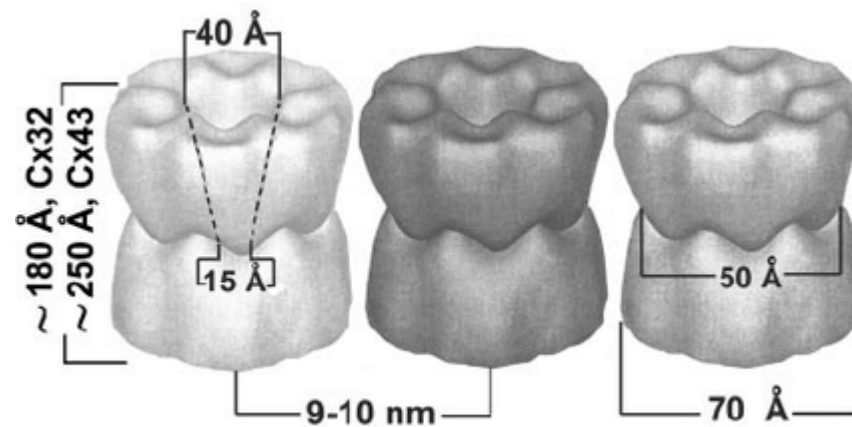


A physics perspective on cancer



João Carvalho

CFisUC : Physics Department : University of Coimbra

15 February 2023



UNIVERSIDADE DE
COIMBRA



"There's a belief in underlying simplicity that has guided physics since forever. Where we see complication, that's where we don't understand the **principles** yet." Andrew Ewald

in **Cancer and the artillery of physics** Gabriel Popkin, Johns Hopkins Magazine, 2018

Disclaimer (personal view)

The opinions expressed in this presentation are those of the **author**. They do not purport to reflect the opinions or views of the CFisUC, UC or its members.

The presented views result mainly from conceptual work, bibliographic research, discussions with collaborators, thought experiments and theoretical reasoning (only the researcher ego was hurt in this work)



Every week media
present good news
about cancer
advancements

But people are still
dying from **cancer**,
and every diagnosis
is a Damocles
sword in the person
(& family) life

Sword of Damocles, Richard Westall, 1812

Drugs combo hope for cancer patients

Prostate cancer patients could get their first targeted treatment after "highly exciting" results from a drug already used for ovarian and breast cancer. Men with key genetic mutations found that elaprevir stopped their tumours growing for an average of eight months, with a third seeing cancer shrink for more than a year. The treatment is likely to be available within two years and could benefit about 4,000 men a year in Britain, experts believe.

Drugs that target particular genetic mutations have become one of the mainstays of cancer treatment in the past generation, improving survival for patients with many types of cancers. However, none has been discovered for prostate cancer, which is Britain's most common male cancer and affects 47,000 men a year.

The latest results suggest targeted therapy for prostate cancer has finally arrived and genetic testing to decide on



Breast cancer drug could be used to fight prostate tumours

Chris Smyth Health Editor, Chicago

Analysis

A few previous years' treatment over gene-changing medicines, making the most of what we already know has been a theme of the world's largest cancer conference this year.

A series of encouraging studies in prostate cancer has started to close a treatment gap with other forms of the disease, and doctors are increasingly interested in simple ways to help patients' control of their health, from exercise and diet to medication.

Though prostate cancer

is Britain's most common male cancer and kills 12,000 a year, recently overtaking breast cancer as the third biggest cancer killer, research has lagged behind.

In the past two decades, twice as many studies have been published on prostate cancer as on breast cancer, and in Britain research spending on the most common male cancer is half that of its female counterpart.

John de Bono, of the Cancer Research UK, said that pharmaceutical companies were reluctant to fund his research into

targeted prostate cancer drugs, forcing him to turn to charities for help to support an existing ovarian and breast cancer as the third biggest cancer killer, research has lagged behind.

The result is that while targeted therapies began to emerge for other cancer types 20 years ago, only now are they emerging for prostate cancer. There is still no screening programme, tests are inaccurate and picking out the most aggressive cancers is still a struggle. One reason for this may be that while

breast cancer has benefited from a powerful patient voice and celebrity supporters, there is nothing similar for prostate cancer.

The emergence of Masquerade, a young men's facial hair to boost the profile of male cancers, suggests that this is starting to change. Last year the government pledged an extra £75 million for research into prostate cancer; and with the emergence of targeted medicine, "search and destroy" radiotherapy and drugs that unleash the immune system against tumours, there is good reason for optimism.

The Guardian

UK edition

New head and neck cancer drug could help patients live longer

Pembrolizumab with platinum chemotherapy less 'aggressive' and extended survival rates

Immunotherapy could help patients with head and neck cancer live long new research suggests.

The drug pembrolizumab, used in combination with platinum chemotherapy, was found to be returned or spread, according to Clinical Oncology annual

The treatment, which works and fight cancer, was also effective produced fewer side effects

The findings of the study, carried out at Royal Marsden in London, suggest treatment for those with an

Drug for women with 'Jolie gene' can also curb prostate cancer

Jy Lesca Dossou

Health Editor in Chicago

A PROMISING drug that boosts survival for women with breast cancer can still progress of prostate cancer in men, a study has found.

The British trials suggest the treatment - which is also used to treat ovarian cancer - could benefit up to 4,000 men a year, delaying the moment when the disease becomes deadly.

Charles said they were "very excited" about the findings from the Institute of Cancer Research (ICR) and the Royal Marsden Hospital.

The drug, called olaparib, is already licensed for the ICR for women with ovarian cancer fuelled by BRCA gene mutations and will soon be reviewed for its use for breast cancer.

The damaged DNA is sometimes known as the "Jolie gene" after Angelina Jolie, the Hollywood actress who underwent a double mastectomy and ovary removal to cut her cancer risk.

Research has now found that the treatment worked in 80 per cent of men with prostate disease who were screened for the same mutation.

Even though the patients had advanced disease, the drug stalled the cancer for an average of eight months, with some for years free of progression.

Charles said: "The next phase of the trial is to see if we can use the treatment to prevent prostate cancer in men with the mutation."

The drug, which costs £3,500 a month, was made available to the ICR for ovarian cancer patients in 2015 and for breast cancer in 2016. It was the first time it had been used in men.

Charles said the study was "very exciting" and that the results were "very promising".

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four with olaparib in one or more genes linked to damaged DNA, these were most commonly because of BRCA mutations.

Overall, 47 per cent of men with olaparib responded to the drug, delaying progression for an average of about six months. In men cases, some had no response at all.

Prof John de Bono, the study leader from the ICR, said: "By testing for DNA repair mutations we can identify those patients with a high chance of responding well, and for a long time, to the targeted drug olaparib."

Overall, between one in three and one in four men with inherited prostate cancer responded - those with BRCA mutations. That means 1,000 or 2,000 men a year in the UK would benefit from these drugs.

He said he hoped the ICR would fund the treatment in future.

Every year, about 47,000 men in the UK are diagnosed with prostate cancer, causing 12,000 deaths.

Prof de Bono said: "The next phase of the trial is now under way and, if the results look as good as we hope, we should see olaparib starting to reach the clinic for men with prostate cancer in the next few months."

"As we move towards the era of personalised medicine, knowing in advance which genetic faults in prostate tumours could provide more options for certain men with the disease."

Prostate cancer cells tracked and zapped

High-tech molecules can 'seek and destroy' tumours ... avoiding chemo and extending lives of thousands

New blood test 'can predict return of breast cancer before treatment'

By PRESS ASSOCIATION
PUBLISHED: 10:44, 2 June 2019 | UPDATED: 10:26, 2 June 2019

A new blood test could help predict whether women with breast cancer will respond to treatment before it begins.

Scientists at the Institute of Cancer Research in London said the "liquid biopsy" can detect genetic changes in the tumours of patients and indicate if they are less likely to respond to a new targeted drug.

'Search and destroy' treatment for prostate cancer may mean longer life for thousands

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"As we move towards the era of personalised medicine, knowing in advance which genetic faults in prostate tumours could provide more options for certain men with the disease."

The study accepted the findings of 102 men, and found more than one in

CANCER CONFERENCE

Blood test can pinpoint if cancer will return

A 'liquid biopsy' is able to identify within minutes the patients who will respond to treatment - and those who will not

Andrew Gregory Health Editor, Chicago

A simple new blood test could revolutionise cancer care by predicting which patients are likely to respond well to treatment and which should be switched to different drugs.

Details of the "liquid biopsy" were unveiled by London scientists were unveiled yesterday at the world's biggest cancer conference, the annual meeting of the American Society of Clinical Oncology in Chicago. The test, which takes minutes to perform, could identify those whose tumours are expected to shrink and those whose cancer is likely to return.

Professor Paul Workman, chief executive of the Institute of Cancer Research (ICR), which developed the test, said: "Cancer's ability to evolve is its greatest strength and its greatest challenge. We have now developed a way to predict which patients will respond well to treatment and which should be switched to different drugs."

The test, which takes minutes to perform, could identify those whose tumours are expected to shrink and those whose cancer is likely to return.

PERSONALISED PROSTATE PILL

Drug that attacks tumours is tailored to genes - and could help 4,000 men a year

ENDING NEEDLESS PROSTATE DEATHS

Prostate cancer is the most common cancer in men in the UK, with 47,000 men diagnosed each year. It is the second leading cause of cancer death in men, with 12,000 men dying each year from the disease.

Prostate cancer is a disease of the prostate gland, which is a small, walnut-sized organ in the male reproductive system. It is the most common cancer in men in the UK, with 47,000 men diagnosed each year. It is the second leading cause of cancer death in men, with 12,000 men dying each year from the disease.

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... but charities lose out


NOTE

The medical condition usually designated by cancer is not a single disease

Cancer is a group of diseases (actually more than 100) characterized by the uncontrolled proliferation of cells

A full-blown
war on cancer
started 50
years ago
What have we
advanced?

Mr. Nixon: You can cure cancer



If prayers are heard in Heaven, this prayer is heard the most:

"Dear God, please. Not cancer."

Still, more than 318,000 Americans died of cancer last year.

This year, Mr. President, you have it in your power to begin to end this curse.

As you agonize over the Budget, we beg you to remember the agony of those 318,000 Americans. And their families.

We urge you to remember also that we spend more each day on military matters than each year on cancer research. And, last year, more than 21 times as much on space research as on cancer research.

We ask a better perspective, a better way to allocate our money to save hundreds of thou-

sands of lives each year.

America can do this. There is not a doubt in the minds of our top cancer researchers that the final answer to cancer can be found.

Already, 4 out of about 200 types of cancer can be cured with drugs. And 37 other drugs will cause temporary remission in 17 other types of cancer.

Dr. Sidney Farber, Past President of the American Cancer Society, believes: "We are so close to a cure for cancer. We lack only the will and the kind of money and comprehensive planning that went into putting a man on the moon."

Why don't we try to conquer cancer by America's 200th birthday?

What a holiday that would be! Cancer could be then where smallpox, diphtheria and polio

are today—almost nonexistent.

If you fail us, Mr. President, this will happen: One in six Americans now alive, 34,000,000 people, will die of cancer unless new cures are found.

One in four Americans now alive, 51,000,000 people, will have cancer in the future.

We simply cannot afford this.

Our nation has the money on one hand and the skills on the other. We must, under your leadership, put our hands together and get this thing done.

Surely, the war against cancer has the support of 100% of the people. It is a war in which we lost 21 times more lives last year than we lost in Viet Nam last year. A war we can win and put the entire human race in our debt.

To the public, cancer patients, their friends and relatives:

Write or wire the President, urging him to put more funds behind cancer research. Or, please use this coupon.

Dear Mr. Nixon:
Cancer research needs more funds. Please provide them in your 1971 budget. Please.

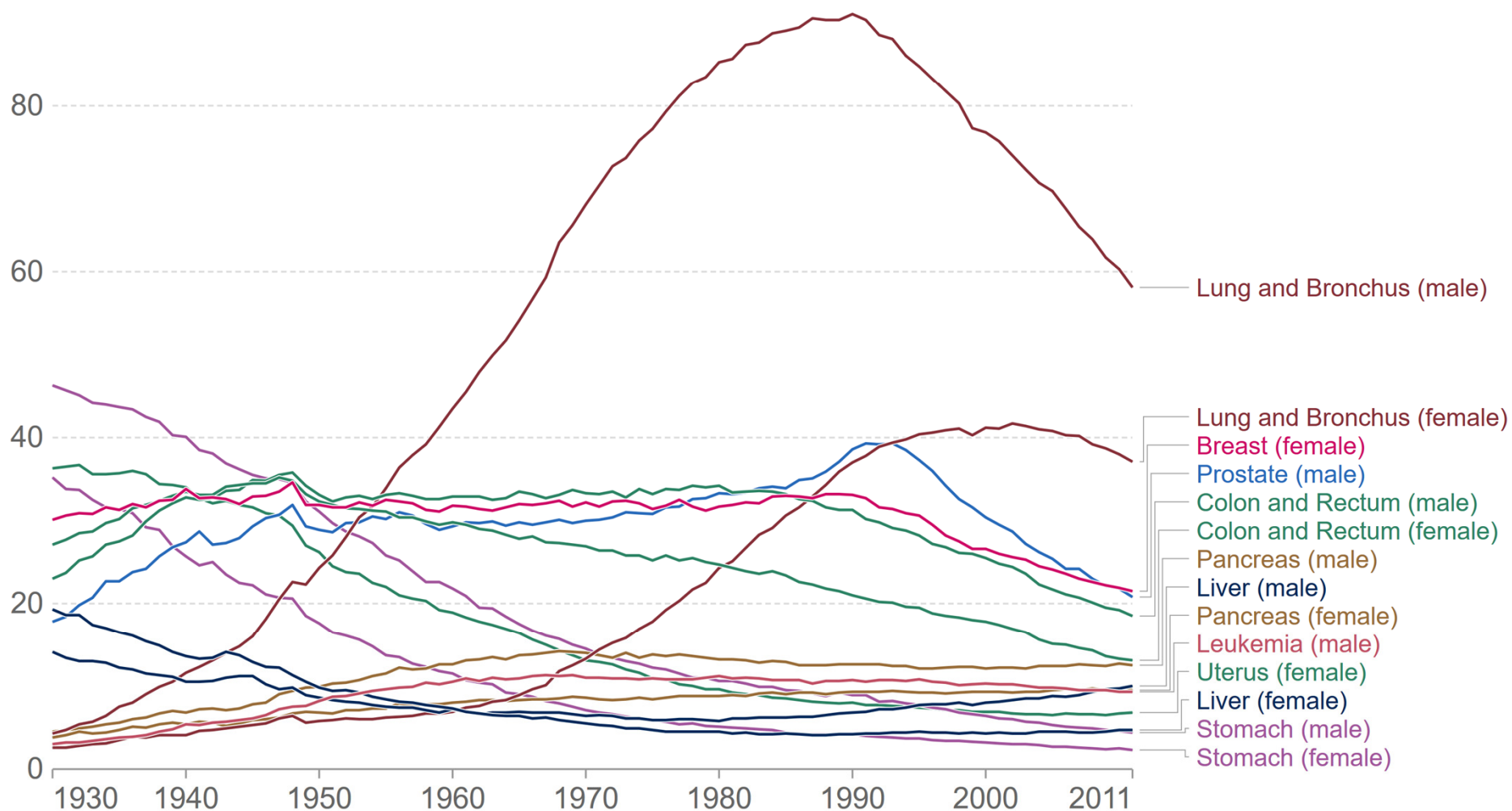
NAME _____
ADDRESS _____
CITY _____ STATE _____ ZIP _____

Mail this coupon to: The President
The White House
Washington, D.C.

CITIZENS COMMITTEE FOR THE CONQUEST OF CANCER
866 United Nations Plaza, New York, N.Y., Solomon Garb, M.D., Emerson Poole, Co-chairmen

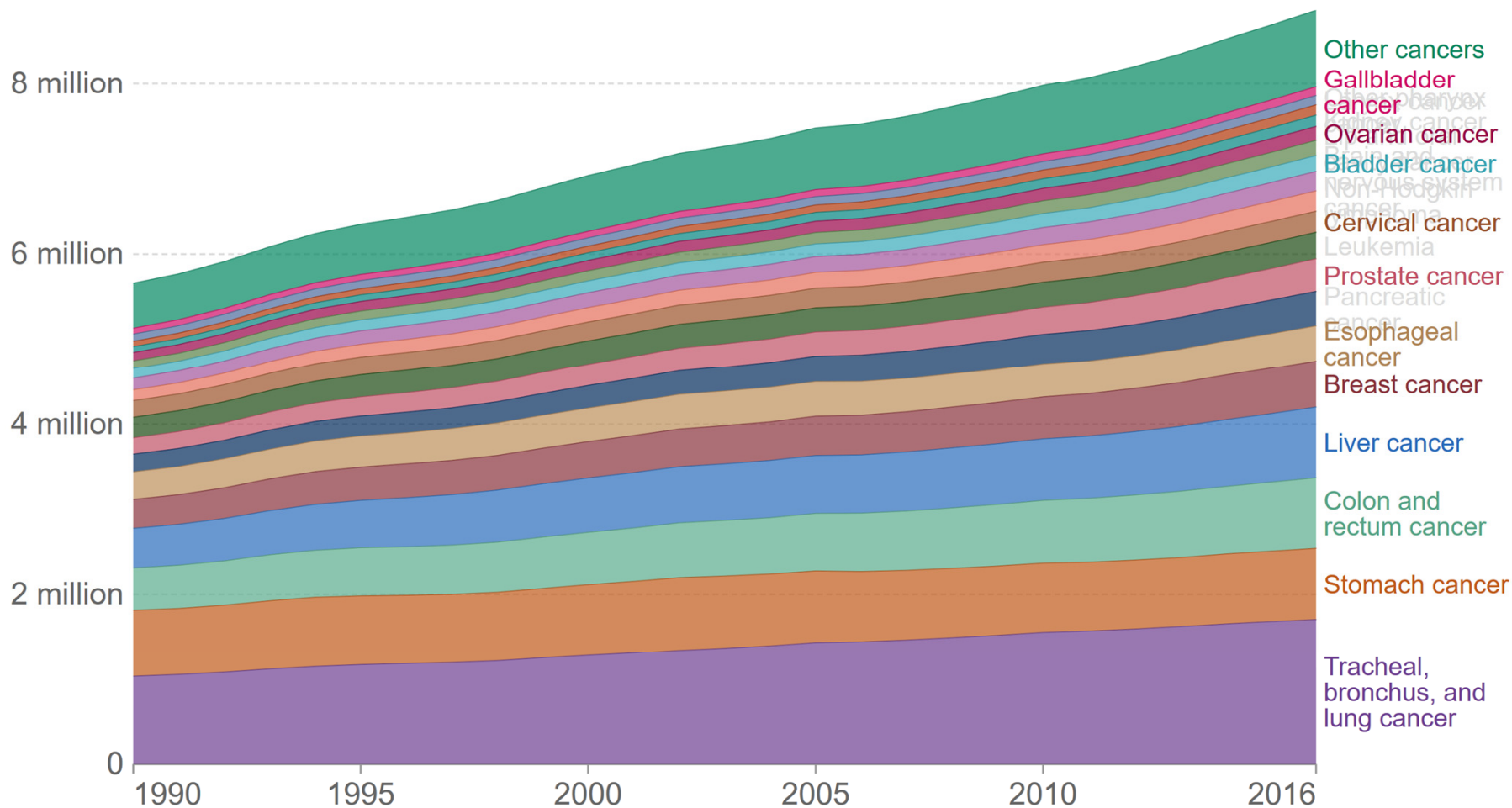
Cancer death rates in the United States over the long-run

Age-standardized death rates from various forms of cancer in males and females, measured as the number of deaths per 100,000 individuals. Age-standardization is based on normalisation to the standard US population structure in the year 2000.



Cancer deaths by type, World, 1990 to 2016

Annual cancer deaths by cancer type, measured as the total number of deaths across all age categories and both sexes. Smaller categories of cancer types with global deaths <100,000 in 2016 have been grouped into a collective category 'Other cancers'. See sources for list of grouped cancers.



Source: IHME, Global Burden of Disease (GBD)

OurWorldInData.org/cancer • CC BY

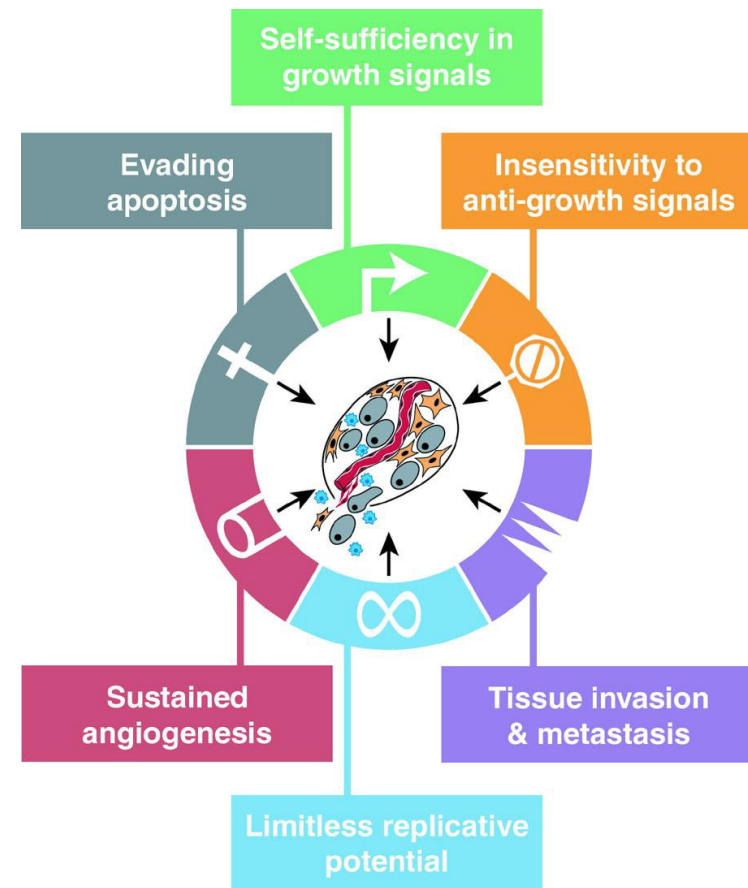
Note: All cancer types with less than 100,000 global deaths in 2016 into a collective category 'Other cancers'.

World population: 1990=5.3G, 2016=7.5G

Very Influential Publication (VIP)

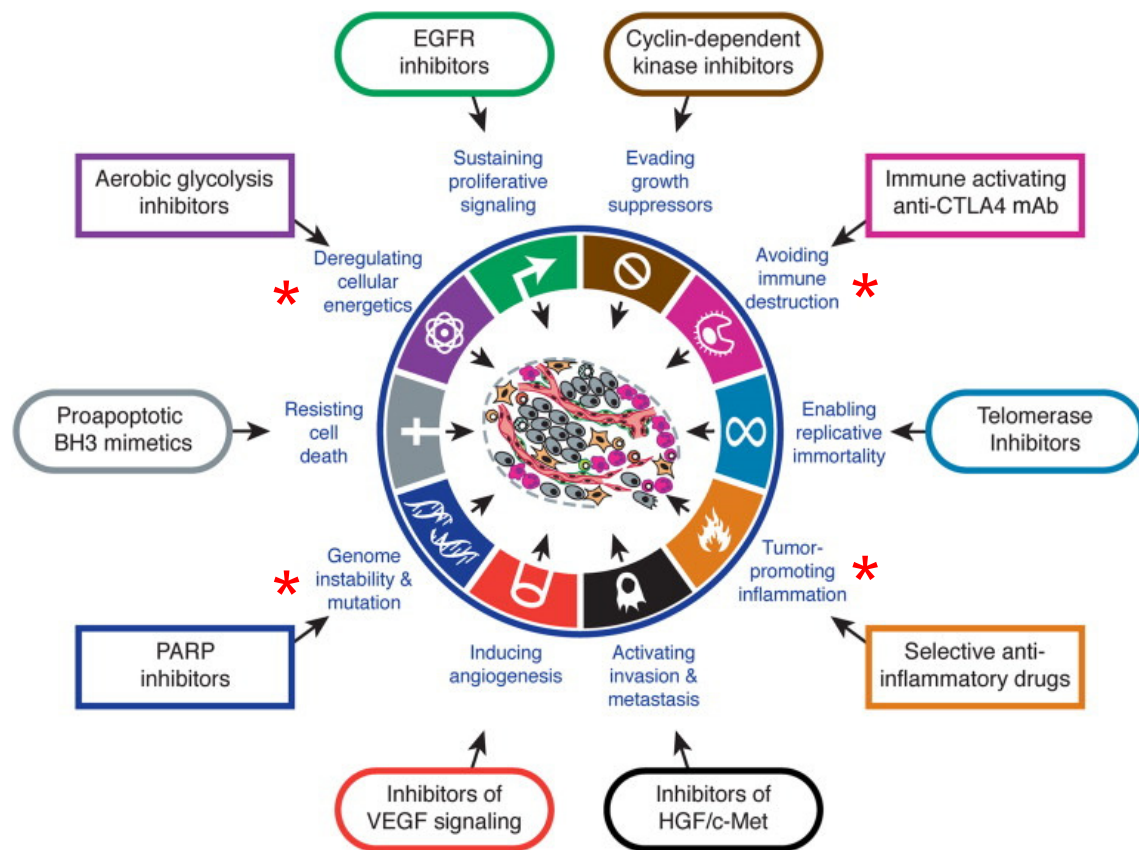
Douglas Hanahan and Robert A. Weinberg “**The hallmarks of cancer**”, Cell, 2000 (~40k citations)

Six characteristics of cancer cells acquired through successive **genetic mutations** at the disease origin and progress: the **cancer cell** paradigm



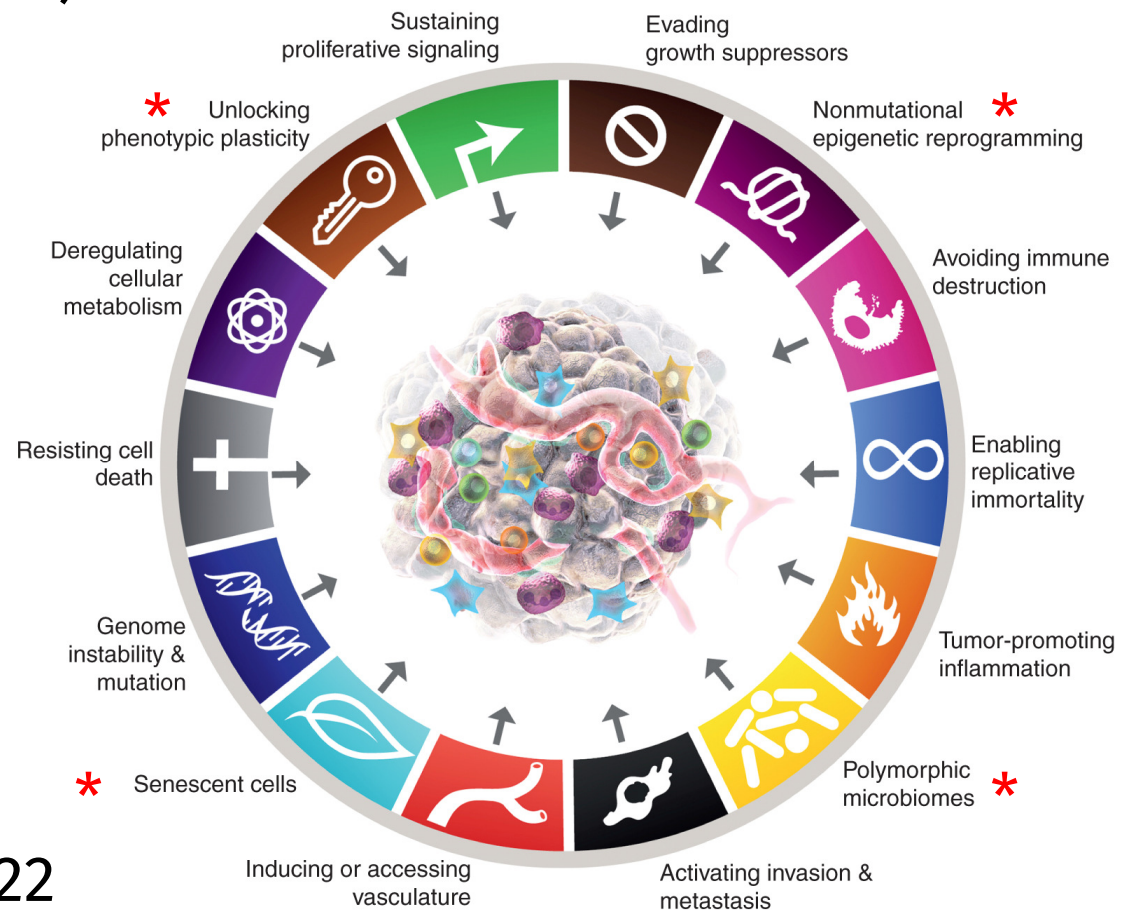
Later updated with further 4 hallmarks:

Douglas Hanahan and Robert A. Weinberg
“Hallmarks of Cancer: The Next Generation”,
Cell, 2011 (~67k citations)



And it was further updated with extra 4 properties (growing into irrelevance, almost everything fits in!)

Douglas Hanahan
“**Hallmarks of Cancer: New Dimensions**”,
Cancer Discov., 2022

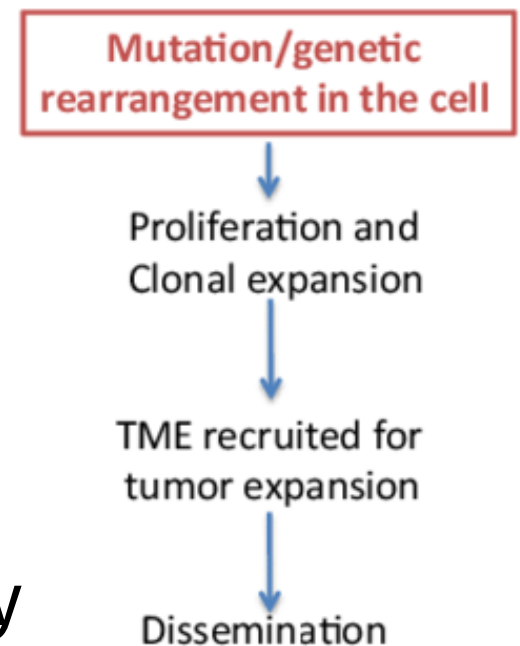


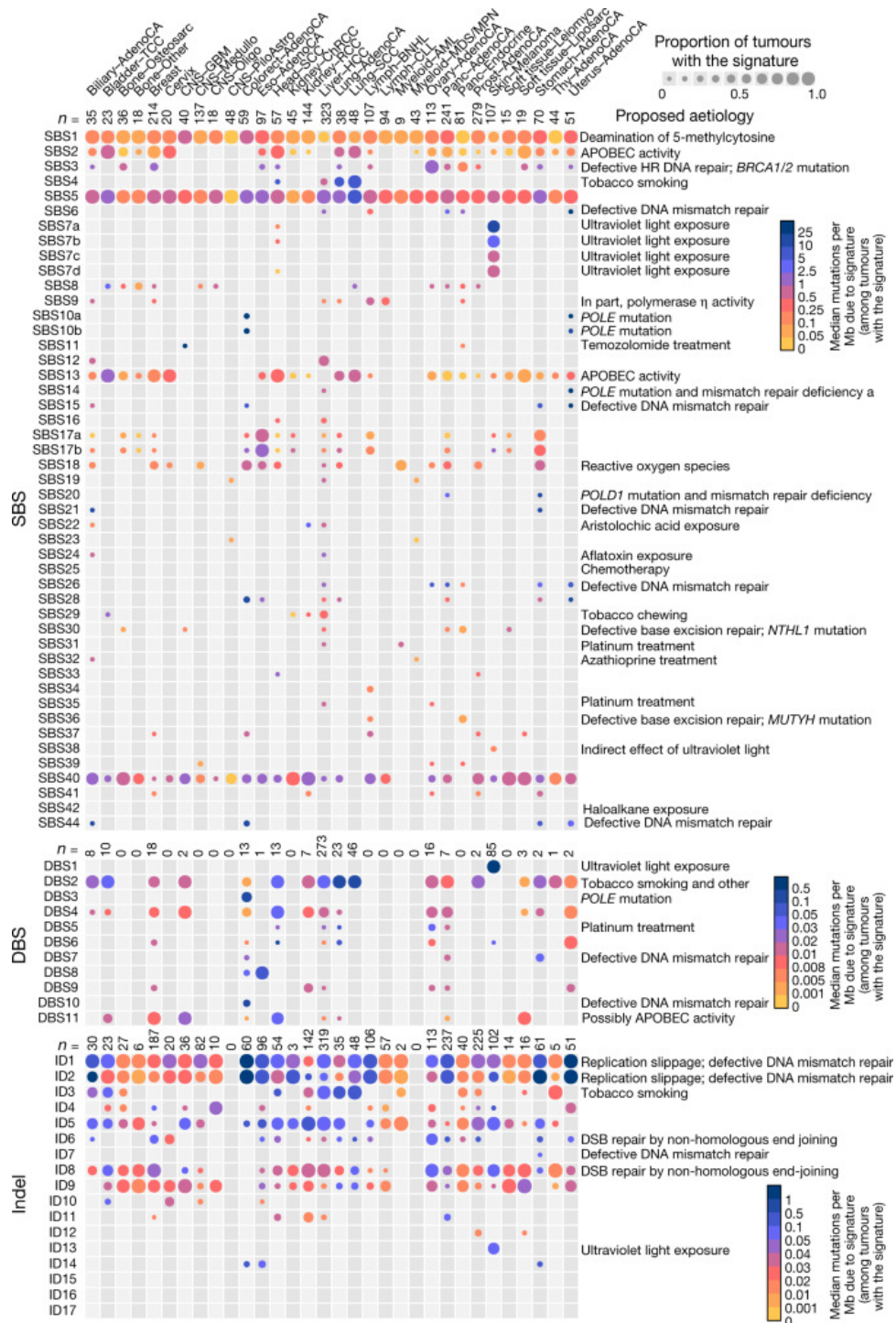
The standard model of cancer origin: the somatic mutation theory

Some **successes** (like hereditary susceptibility, or the prevention of mutagenic chemicals or radiation, genetic therapies, etc.)

Many **contradictions** under the proverbial carpet (cancers without mutations, cancers originated by foreign objects or by no mutagenic chemicals, normal tissues with many mutations, cancer reversal, ...)

SMT





No relevant genetic signatures for mutations in different cancer types (even after huge investment)

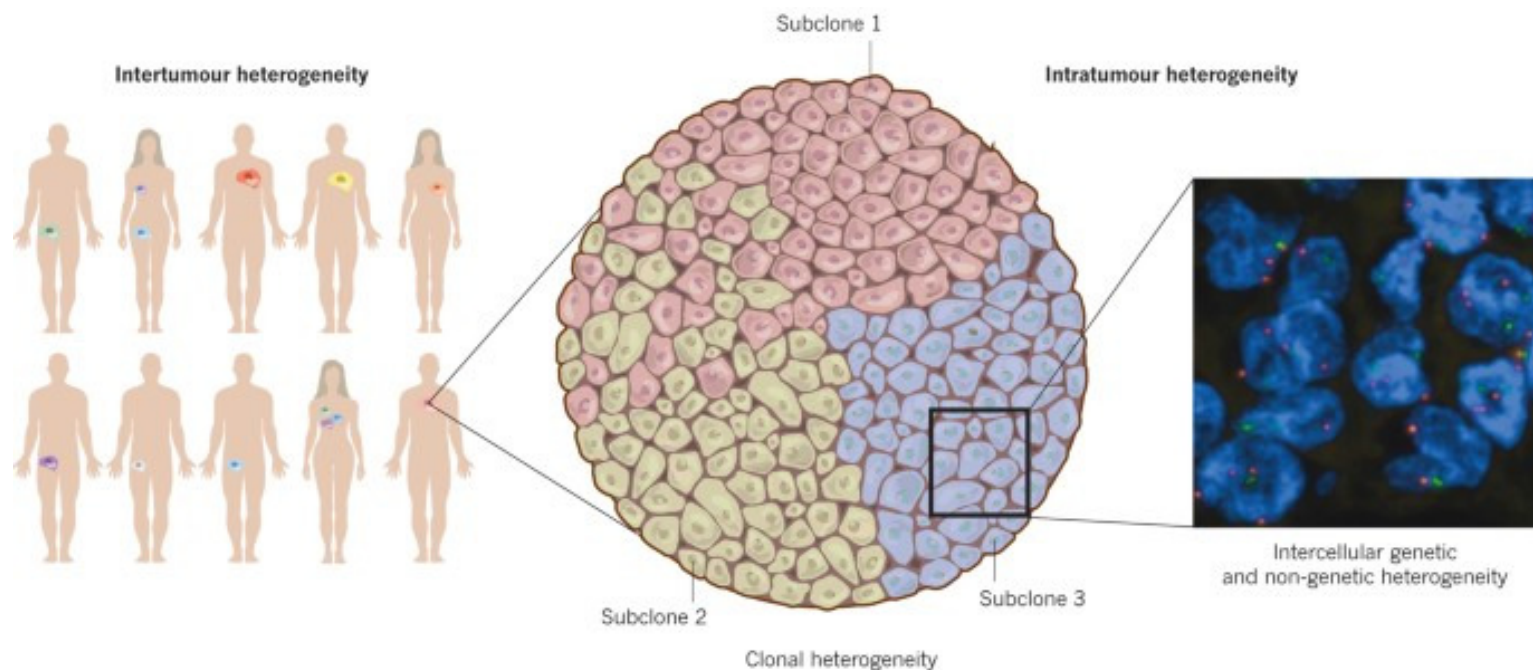
Very **heterogeneous**, even in a single tumor in a particular patient

Alexandrov, L.B., Kim, J., Haradhvala, N.J. *et al.* The repertoire of mutational signatures in human cancer. *Nature* **578**, 94–101 (2020). <https://doi.org/10.1038/s41586-020-1943-3>

No two tumors are genetically equal

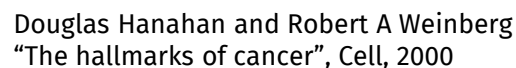
Huge genetic **heterogeneity**, even in the same tumor tissue

No coherent theory guides the results interpretation



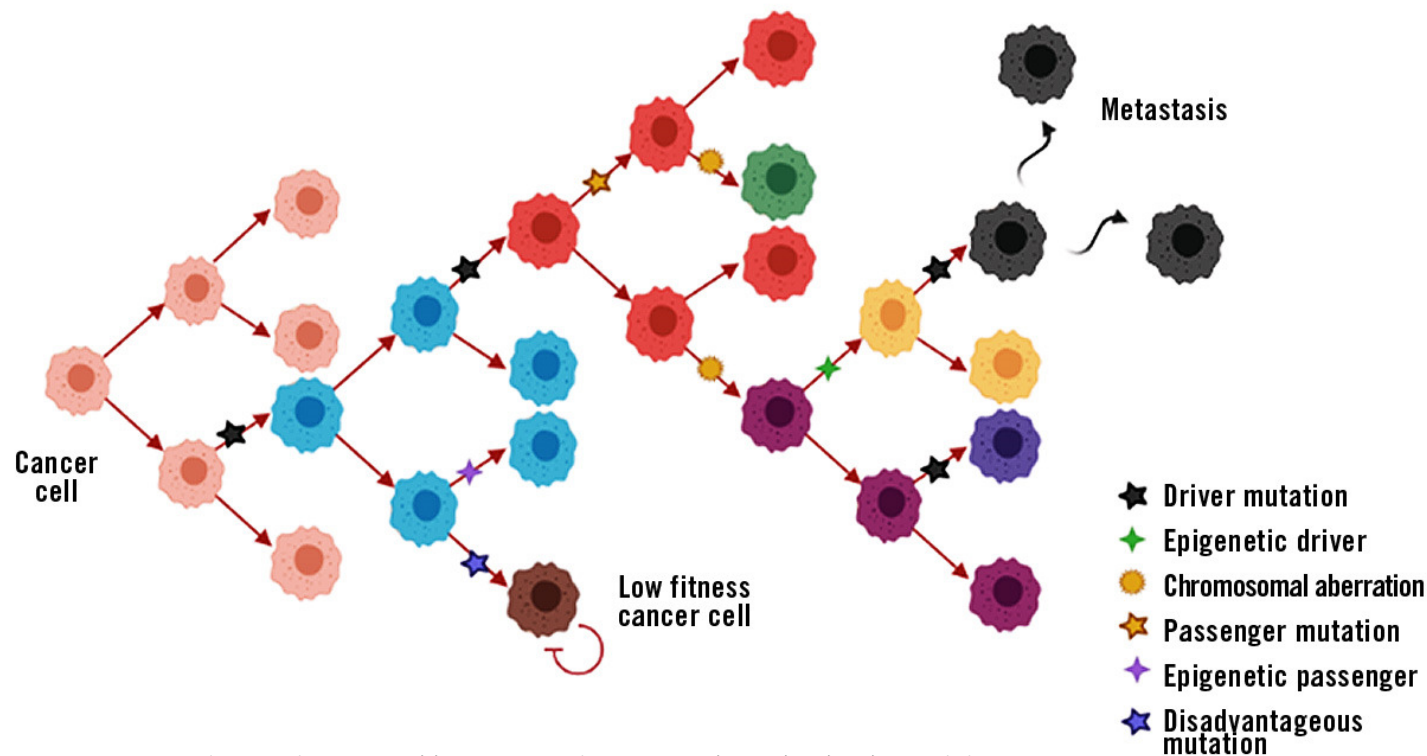
Burrell, R., McGranahan, N., Bartek, J. *et al.* The causes and consequences of genetic heterogeneity in cancer evolution. *Nature* **501**, 338–345 (2013). <https://doi.org/10.1038/nature12625>

In the absence of a reliable **theory**, and when (almost) all results are possible, publish the ones that better fit your (and the editor) prejudices...



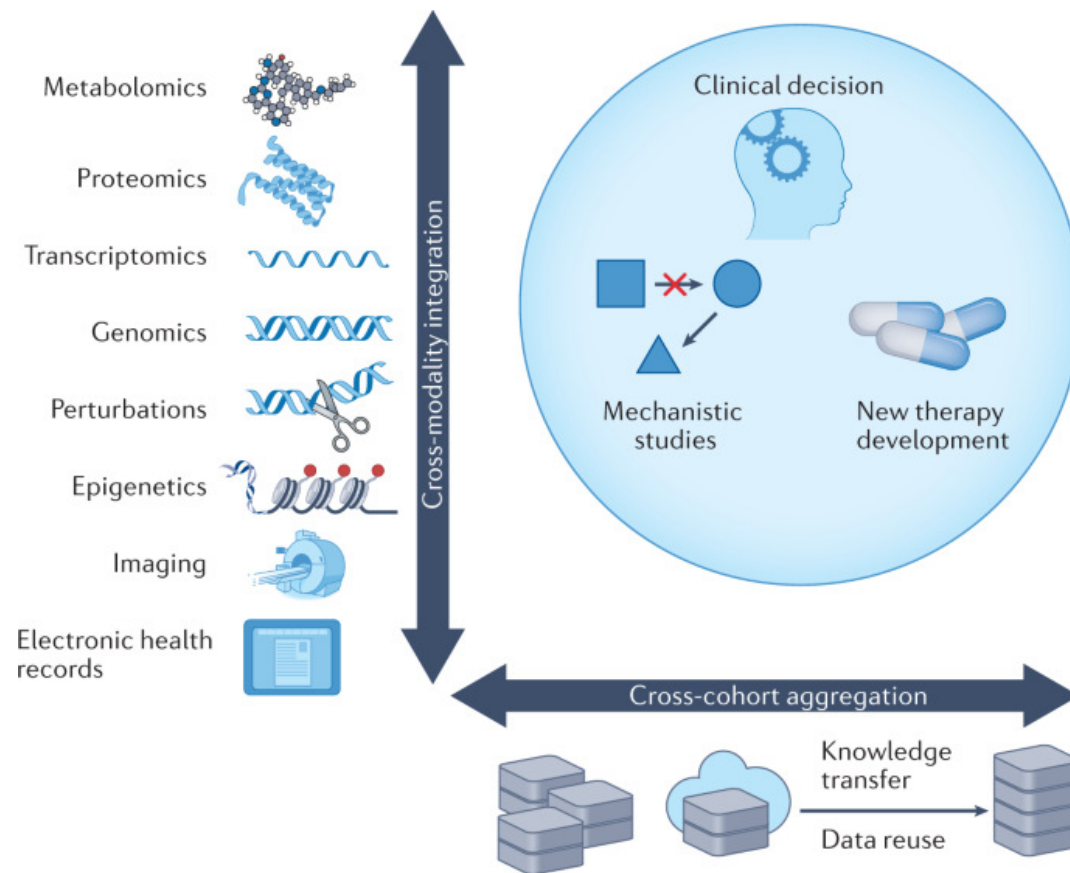
Houdini-like escapes, as driver and passenger mutations (Ugh!)

Every justification for each contradiction found in data is reasonable per se (but all together are a mess!)



What to do about the (unexplained) problems?

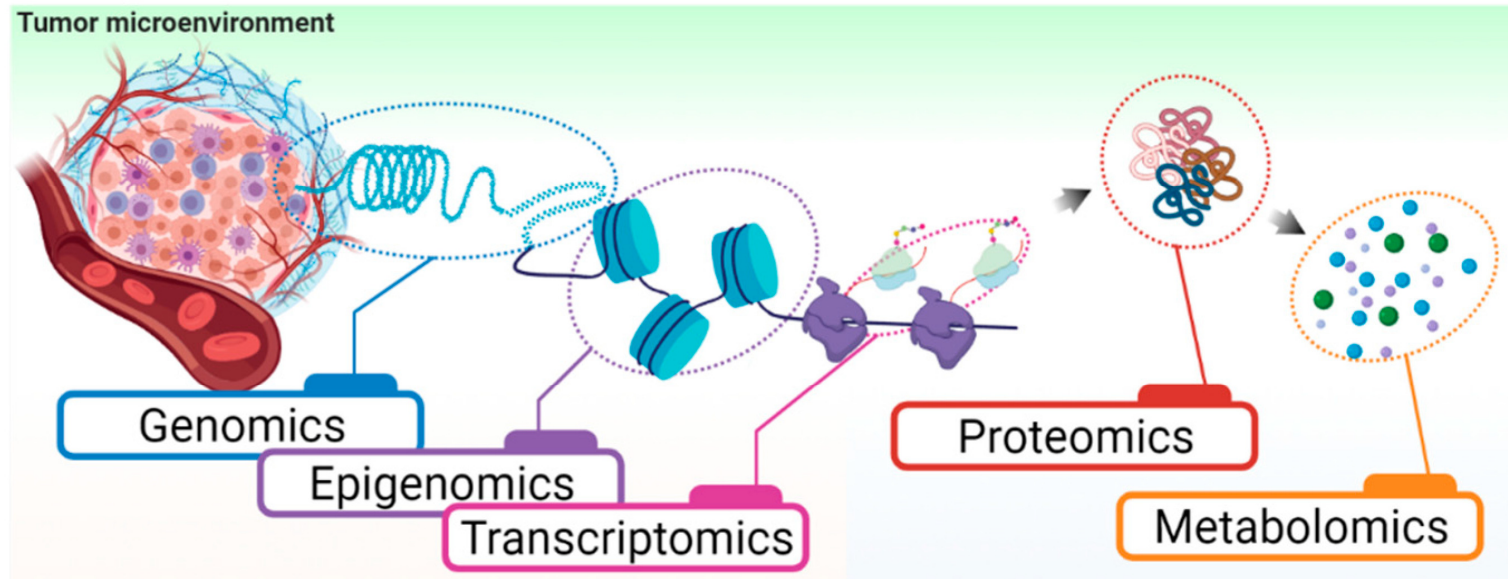
If something doesn't work, put (much) more **resources** on it... (actually almost all resources!)



Jiang, P., Sinha, S., Aldape, K. *et al.* Big data in basic and translational cancer research. *Nat Rev Cancer* **22**, 625–639 (2022).
<https://doi.org/10.1038/s41568-022-00502-0>

A fantastic technology was developed for genomic sequencing and it is the prime tool to acquire (huge amounts of) data on cancer (**omics** revolution)

Problem: more exception cases than real (or useful and usable) predictions



This is a huge and long **mutational** bet (all eggs in a very fragile basket)

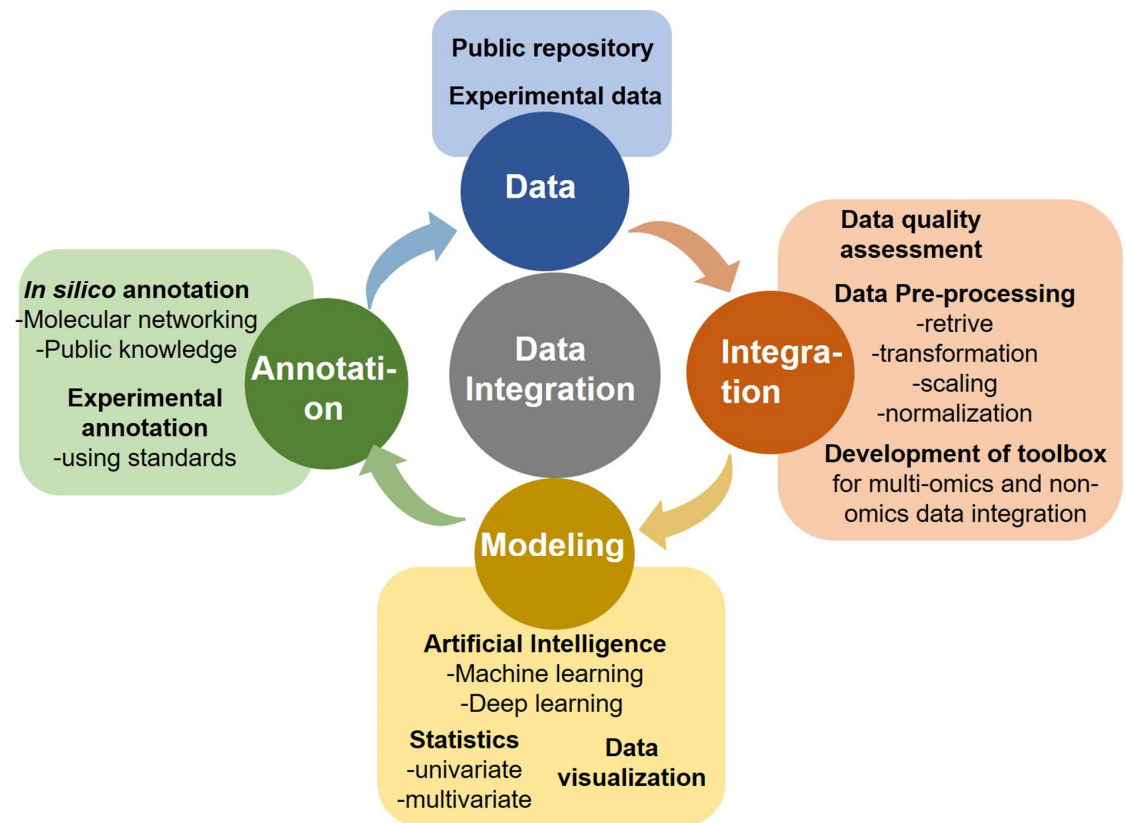
(Important) **biases** in experiments design, results interpretation and accepted publications



Great AI life sciences challenge
















What can be learned from so many millions of experiments and publications?

Put together all the in vitro, in vivo and clinical trials and try to extract useful information and patterns



However, important negative or contradictory results don't see the daylight (or are overlooked)

Problem if the published information is limited by **biases...**

Groupthink  <small>The team wondered if he would ever fit in.</small>	Belief Bias 	Courtesy Bias  <small>Courtesy</small>	Anchoring Effect 	Availability Heuristic 
Bandwagon Effect 	Status Quo Bias 	Gambler's Fallacy 	Ostrich Effect 	Illusion Of Validity 
Reactive Devaluation 	Demotivating Effect 	Pygmalion Effect  <small>GOOD BAD</small>	Bystanders Uncertainty 	Confirmation Bias 

But, are there alternatives?



Promotional still from "Crossroads", in <https://owlcation.com/humanities/The-Crossroads-a-liminal-setting-for-occult-and-supernatural-activities>

And if genetic
mutations found in
cancer tissues are not
a cause but a
consequence of the
cancer progress?

Beware: controversial views

Health and wealth (there are HUGE commercial interests involved)!

Follow the (research and clinical) **money**

Please do not rock the very influential health research boat...

Follow the Money : Funding Research in a Large Academic Health Center,
Bourne, Henry R., Vermillion, Eric B., University of California Health Humanities
Press, 2016 ISBN 10: 0996324216



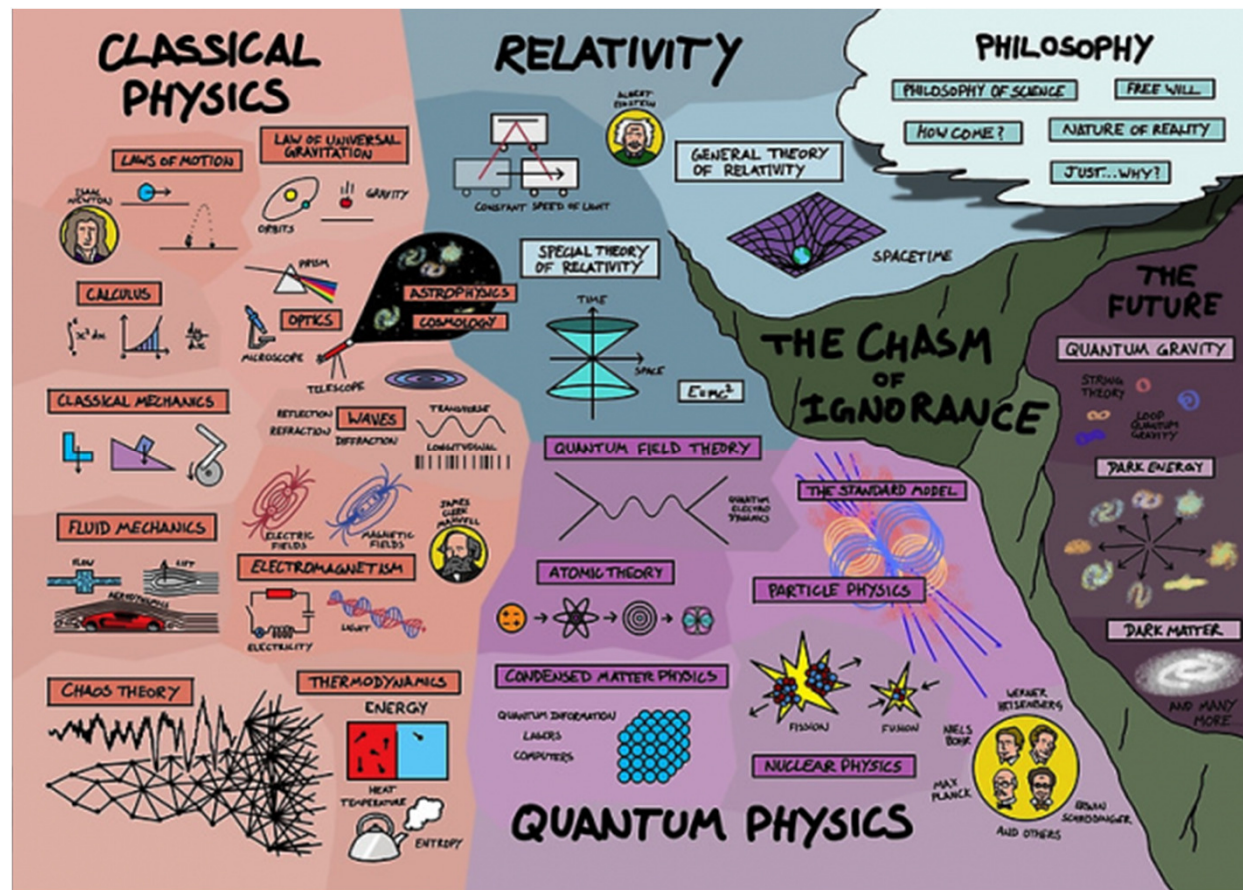
Funding constraints and career advancement obstacles (if not following the herd thinking!)

Please keep (your research) inside the box!



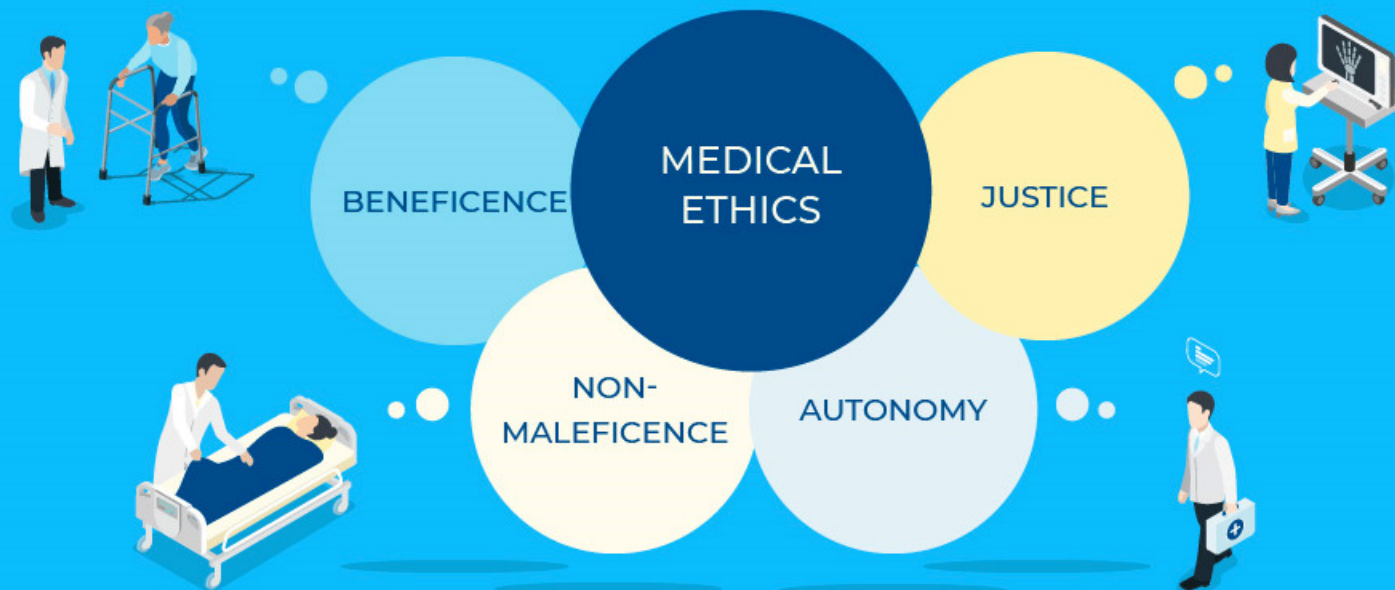
Conservation laws in medicine (but not what you are thinking about)

Why not support more broad views and speculative ideas, as in Physics?



There are very important (and required) **ETHICS** barriers in medical research (to avoid unreasonable risks)

But, what about the fundamental importance of **contrary** thinking for scientific development?



A fluorescence microscopy image showing a cluster of cells. The cells are stained with two different fluorescent dyes, one green and one red, which highlight different cellular components. The background is black, making the brightly colored cells stand out. The text is overlaid on the image in a white, serif font.

My opinion

**Cancer: the triumph of life
against adversity!**

**No cancer cell exist, (initially)
cells in a tumor are just
normal ones running riot**

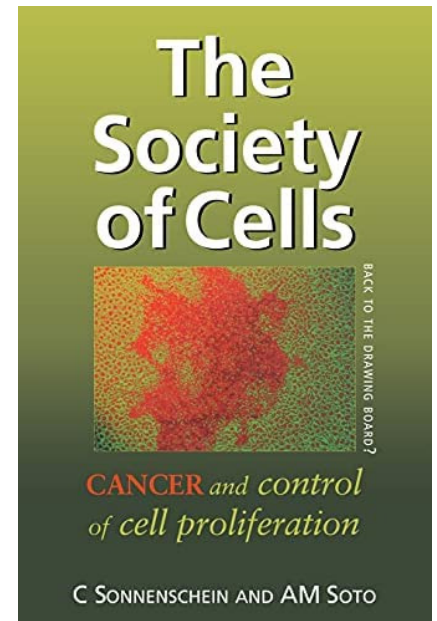
Tissue Organization Field Theory

(TOFT) by Carlos Sonnenschein and Ana M Soto



Cancer as a tissue **organization** problem, not a cell-centered issue (you'll never solve a traffic jam by knowing everything about a car...)

Endorse a theory-guided research



A microscopic image showing several cells with blue and red fluorescence. The cells are irregular in shape and have a granular texture. The background is dark with some red and blue streaks.

Main premise: cells default state is proliferation (biological evidence)

Cancer is due to disruption of proliferation **control** mechanisms (chemical, physical, ...)

RESEARCH ARTICLE | 15 MARCH 2004

The stroma as a crucial target in rat mammary gland carcinogenesis **FREE**

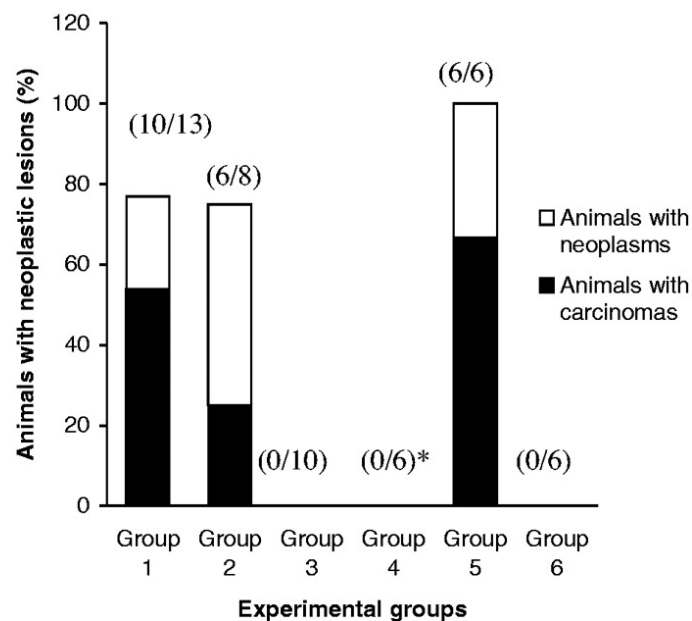
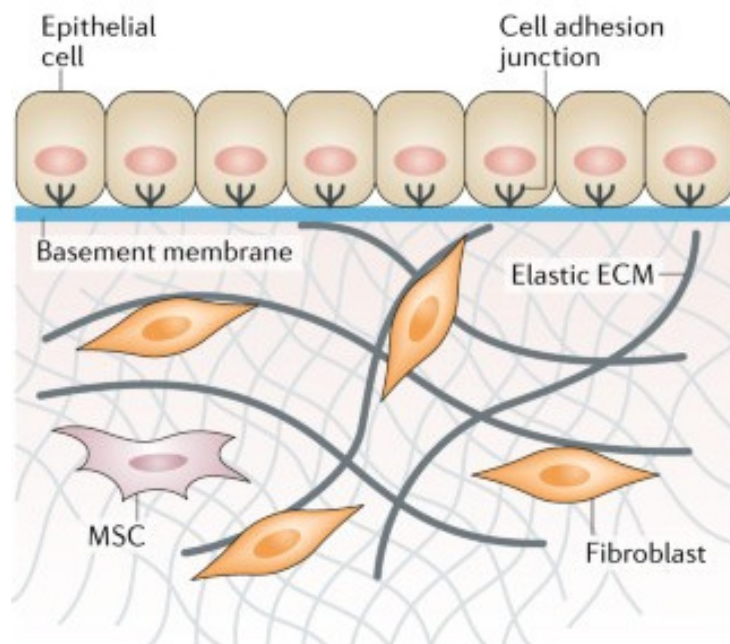
Maricel V. Maffini, Ana M. Soto , Janine M. Calabro, Angelo A. Ucci, Carlos Sonnenschein

+ [Author and article information](#)

J Cell Sci (2004) 117 (8): 1495–1502.

<https://doi.org/10.1242/jcs.01000> [Article history](#) 

Simple **experiment**: rat mammary tissue recombination model (with the chemical carcinogen N-nitrosomethylurea (NMU)) to determine whether the primary target of the carcinogen is the epithelium, the stroma or both tissue compartments



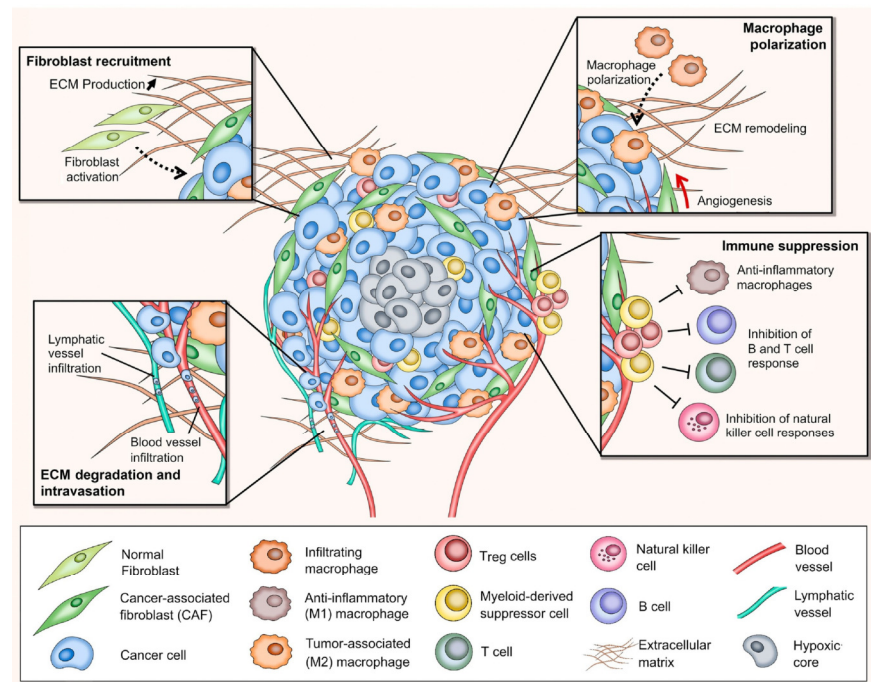
Group	Epithelial	Stroma
1	Vehicle	NMU
2	NMU	NMU
3	NMU	Vehicle
4	Vehicle	Vehicle
5	NMU	
6	Vehicle	

Conclusion: the carcinogen in the environment (stroma fat pad) is at the cancer origin, independent of epithelial cells exposure

Consequences

Move from a cell-centered research to a more holistic approach

Discover the **tissue organization** mechanisms (fundamental for cancer prevention and therapy)

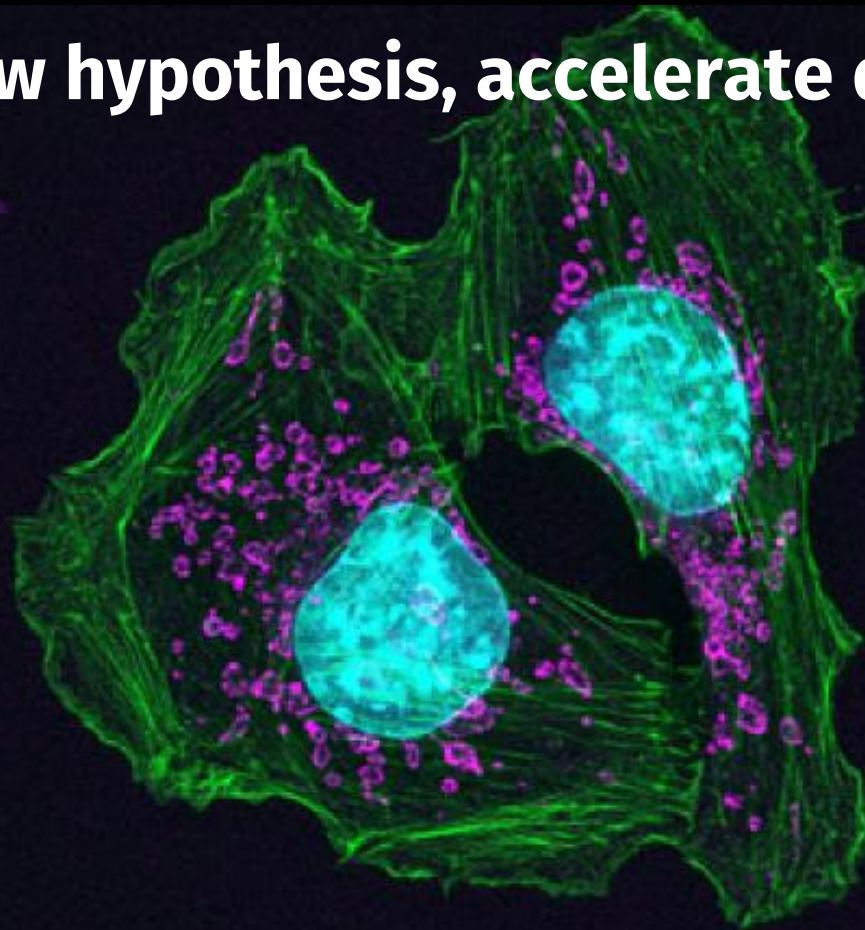


Rodrigues J, Heinrich MA, Teixeira LM, Prakash J. 3D In Vitro Model (R)evolution: Unveiling Tumor-Stroma Interactions. Trends Cancer. 2021;7(3):249-264. doi: 10.1016/j.trecan.2020.10.009.

A theory-guided research allows for its proof or disproof (scientific method)

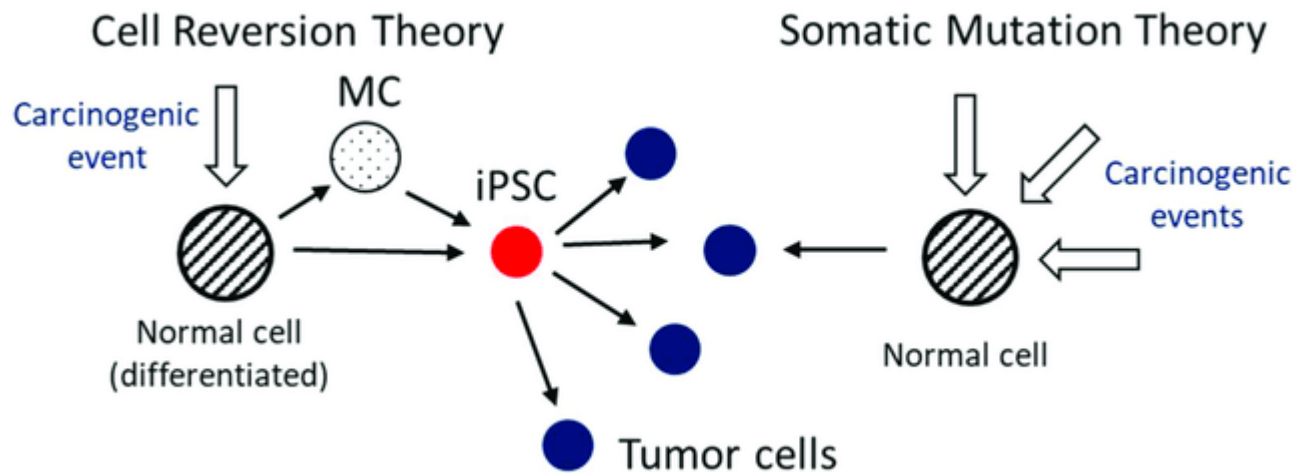
Avoid the tunnel vision and ad-hoc explanations...

Try new hypothesis, accelerate discoveries



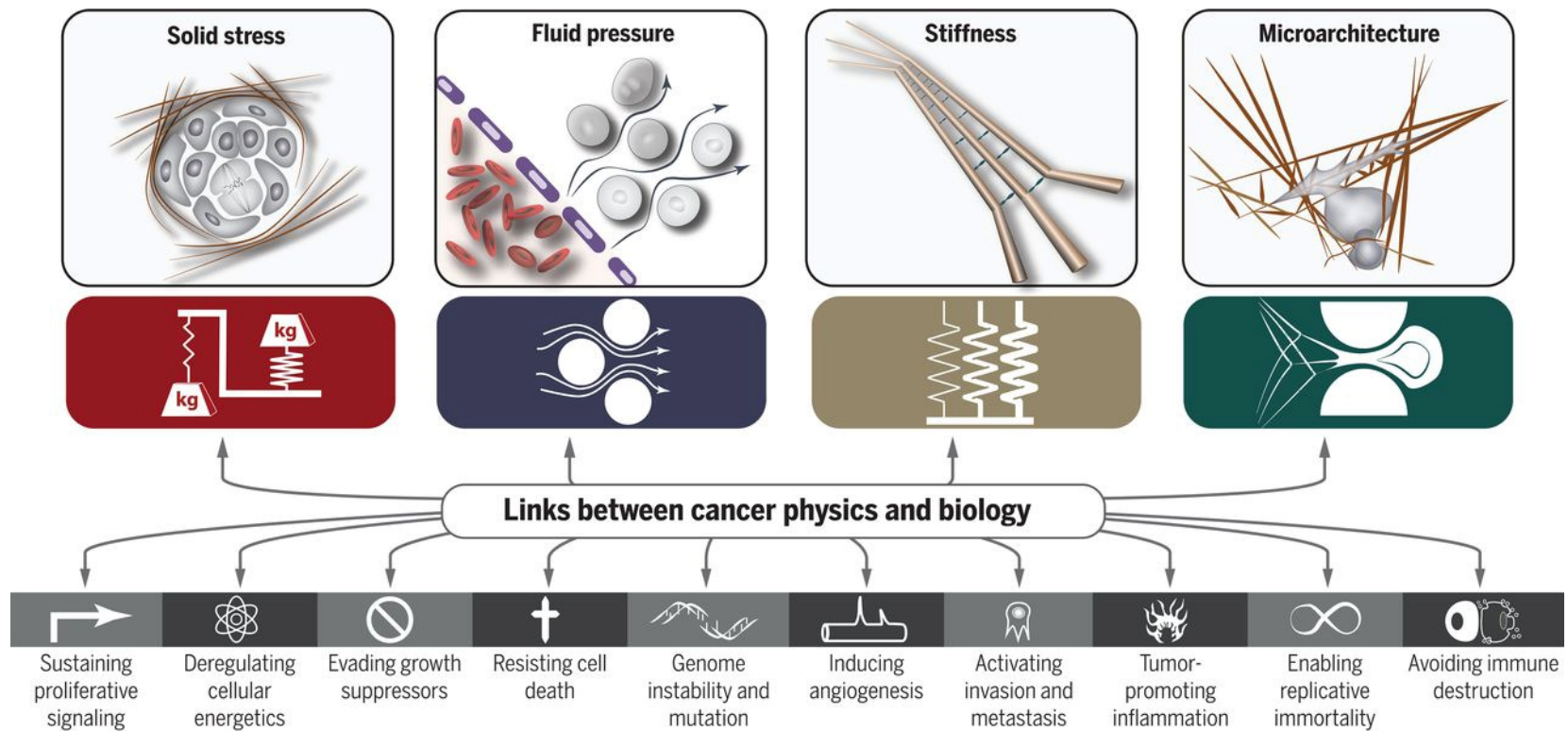
There are other **alternative** theories of cancer initiation, but none of them is mainstream or being actively and intensively tested, as

- Detached pericyte hypothesis
- Brücher–Jamall paradigm (chronic inflammation)
- Cell reversal theory

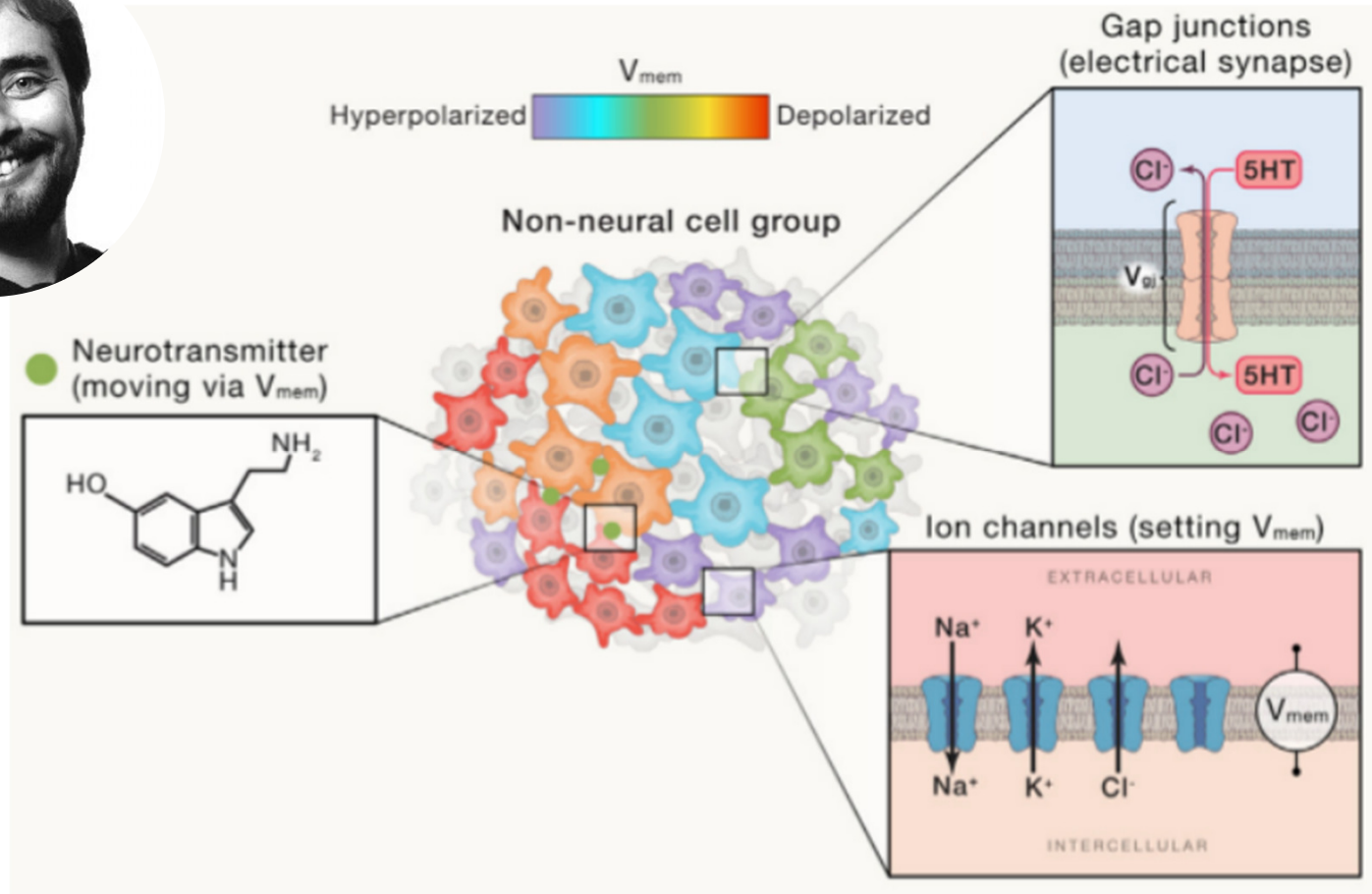


What are the consequences for (our) research at the CFisUC's Soft and Biological Matter group?

Where can **Physics** contribute?



Michael Levin et al. Bioelectricity Lab at the Tufts University (regeneration and cancer)



Levin M. Bioelectric signaling: Reprogrammable circuits underlying embryogenesis, regeneration, and cancer. *Cell*. 2021;184(8):1971-1989. doi: 10.1016/j.cell.2021.02.034.

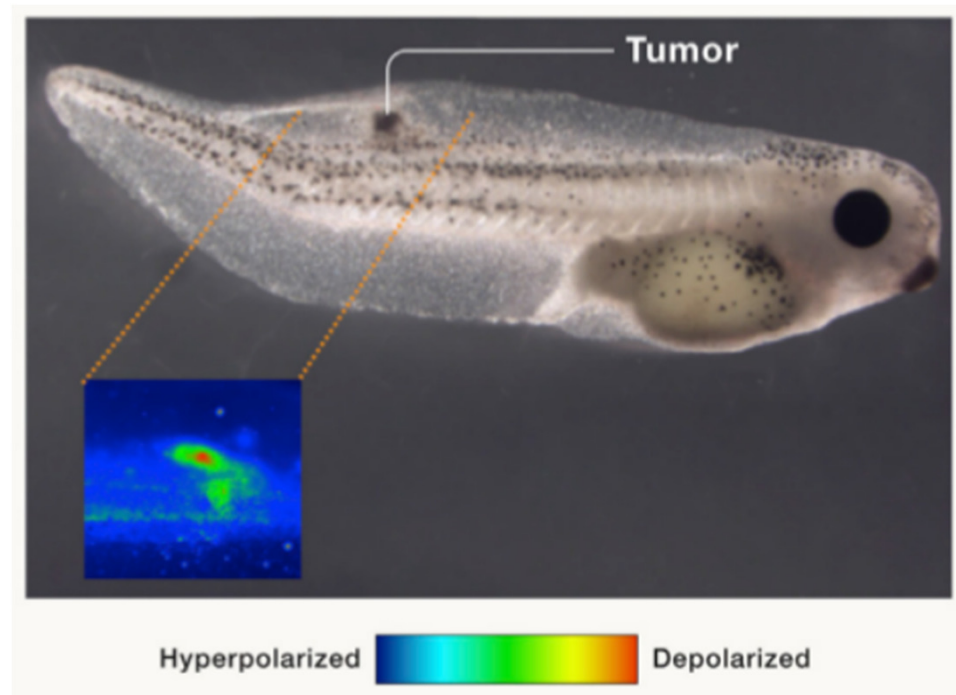
The bioelectric hypothesis

(a clear bias, because we are physicists...)

The **Tissue organizing field** is the cells bioelectric communication and electric environment

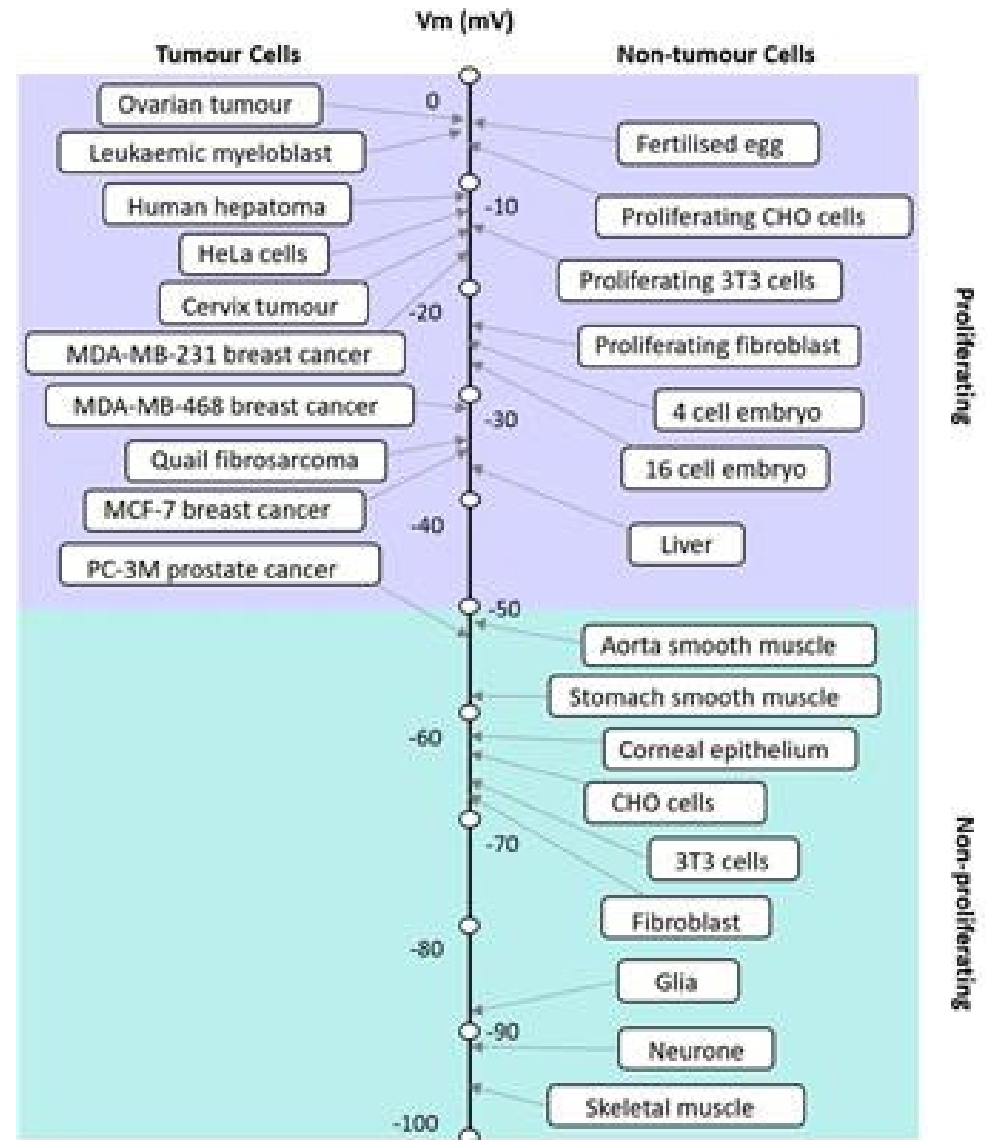
Involved in genomic regulation, as cell proliferation

Levin M. Bioelectric signaling: Reprogrammable circuits underlying embryogenesis, regeneration, and cancer. Cell. 2021;184(8):1971-1989. doi: 10.1016/j.cell.2021.02.034.



(Hyper)polarized cells are quiescent and depolarized cells are proliferative

Hypothesis: cancer initiation by cell membrane depolarization, which propagates in the tissue?

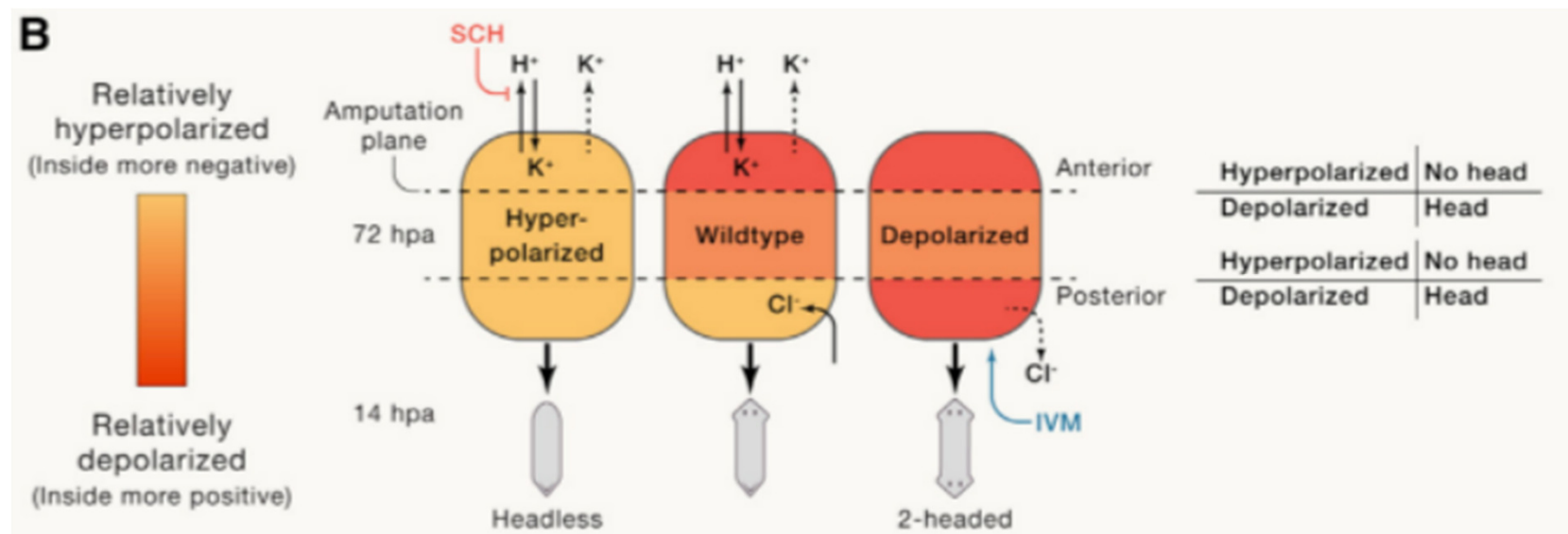


Performed fundamental regeneration experiments with planarian worm



Change of tissue bioelectric **polarization** state by ionic channels manipulation drives the organism development

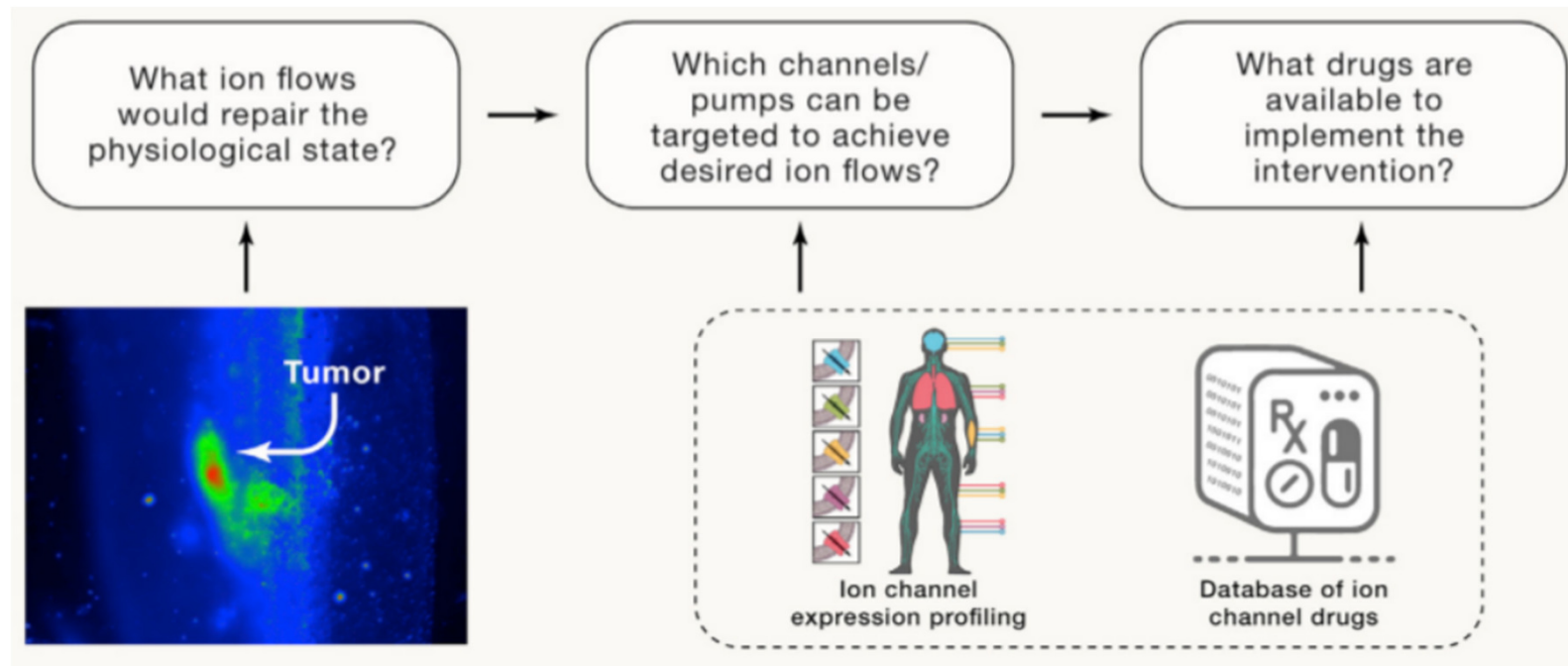
<https://en.wikipedia.org/wiki/Planarian>



Levin M. Bioelectric signaling: Reprogrammable circuits underlying embryogenesis, regeneration, and cancer. Cell. 2021;184(8):1971-1989. doi: 10.1016/j.cell.2021.02.034.

The good news: the manipulation of ionic channels, pumps and gap junctions can change the cells/tissue polarization state

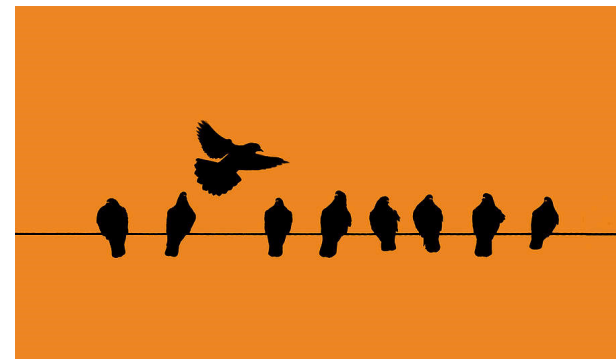
If bioelectricity is at the origin of cancer, its state is **reversible** (at least initially, electroceuticals)



Bioelectric research in CFisUC

Successive submission of exploratory projects on this area to Portuguese FCT (need a multidisciplinary evaluation)

The challenge of not fitting in the box (or in the right panel)



Developing collaboration with FMUC (Filomena Botelho's Biophysics Lab), DCV (Paulo Rocha's Bioelectric Lab) and CNC (João Peça's Neuronal Circuits and Behavior Lab)



Some publications in cancer and bioelectric models and simulations in different settings

HYPOTHESIS AND THEORY article

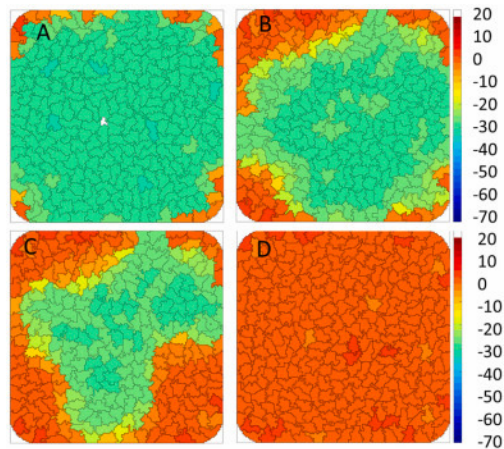
Front. Oncol., 15 April 2020
Sec. Molecular and Cellular Oncology
<https://doi.org/10.3389/fonc.2020.00541>

This article is part of the Research Topic
The Role of Epigenetic Modifications in Cancer Progression
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Cell Reversal From a Differentiated to a Stem-Like State at Cancer Initiation

 João Carvalho*

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Article | [Open Access](#) | [Published: 30 June 2021](#)

A bioelectric model of carcinogenesis, including propagation of cell membrane depolarization and reversal therapies

[Joao Carvalho](#) 

[Scientific Reports](#) **11**, Article number: 13607 (2021) | [Cite this article](#)

Article | [Open Access](#) | [Published: 02 June 2022](#)

A computational model of organism development and carcinogenesis resulting from cells' bioelectric properties and communication

[Joao Carvalho](#) 

[Scientific Reports](#) **12**, Article number: 9206 (2022) | [Cite this article](#)



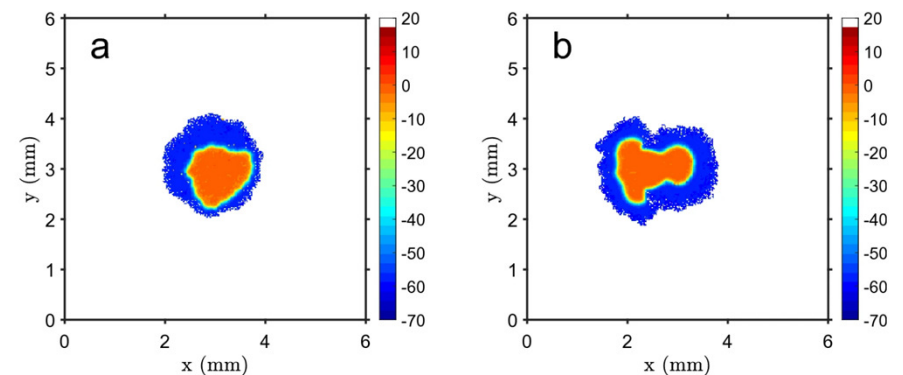
Journal of Theoretical Biology

Volume 557, 21 January 2023, 111338



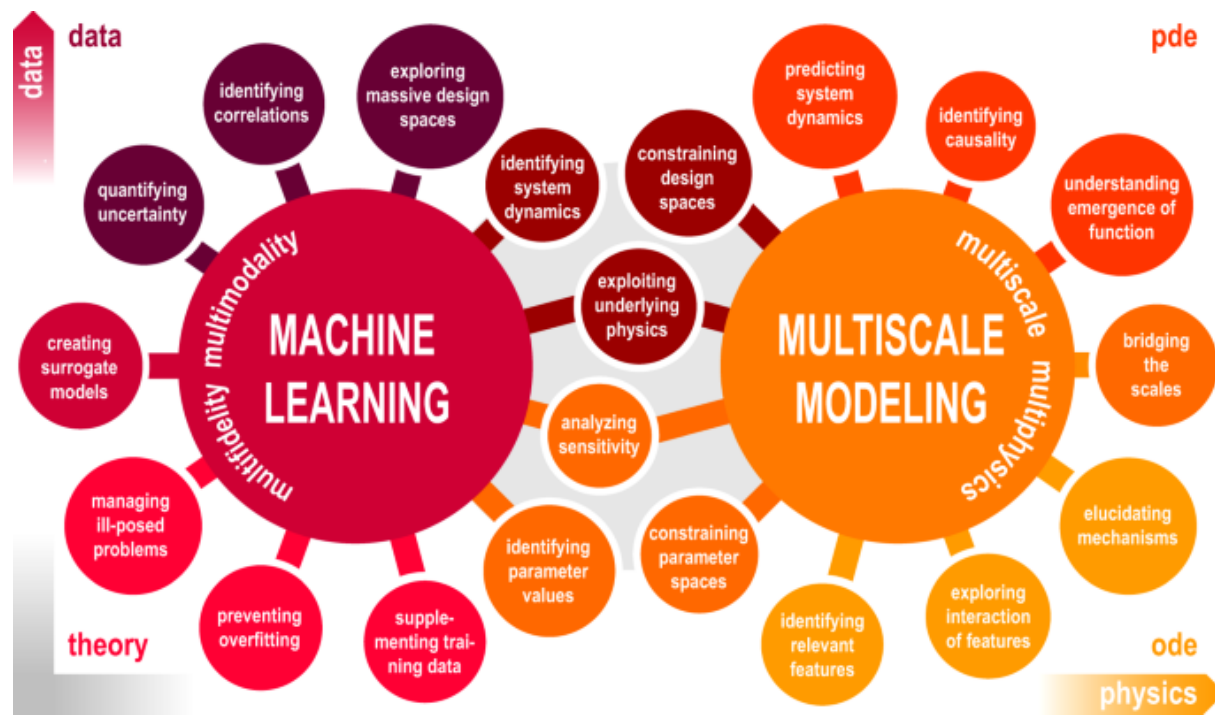
A computational model of cell membrane bioelectric polarization and depolarization, connected with cell proliferation, in different tissue geometries

[Joao Carvalho](#) 



A brave new world of **cancer research** is ahead of us: how and when to move from the current (stagnant) paradigm?

Many obstacles to overcome to **open** cancer research landscape to new visitors and visions



NATIONAL CANCER INSTITUTE PRECISION MEDICINE IN CANCER TREATMENT

Discovering unique therapies that treat an individual's cancer based on the specific genetic abnormalities of that person's tumor.



www.cancer.gov

(Near) future cancer therapies for **precision oncology**:

immunotherapy, genetic vectors... (with help from Artificial Intelligence tools)

But at what cost and with which effectivity? (need to consider its accessibility, secondary effects...)

Take home messages

Cancer research needs a robust
and (very) wide
multidisciplinary approach

Not all hope is lost

A black and white photograph of Richard Feynman standing in front of a large chalkboard covered in complex mathematical and physical equations. He is wearing a light-colored shirt and dark trousers, gesturing with his hands as if explaining a concept. The chalkboard contains various formulas, including wave functions like $\psi(x,t)$, integrals, and terms related to quantum mechanics such as $K(x,x')$. A small white bucket sits on the floor near the board. The overall scene captures Feynman in his role as a physicist and educator.

In health and disease, always think like a physicist

<https://www.cantorsparadise.com/richard-feynman-on-the-differences-between-mathematics-and-physics-c0847e8a3d75>



**‘There is
nothing more
practical than
a good
theory’ Kurt
Lewin**

(actually a psychologist!)

Lewin, K. (1952). *Field theory in social science: Selected theoretical papers by Kurt Lewin*. London: Tavistock.

Kurt Lewin - 1936, Iowa City, from the collection of Tomasz Kardaś, in <https://history.easp.eu/people/lewin-kurt>

CFisUC Rui Travasso, Marcos
Gouveia, António Cardoso



Collaborators

Bioelectricity Lab Paulo Rocha



Biophysics Lab Filomena Botelho,
Margarida Abrantes, Salomé Pires



Neuronal Circuits and Behavior Lab
João Peça, Ana Maria Cardoso



Tufts University Michael Levin,
Carlos Sonnenschein, Ana Soto



MD Anderson Cancer Center Jinsong Liu,
National Cancer Institute Stuart Baker



The background is a deep blue with several out-of-focus, glowing purple and blue circles. Two large, colorful, textured spheres are positioned on the left and right sides. Each sphere has a yellow and orange core with blue and green speckles. A bright yellow line connects the top of the left sphere to the top of the right sphere, and another yellow line connects the bottom of the left sphere to the bottom of the right sphere, forming a frame around the text.

Thank you for your
time and patience!

All questions welcome



<https://www.mdanderson.org/cancerwise.html>