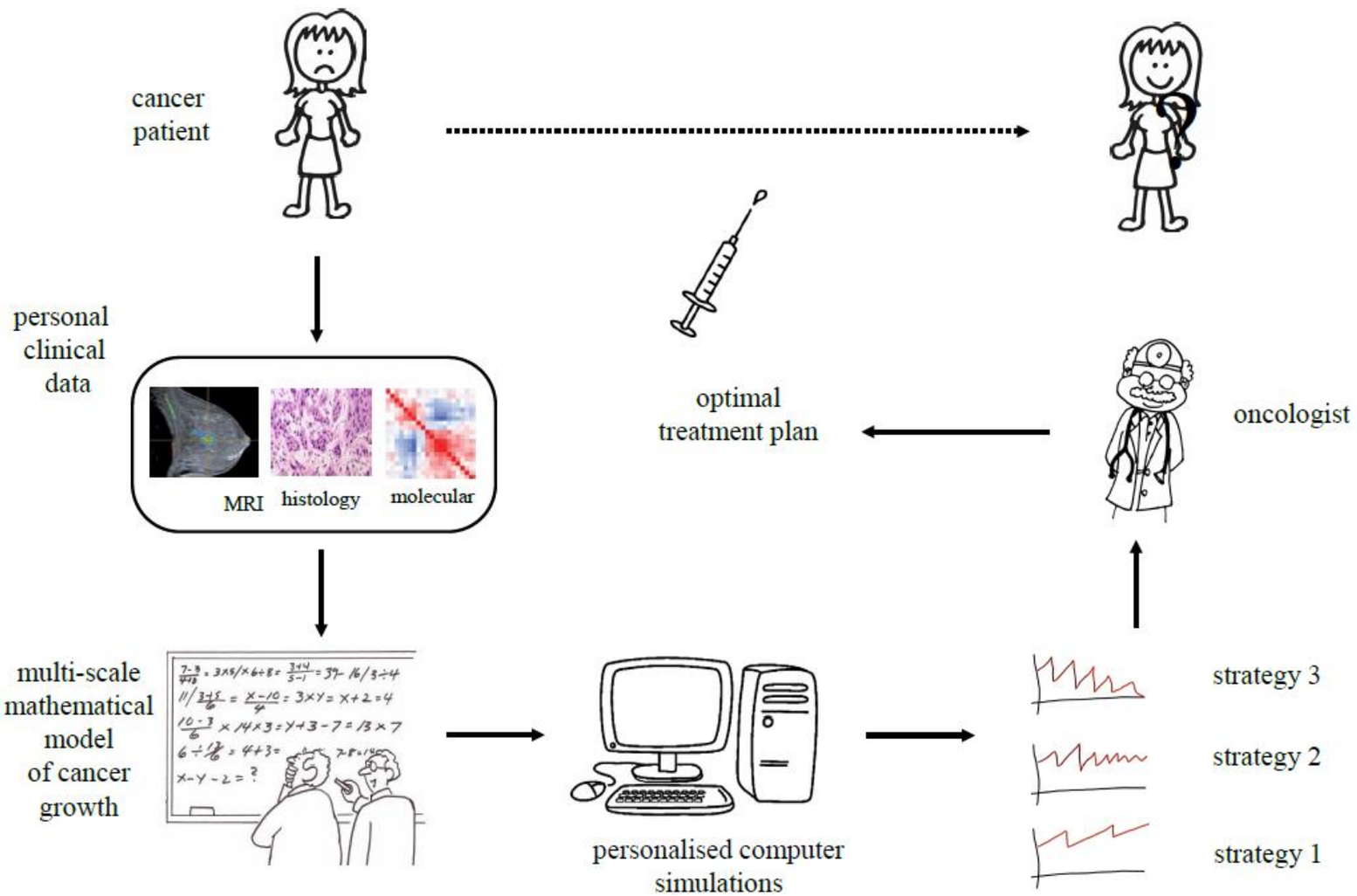




Patient 3 with two doses of Avastin: less is better

VERY FIRST STEPS

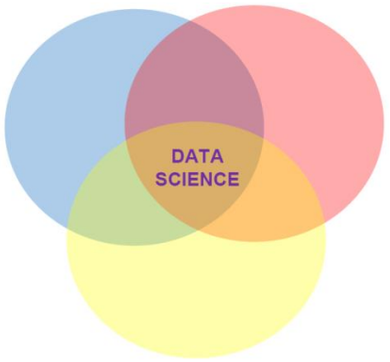
Sampling & Heterogenity



Regulatory approval

- **Heterogenity**
- **Genomics θ(genes)**
- **Drug mechanisms**

Optimisation



2. Data science for medical care

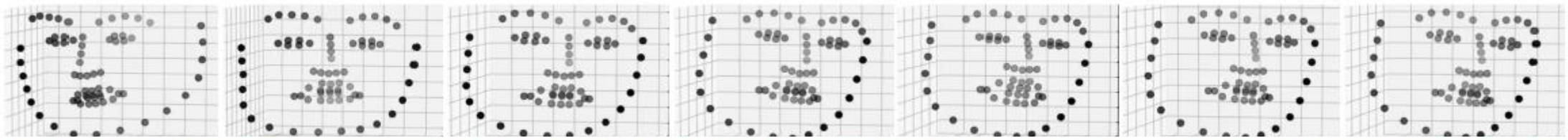
Measuring Depression Symptom Severity from Spoken Language and 3D Facial Expressions

Albert Haque[1] **Michelle Guo**[1] **Adam S Miner**[2,3] **Li Fei-Fei**[1]
[1]Department of Computer Science, Stanford University
[2]Department of Psychiatry and Behavioral Sciences, Stanford University
[3]Department of Health Research and Policy, Stanford University

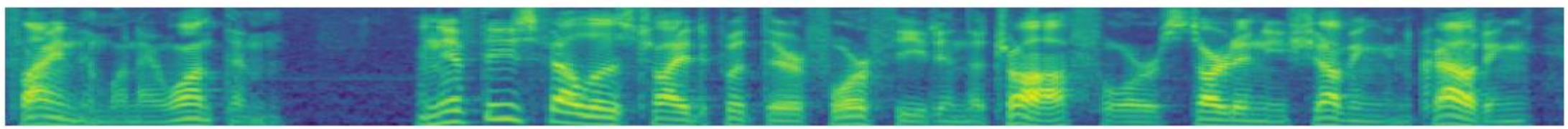
27 nov 2018

Data: 189 clinical interviews with a robot (5-25 minutes)
170 possible questions
‘How are you?’
‘Do you consider yourself to be an introvert?’
dialogic feedback (‘I see’, ‘that sounds great’).

Face



Sound



Text

um . . . yeah . . . . i mean they've always given me great advice . . they've always kept it real

Deep learning

Detected depression using language, voice and facial expressions.

npj | Digital Medicine

www.nature.com/npjdigitalmed

BRIEF COMMUNICATION OPEN

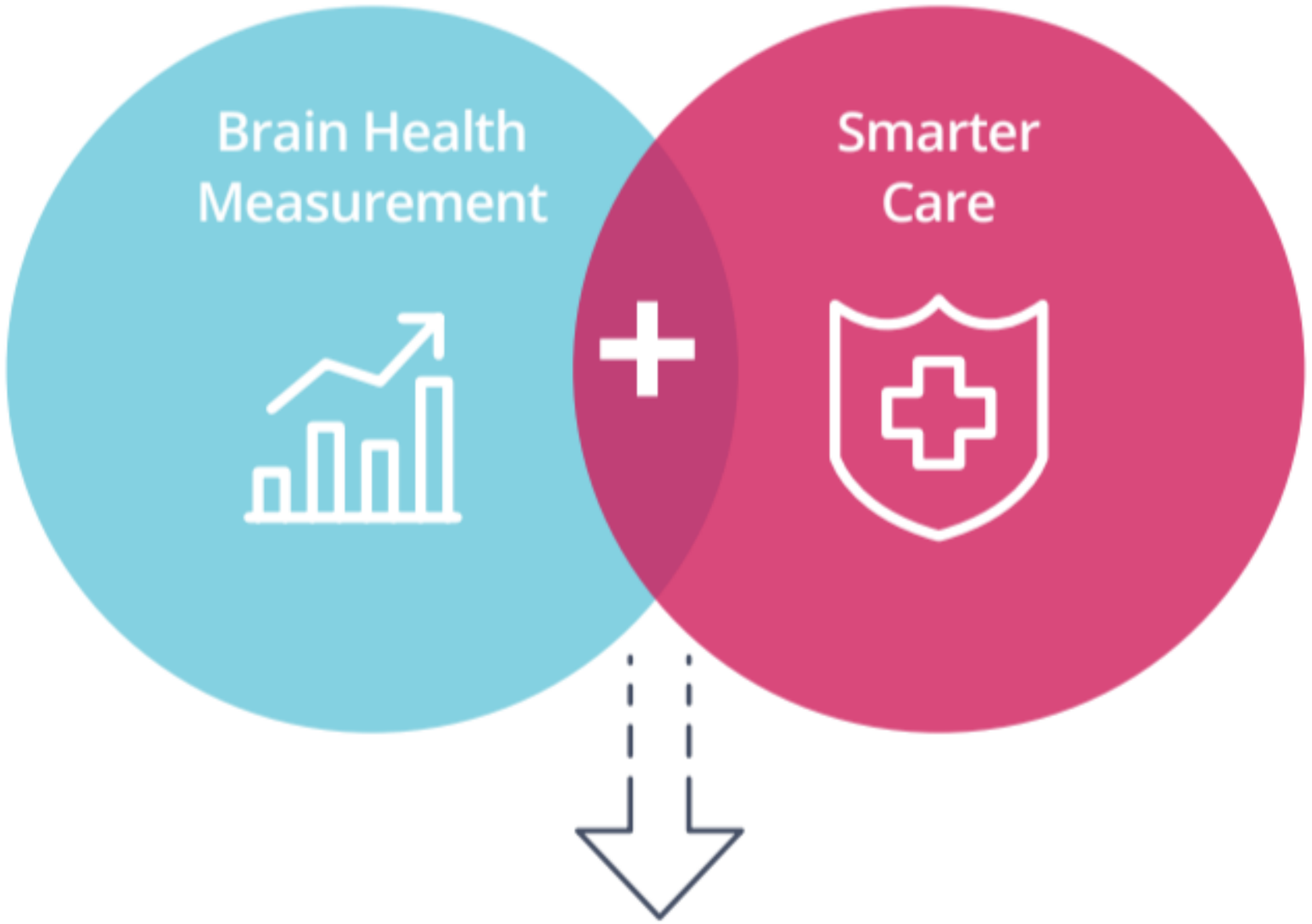
Digital biomarkers of cognitive function

Paul Dagum[1]

- Generate digital biomarkers from passively acquired data from a smartphone

	Mean (SD)	Range	*R* (predicted), *p*-value
Working memory			
Digits forward	10.9 (2.7)	7–15	0.71 ± 0.1[illegible], 10^{-4}
Digits backward	8.3 (2.7)	4–14	0.75 ± 0.0[illegible], 10^{-5}
Executive function			
Trail A	23.0 (7.6)	12–39	0.70 ± 0.1[illegible], 10^{-4}
Trail B	53.3 (13.1)	37–88	0.82 ± 0.0[illegible], 10^{-6}
Symbol digit modality	55.8 (7.7)	43–67	0.70 ± 0.1[illegible], 10^{-4}
Language			
Animal fluency	22.5 (3.8)	15–30	0.67 ± 0.1[illegible], 10^{-4}
FAS phonemic fluency	42 (7.1)	27–52	0.63 ± 0.1[illegible], 10^{-3}
Dexterity			
Grooved pegboard test (dominant hand)	62.7 (6.7)	51–75	0.73 ± 0.0[illegible], 10^{-4}
Memory			
California verbal learning test (delayed free recall)	14.1 (1.9)	9–16	0.62 ± 0.1[illegible], 10^{-3}
WMS-III logical memory (delayed free recall)	29.4 (6.2)	18–42	0.81 ± 0.0[illegible], 10^{-6}
Brief visuospatial memory test (delayed free recall)	10.2 (1.8)	5–12	0.77 ± 0.0[illegible], 10^{-5}
Intelligence scale			
WAIS-IV block design	46.1(12.8)	12–61	0.83 ± 0.0[illegible], 10^{-6}
WAIS-IV matrix reasoning	22.1(3.3)	12–26	0.80 ± 0.0[illegible], 10^{-6}
WAIS-IV vocabulary	40.6(4.0)	31–50	0.67 ± 0.1[illegible], 10^{-4}

mindstrong



Better Mental Health

nature COMMUNICATIONS

4 December 2018

ARTICLE

DOI: 10.1038/s41467-018-07262-2 OPEN

Smartphone app for non-invasive detection of anemia using only patient-sourced photos

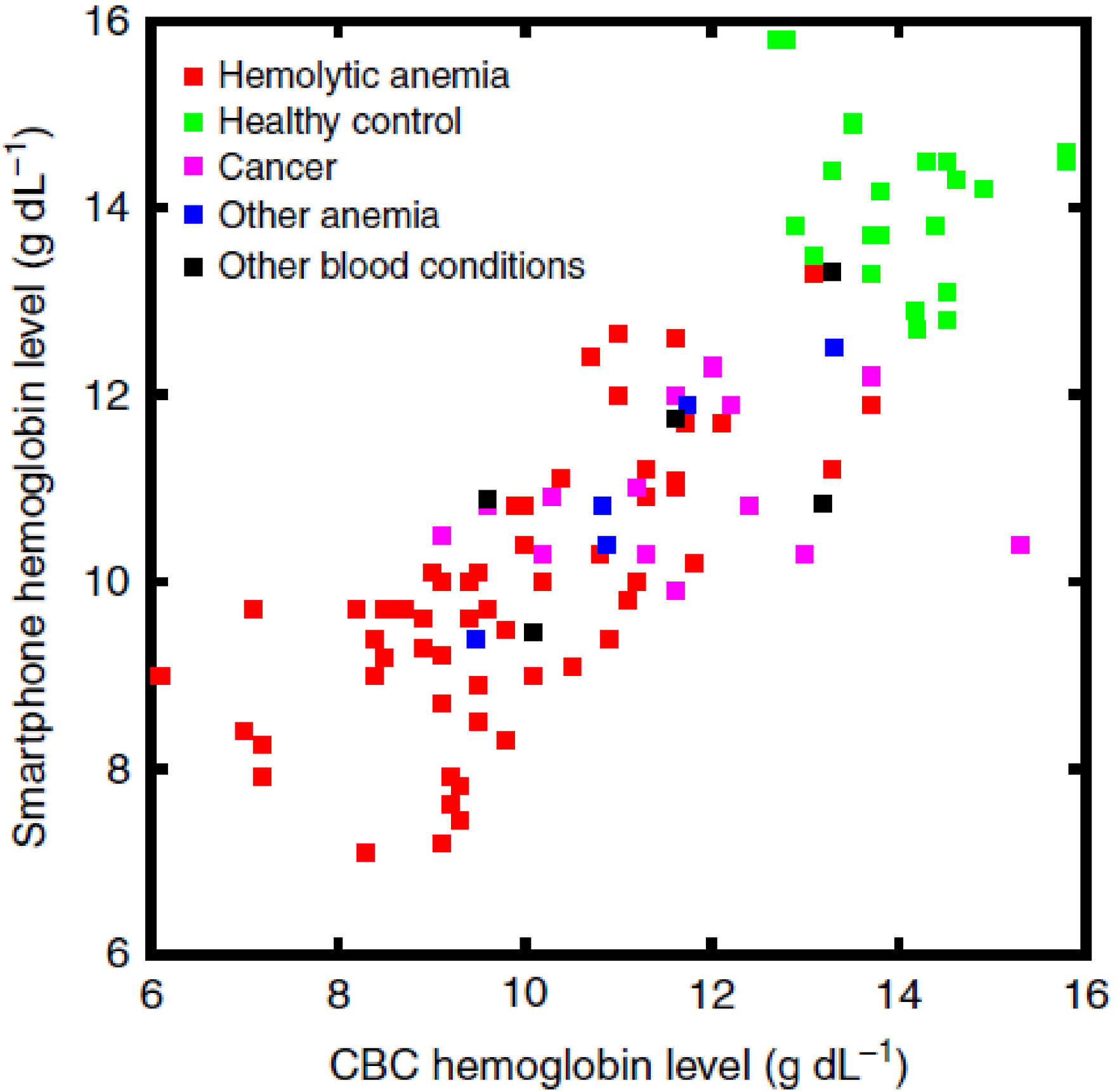
Robert G. Mannino[1,2,3], David R. Myers[1,2,3], Erika A. Tyburski[1,2,3], Christina Caruso[2], Jeanne Boudreaux[2], Traci Leong[4], G. D. Clifford[1,5] & Wilbur A. Lam[1,2,3]

Open app and take photos of fingernails

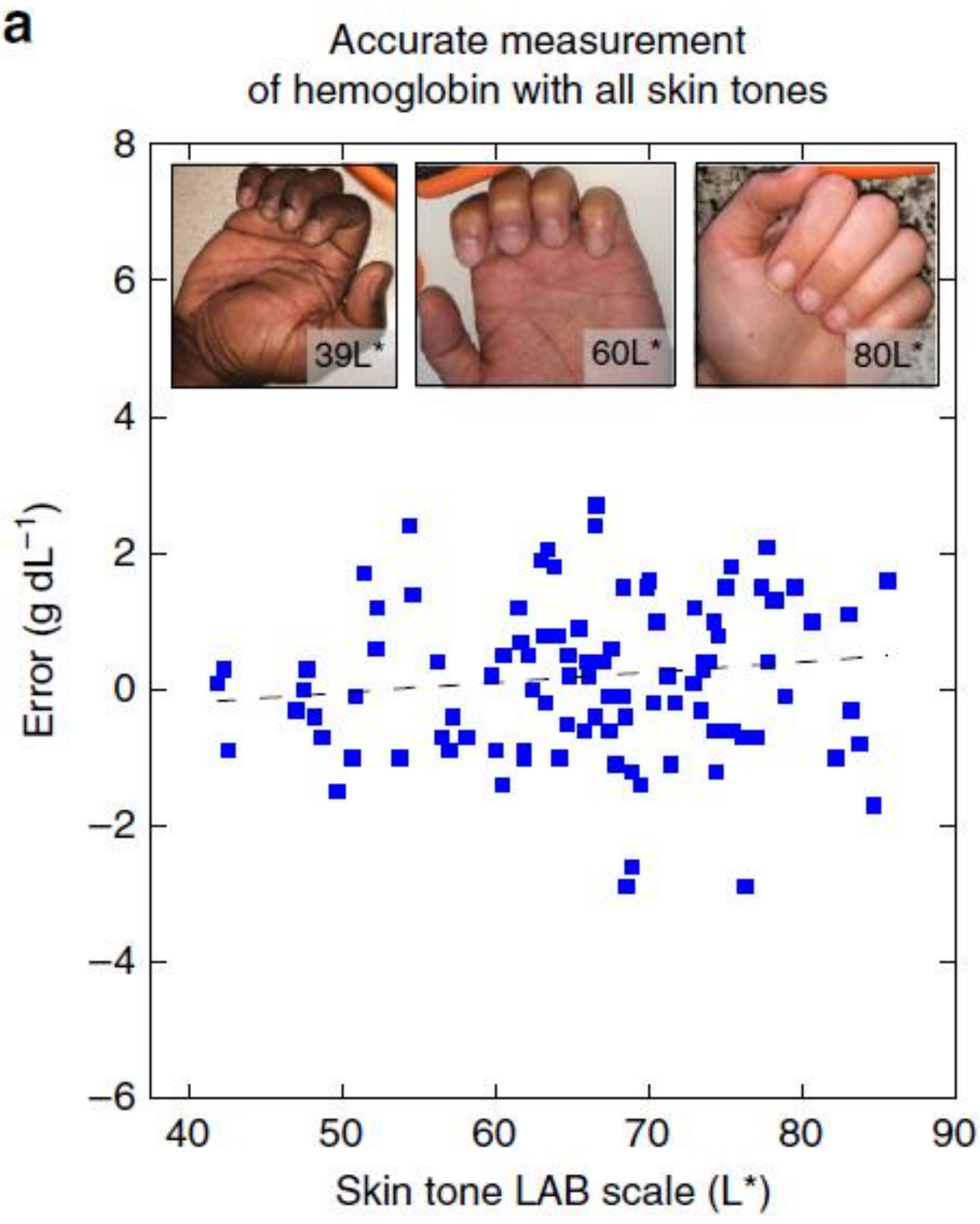
Select fingernails

Calculate hemoglobin

with personalised calibration, accuracy of ±0.92 g dL−1 wrt blood count hemoglobin levels


Hemolytic anemia
Healthy control
Cancer
Other anemia
Other blood conditions
Smartphone hemoglobin level (g dL^{-1})
CBC hemoglobin level (g dL^{-1})
6
8
10
12
14
16

a


Accurate measurement
of hemoglobin with all skin tones
39L*
60L*
80L*
8
6
4
2
0
−2
−4
−6
Error (g dL⁻¹)
40
50
60
70
80
90
Skin tone LAB scale (L*)

Smartphone app for non-invasi | Online Doctor Consultations & | Ada: Your Personal Health Guid

https://www.babylonhealth.com

Search

babylon

About Product Blog NHS Sign up Log in Download App

Hello, how can I help you?

I want to talk to a GP

Ask Babylon

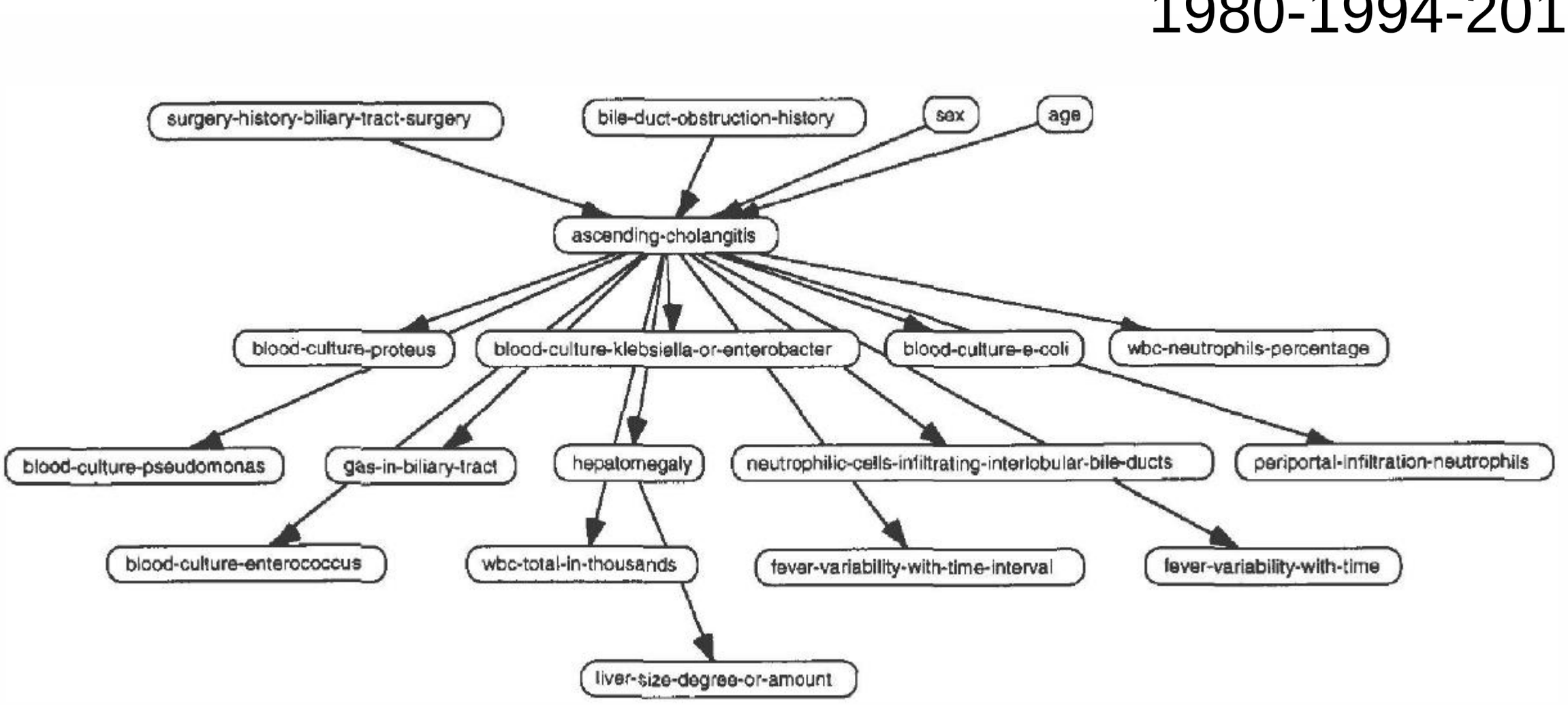
"As easy as ordering a cab on your smartphone"

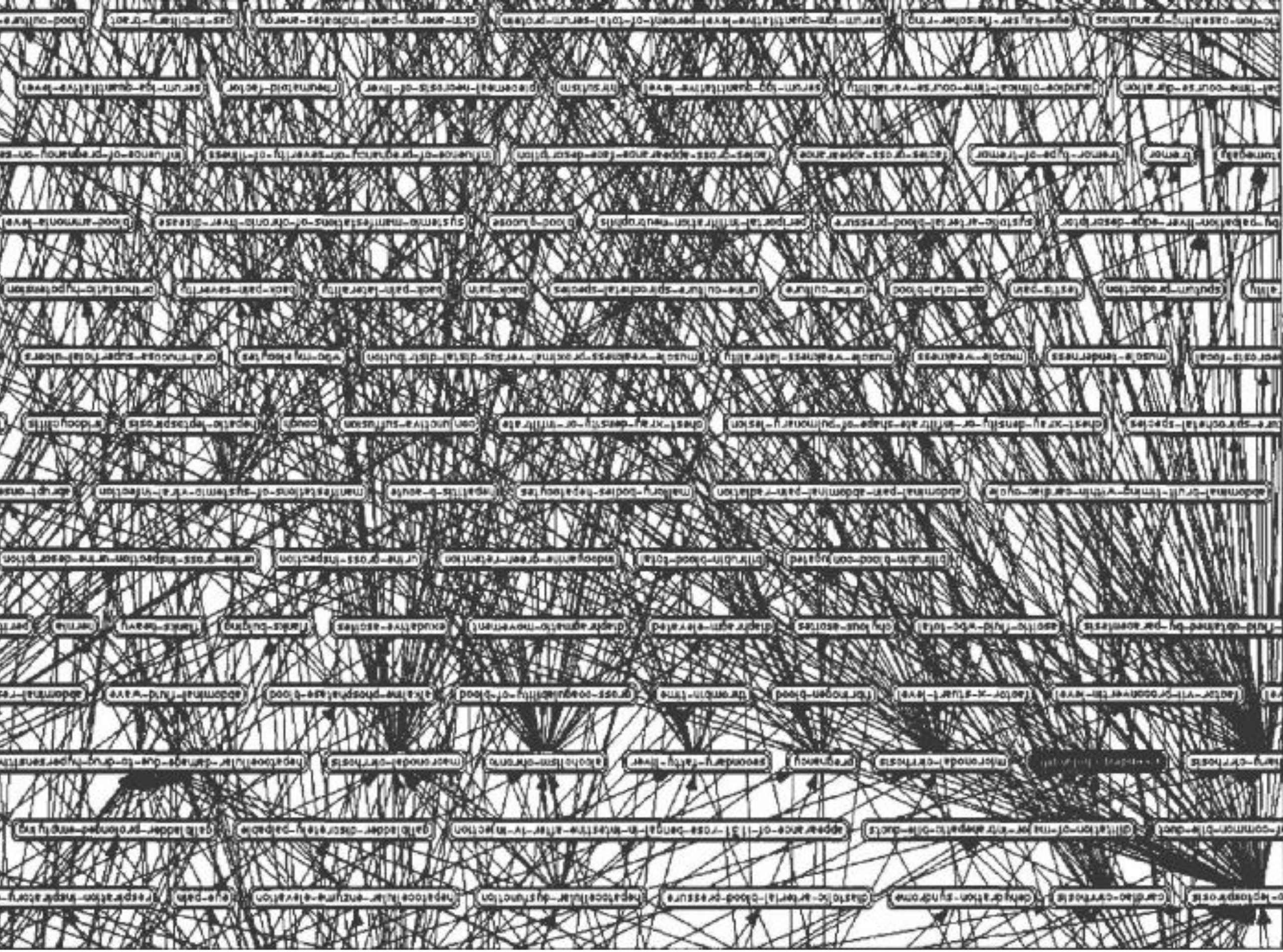
Help us improve by sharing your feedback.

NO 19:58 17.12.2018

Babylon is based on a Probabilistic Graphical Model of primary care medicine, which models the prior probabilities of diseases and the conditional dependencies between diseases, symptoms and risk factors via a directed acyclic graph.

1980-1994-2018





A comparative study of artificial intelligence and human doctors for the purpose of triage and diagnosis

Salman Razzaki*, Adam Baker*, Yura Perov*, Katherine Middleton*, Janie Baxter*, Daniel Mullarkey*, Davinder Sangar*, Michael Taliercio*, Mobasher Butt*, Azeem Majeed†, Arnold DoRosario‡, Megan Mahoney§ and Saurabh Johri*,¶

* Babylon Health.
† School of Public Health, Faculty of Medicine, Imperial College London.
‡ Northeast Medical Group, Yale New Haven Health.
§ Division of Primary Care and Population Health, School of Medicine, Stanford University.
¶ Corresponding author: Saurabh Johri, saurabh.johri@babylonhealth.com.

27 june 2018

Triage =henvisning

- The MRCGP final exam to become a GP

- Average pass mark for real-life doctors was **72%**
- Babylon scored **81%**.

- Accuracy of Babylon was 98% when assessed against most frequent conditions in primary care.
- In comparison, experienced clinicians: 52%-99%.

- **“We're pioneering AI to make healthcare universally accessible and affordable”**

Smartphone app | Press room | Baby | Probabilistic Grap | Overview and Mo | [1806.10698] A cc | assets.babylonhealth | Ada: Your Per

https://ada.com

Search

ada

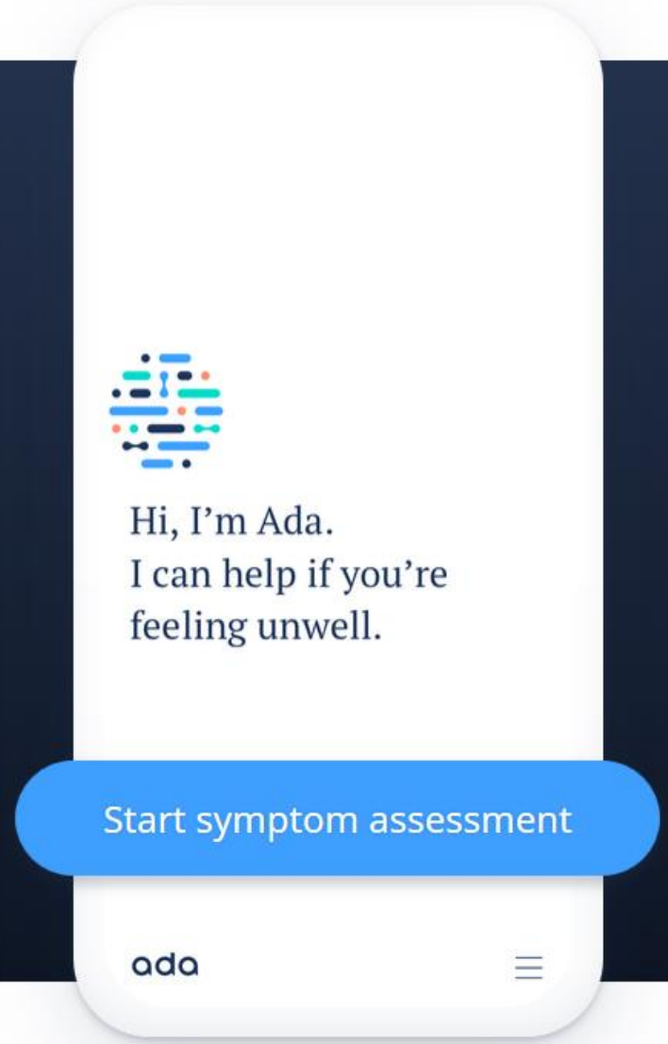
Get Ada | About us | Medical Library

Your personal health guide

Created by doctors, scientists, and engineers to put free, AI-powered healthcare in everyone's hands.

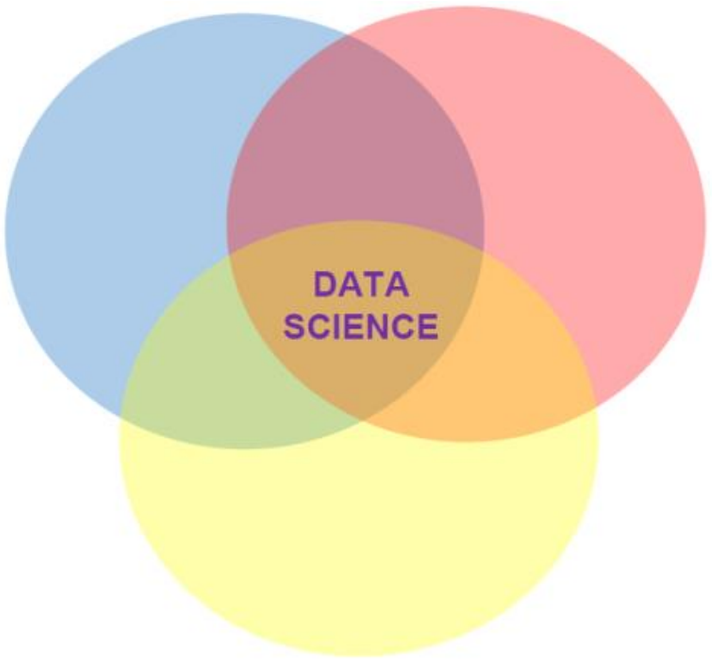
Download on the App Store

GET IT ON Google Play



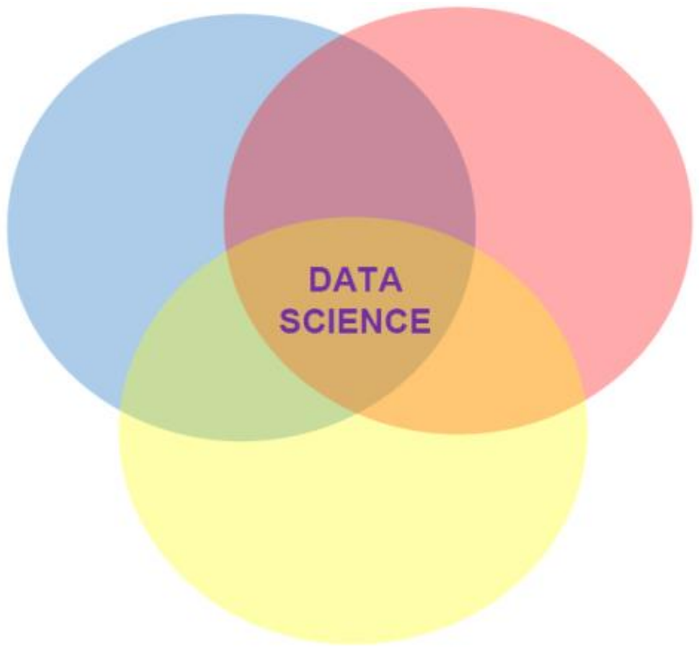
NO 20:45 17.12.2018

Data Science in Health



- All this comes, not tomorrow, but soon
- We have time to build competence
  (but better start)
  - legeutdanning
  - reskilling
- Cross-disciplinarity essential, and research must reshape
- Enormous advantages
- Understand the purpose
- Responsible use of AI.
  Prevent catastrophes.

Data Science in Health

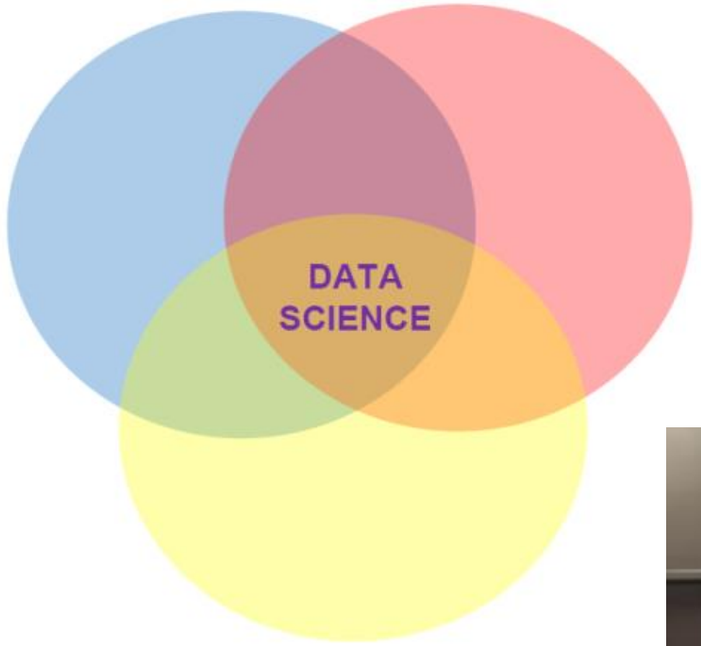


- Medfak: prepare
- Next step for legeutdanning?
- Continuum education of doctors and researchers?

- Are current «research groups» useful? Faculties?

- Data Science at UiO
- Data Science centre at LV byggning.

Data Science in Health



- Medfak: OCBE!

- Very costly
- More unequal health system.

- Genetically modified human embryo to prevent cancer in children from parents with a risky mutation.
- Designer baby
- There is a distinction between preventing disease and picking traits. (or maybe not?)
- Expensive.
- We risk creating a world, where some people bear more genetic disease, because of their social, economical, geographic status.

WEARABLES SHIFT TO NEW MARKETS AND APPLICATIONS

Wearable Technology
2016-2026

Head

Immersive infotainment, military apparel, helmets (workwear)

Ear

Hearing aids, headphones, ear-worn trackers

Eyes

Smart eyewear, AR/VR, smart contact lenses.

Body (torso)

Smart clothing, chest straps, workwear, medical, skin patches., sports

Arms

Medical smartwear, arm-worn trackers, skin patches, sports apparel

Wrist

Smartwatches, wrist-worn fitness trackers

Legs & feet

Smart footwear, medical devices, military, sports apparel

Other

Implantable, multi-location, adaptability by user or use case.

LOCATION ON BODY
OF VARIOUS WEARABLES

Inspired by IDTechEx

http://ow.ly/QCYd306WF7u