NUS National University of Singapore | Healthy Longevity Translational Research Programme, Yong Loo Lin School of Medicine

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GLOBAL CONFERENCE ON GEROPHYSICS

WHERE PHYSICS MEETS AGEING BIOLOGY

GLOBAL CONFERENCE ON GEROPHYSICS (#GCGP1ST) 2025

5 - 6 March 2025

Paradox Singapore Merchant Court





BIOAGE VitaDAO genomax Technologies nature aging

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WELCOME MESSAGE

We are thrilled to have you join us in Singapore for this groundbreaking event where physics meets ageing science. Over the next two days, we will dive into innovative research, explore new ways to extend healthspan and connect with experts shaping the future of longevity.

This is more than just a conference—it's a chance to spark new ideas, build collaborations and push the boundaries of what's possible in ageing research. So, get ready for insightful talks, engaging discussions and meaningful connections.

Let's make the most of it!

From the GCGP Organizing Team

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WHY THIS CONFERENCE MATTERS ?

Ageing biology now stands at a turning point. Robust models of cellular senescence, DNA damage repair, proteostasis, and metabolic regulation can, when combined with rigorous physical theories and mathematical modeling, shed new light on the fundamental laws governing life's progression and decline.

We believe the time is right to apply the powerful toolkit of theoretical physics to one of humanity's greatest challenges: **understanding and controlling the ageing process**.

The goal is not only to identify parallels and common frameworks but also to forge long-term collaborations.

CONFERENCE SCHEDULE

DAY 1: 5th March

Registration + Light Refreshments 08.45 - 09.30

Welcome Remarks 09.30 - 09.45

Session 1: The Emergence of Gerophysics: Integrating Physical Laws into Ageing Science 09.45 - 11.00

Chaired by: Prof. Jan Gruber

Prof. Uri Alon Using physics-style math models to dissect core drivers of ageing (Long Talk)

Prof. Marija Cvijovic The Ageing Game: Why Gilgamesh Should Have Studied Physics and Math (Long Talk)

Dr. Yifan Yang Compression of morbidity by interventions that steepen the survival curve (Long Talk)

Group Picture + Coffee Break + Exhibition Viewing 11.00 - 11.30

Session 2: Fundamental Principles: Physics-Derived Models for Understanding Ageing 11.30 - 13.00

Chaired by: Prof. Ee Hou Yong

Dr. Michael Rera Ageing as a two-phase process. Reinterpreting hallmarks of ageing and its evolution (Long Talk)

Dr. Peter Fedichev Beyond Hallmarks: A Thermodynamic Framework for Radical Lifespan Extension (Long Talk)

Prof. Jan Gruber Rethinking Ageing: Effective Temperature Concept Resolves Perplexing Ageing Data (Long Talk)

Mr. Ben Shenhar Heritability of human lifespan in the light of the saturated removal model of ageing (Short Talk)

Lunch Break + Exhibition Viewing 13.00 - 14.00

Long Talk is 20 MIN + 5 MIN Q&A
Short Talk is 10 MIN + 2 MIN Q&A

Session 3: Synergizing Physics and AI: Computational Approaches to Ageing Biology — 14.00 - 15.30

Chaired by: Prof. Brian Kennedy

Dr. Kumar Selvarajoo Can predictive models be developed for understanding complex ageing cellular dynamics? (Long Talk)

Prof. Matt Kaeberlein Beyond the Hallmarks: What are we missing in ageing research? (Long Talk)

Prof. Morten Scheibye-Knudsen Computational approaches to target ageing (Long Talk)

Dr. Andrei Tarkhov No easy answers - AI-assisted protein design for lifespan extension (Short Talk)

Coffee Break + Exhibition Viewing — 15.30 - 16.00

Session 4: Short Talks and 1 Min Pitches — 16.00 - 17.15

Chaired by: Dr. Max Unfried

Dr. Weilan Wang Use of potential gerotherapeutic drugs and mortality of geriatric rehabilitation inpatients (Short Talk)

Dr. Glen Pridham Dynamical modelling of the frailty index indicates that health reaches a tipping point near age 75 (Short Talk)

Mr. Kamil Pabis Analysis of individual mouse survival data from large databases – insights and findings (Short Talk)

1-Minute Pitches

Session 5: Whiteboard and Poster Session (with refreshments) — 17.15 - 19.00

Thematic Poster Session — 17.15 - 18.15

Networking and Performance
Songs and Tales from Academia — 18.15 - 19.00

Long Talk is 20 MIN + 5 MIN Q&A
Short Talk is 10 MIN + 2 MIN Q&A

CONFERENCE SCHEDULE

DAY 2: 6th March

Registration + Light Refreshments 08.00 - 08.30

Session 6: Stochasticity and Dynamics of Biomarkers & Ageing Clocks 08.30 - 10.00

Chaired by: Prof. Vadim Gladyshev

Prof. Andrew E Teschendorff Physics in Ageing Biology: from applications to fundamental theories (Long Talk)

Prof. Steffen Rulands Stochasticity and memory in epigenetic ageing (Long Talk)

Prof. Andrew Rutenberg Dynamics of Ageing Biomarkers (Long Talk)

Dr. Dmitrii Kriukov Estimating Uncertainty in Biological Age Prediction: A Fundamental Challenge (Short Talk)

Coffee Break + Exhibition Viewing 10.00 - 10.30

Session 7: Lifelong Dynamics: Insights into Developmental Processes and Ageing Patterns 10.30 - 12.00

Chaired by: Prof. Koh Woon Puay

Prof. Vadim Gladyshev Insights into ageing, longevity and rejuvenation (Long Talk)

Prof. Nir Eynon Dynamic ways of quantifying the human ageing methylome and exercise rejuvenation (Long Talk)

Dr. Wang Haiyang Rejuvenating aged oocytes for female reproductive longevity (Long Talk)

Dr. Leong Kim Whye Critical phenomenon associated with luminogenesis during ovarian follicle development (Short Talk)

Lunch Break + Exhibition Viewing 12.00 - 13.00

Long Talk is 20 MIN + 5 MIN Q&A
Short Talk is 10 MIN + 2 MIN Q&A

Session 8: Complex Systems and Connectivity: A Network Approach to Ageing & Metabolism — 13.00 - 14.15

Chaired by: Dr. Kumar Selvarajoo

Prof. Ee Hou Yong Understanding Evolution, Ageing, and Repair using Complex Network (Long Talk)

Prof. Feng Ling Percolation theory in complex networks (Long Talk)

Mr. Weihan Huai Identifying Novel Geroprotectors through Ageing Network Mapping (Short Talk)

Dr. Csaba Kerepesi Methylation and network entropy measurements during ageing (Short Talk)

Coffee Break + Exhibition Viewing — 14.15 - 14.35

Session 9: Quantitative Insights into Metabolism and Longevity — 14.35 - 15.40

Chaired by: Asst. Prof. Yifan Yang

Dr. Peter James Mullen Metabolic drivers of species ageing (Short Talk)

Dr. Max Unfried A Network Perspective on Comparative Lipidomics and Lifespan Regulation (Short Talk)

Dr. Yumi Kim Age-dependent remodeling of ribosomes in skeletal muscle: Structural and functional changes (Short Talk)

Prof. Brian Kennedy Unanswered Questions in Ageing Research (Long Talk)

Short Break — 15.40 - 16.00

Session 10: Panel on The Future of Gerophysics — 16.00 - 16.40

Moderator: Dr. Sebastien Thuault
Panelists: Prof. Uri Alon, Prof. Peter Fedichev, Prof. Marija Cvijovic, Prof. Jan Gruber

Wrap up: Dr. Max Unfried & Prof. Brian Kennedy — 16.40 - 17.00

Speakers and Sponsors Dinner — 19.00 - 22.00

Long Talk is 20 MIN + 5 MIN Q&A
Short Talk is 10 MIN + 2 MIN Q&A

LONG TALKS


Prof. Uri Alon, Israel


Prof. Vadim Gladyshev, USA


Prof. Marija Cvijovic, Sweden


Prof. Matt Kaeberlein, USA


Prof. Andrew E Teschendorff, China


Prof. Andrew Rutenberg, Canada


Dr. Peter Fedichev Singapore


Asst Prof. Feng Ling, Singapore


A/Prof. Morten Scheibye-Knudsen, Denmark


Dr. Wang Haiyang, Singapore


Asst Prof. Yong Ee Hou, Singapore


Dr. Kumar Selvarajoo, Singapore


Prof. Steffen Rulands Germany


Dr. Michael Rera, France


Dr. Yifan Yang, China


Prof. Nir Eynon, Australia


A/Prof. Jan Gruber, Singapore


Prof. Brian Kennedy, Singapore

SHORT TALKS


Dr. Weilan Wang


Mr. Ben Shenhar


Dr. Csaba Kerepesi


Mr. Weihan Huai


Dr. Maximilian Unfried


Dr. Leong Kim Whye


Dr. Yumi Kim


Dr. Peter J Mullen


Mr. Kamil Pabis


Dr. Dmitrii Kriukov


Dr. Glen Pridham


Dr. Andrei Tarkhov

HOW TO GET TO THE VENUE FROM THE SINGAPORE CHANGI AIRPORT?

The Venue, 5 - 6 March

Paradox Singapore Merchant Court (Ballroom), see map.

Local taxi

Takes around 25 minutes and costs ~ 25 - 40 SGD

Public transport

Takes around 1 hour and costs ~ 2 - 4 SGD

From Changi Airport, take the Green Line (East-West Line) to Expo station (one stop), then transfer to the Blue Line (Downtown Line) to Fort Canning station

VENUE INFORMATION

PARADOX SINGAPORE, MERCHANT COURT BALLROOM

Conference Hall, 5 - 6 March 2025

Address: 20 Merchant Rd, Singapore 058281

We have secured a special rate of 250++ per night for our attendees. Book Your room here

EMPRESS RESTAURANT

Speakers and Sponsors Dinner*, 6 March 2025

1 Empress Place,
Asian Civilisations Museum,
#01-03, Singapore 179555

By Invite Only*

Recommended Dress Code: **Smart Casual**

CULTURAL TIPS AND LOCAL INSIGHTS

LOCAL FOOD

Try Traditional Hainanese Chicken Rice at Hawker Center
HERE

Try Bak Kut Teh at Song Fa, ideally with some friends
HERE

Try Chilli Crab
HERE

LOCAL FOOD (Vegetarian)

Try Local Vegetarian Dishes at D'Life
HERE

Japanese Vegetarian Restaurant, Herbivore
HERE

Vegetarian Fine Dining at Sufood
HERE

BUBBLE TEA

Koi Tea

ChiCha San Chen

Mr Coconut

THINGS TO DO

Visit The Cloud Forest
HERE

Learn about Singapore History during WW2 at Fort Canning battlebox
HERE

Go to the Peranakan Museum
HERE

Take a walk in the beautiful Fort Canning Park
HERE

Let the music and lights entertain you at night HERE

Take a walk around Sentosa Island and enjoy a beach day
HERE

WHERE TO PARTY

Singapore is a really lively city. The best places to party are Clarke Quay, Amoy Street, Haji Lane, Club Street

THE GLOBAL CONFERENCE ON GEROPHYSICS

is honoured to present our

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LONG TALKS

Using physics-style math models to dissect core drivers of ageing

Prof. Uri Alon

The Ageing Game: Why Gilgamesh Should Have Studied Physics and Math

Prof. Marija Cvijovic

One of the key challenges in ageing research is understanding how the events that define cellular ageing are regulated and interconnected. While much is known about individual hallmarks of cellular aging, their complex, multi-scale interactions hinder our progress in gaining deeper insights into the emergent effect on an organism. Experimental methods alone are insufficient for capturing the full intricacy of these networks and their dynamics.

In this talk, I will present our recent efforts in developing computational methods and mathematical models that help disentangle causes and consequences and reveal pathways' synergistic roles in ageing. Additionally, I will discuss how data-driven approaches, as one of the building blocks of gerophysics, can bridge gaps in ageing research by integrating the often-fragmented knowledge and expertise. This interdisciplinary perspective has the potential to transform our understanding of ageing and unlock new strategies to extend healthspan.

Compression of morbidity by interventions that steepen the survival curve

Dr. Yifan Yang

Longevity research aims to extend the healthspan while minimizing the duration of disability and morbidity, known as the sickspan. Most longevity interventions in model organisms extend healthspan, but it is not known whether they compress sickspan relative to the lifespan. Here, we present a theory that predicts which interventions compress relative sickspan, based on the shape of the survival curve. Interventions such as caloric restriction that extend mean lifespan while preserving the shape of the survival curve, are predicted to extend the sickspan proportionally, without compressing it. Conversely, a subset of interventions that extend lifespan and steepen the shape of the survival curve are predicted to compress the relative sickspan. We explain this based on the saturating-removal mathematical model of aging, and present evidence from longitudinal health data in mice, Caenorhabditis elegans and Drosophila melanogaster. We apply this theory to identify potential interventions for compressing the sickspan in mice, and to combinations of longevity interventions. This approach offers potential strategies for compressing morbidity and extending healthspan.

Ageing as a two-phase process. Reinterpreting hallmarks of ageing and its evolution

Dr. Michael Rera

Ageing is a complex process whose evolutionary origins and molecular mechanisms remain largely not characterized. Traditionally seen as a gradual decline following reproductive years, ageing has often been considered a by-product of evolution. However, recent work challenges this view, proposing ageing as a discontinuous process with two distinct and consecutive phases. This distinction is marked by the “Smurf” phenotype, a set of physiological and molecular changes that identify individuals transitioning to a late-life stage associated with mortality and so-called age-related physiological changes. Initially observed in Drosophila, this phenotype is conserved across species, including nematodes, zebrafish, and mice. Studies show that the risk of transitioning between phases follows a polynomial function of age, while mortality in the second phase follows an exponential pattern. This model highlights phase 1 as the primary source of lifespan variability, with transcriptomic data supporting this distinction. This approach offers new insights into the genetic and evolutionary mechanisms governing longevity and ageing plasticity.

Rethinking Ageing: Effective Temperature Concept Resolves Perplexing Ageing Data

Prof. Jan Gruber

The discovery that single-gene mutations can dramatically extend invertebrate lifespans marked a pivotal milestone in geroscience, demonstrating both the plasticity of aging and the existence of master regulators of lifespan. Yet, years of investigating mechanistic “damage-accumulation” (Hallmarks of Aging) models in C. elegans and other invertebrates have yielded results that are sometimes difficult to explain and that can be outright perplexing. However, scientific breakthroughs often arise less from results that neatly confirm expectations but from unexpected or counterintuitive data that challenge prevailing paradigms. In this talk, I will revisit some of the most perplexing nematode aging observations and reinterpret them through the lens of a recently formulated theoretical framework that synthesizes thermodynamics, information theory, and stochastic processes into a coherent phenomenological model. By illuminating how these puzzling results fit within this new framework, we can deepen our understanding of aging mechanisms and more effectively guide future research.

Beyond Hallmarks: A Thermodynamic Framework for Radical Lifespan Extension

Dr. Peter Fedichev

To effectively intervene in aging, it is critical to understand the mechanisms driving mortality acceleration. Current biological models identify multiple hallmarks of aging, none of which can be considered fundamental. In longevity medicine, these hallmarks are often described as age-related diseases. Drugs targeting them are expected to produce modest lifespan extensions, with metabolic disorder treatments offering the largest benefit of approximately 10 years. This complexity necessitates borrowing approaches from physics, particularly statistical mechanics and complex systems theory, to uncover deeper, universal principles governing the aging process. We present a theoretical framework unifying aging mechanisms through thermodynamics, information theory, and stochastic processes. Analysis of DNA methylation data and longitudinal medical records reveals aging as a process driven by numerous independent, infrequent transitions between metastable states in a vast biological configuration space. These rare, high-energy transitions in a rugged energy landscape lead to effectively irreversible system failures across redundant biological structures. The cumulative impact of these transitions is captured by thermodynamic biological age (tBA), a stochastic variable reflecting entropy production and information loss during aging. This stochastic damage signature provides a theoretical basis for the absolute human lifespan limit, estimated at 120-150 years due to complete physiological resilience loss. Aging appears entropic and largely irreversible, with tBA correlating with physiological drift, reduced resilience, and exponential increases in chronic disease risk. However, we propose a fundamental thermodynamic control variable, the "effective biological temperature," regulating the magnitude of molecular fluctuations across biological pathways. Our analysis, supported by methylation data, identified this variable and demonstrated its influence on initial mortality rates and mortality rate doubling time while remaining independent of maximum lifespan. Notably, reducing the effective temperature could extend the average lifespan from approximately 80 years to 120-150 years, far exceeding the impact of drugs targeting age-related diseases. This work offers the first comprehensive theoretical model of aging, integrating entropic biological aging processes with dynamic resilience loss. It defines a clear path for substantial lifespan extension by targeting thermodynamic and informational drivers of aging, shifting focus from symptom management to the root causes of age-related decline. These insights will be presented at the Eureka Conference on Aging, emphasizing how a deeper understanding of these principles could drive transformative geroprotective interventions capable of suppressing the fundamental aging rate and redefining human longevity potential.

Physics in Ageing Biology: from applications to fundamental theories

Prof. Andrew E Teschendorff

I will describe quantitative methods to study aging cell biology, that are rooted in concepts from physics. First, I will present our approach to map transcription factor regulatory activities across tissues and cell-types, revealing age-associated patterns that are cell and tissue-independent. Second, I will describe our strategy to dissect and quantify the distinct components of epigenetic clocks, with a focus on stochasticity and cell-type heterogeneity. We find many instances of biological aging that are explained by non-stochastic processes, unlike precancerous conditions where increased risk is associated with an increased rate of stochastic DNA methylation change linked to an increased rate of cell-division. Third, I will describe our efforts in applying interpretable deep neural networks to build transcriptomic and epigenetic clocks at cell-type resolution, revealing a number of molecular aging hallmarks. I will end by presenting a brief outlook on how statistical mechanics could provide a theoretical and computational framework for understanding aging and age-related disease.

Stochasticity and memory in epigenetic ageing

Prof. Steffen Rulands

Statistical models termed ageing clocks can predict chronological and biological age from the longitudinal time evolution of DNA methylation. In this talk, I will discuss biophysical approaches to understanding the mechanistic basis of epigenetic ageing. Combining tools from theoretical physics and genomics I will show how ageing manifests in how epigenetic states of genome relate to each other and identify a phase separation mechanism driving changes in the epigenome during ageing. I will then discuss the different contributions of stochastic and deterministic components to ageing and explain a general principle underlying how stochastic and deterministic underlying the balance between both contributions in the genome-wide.

Dynamics of Ageing Biomarkers

Prof. Andrew Rutenberg

We would expect that biomarkers of aging will be approximately homeostatic, but would also exhibit complex interacting and stochastic dynamics during aging. We have analyzed a number of data sets using a highly-interpretable linear stochastic difference dynamics model. We find that the interactions between biomarkers are surprisingly simple (independent of age and health), which allows us to identify natural eigenmodes of aging – each of which is stable with a characteristic timescale. The stable points of these variables often drift with age, in agreement with allostasis. We find that both strongly drifting and slowly recovering variables are associated with adverse outcomes, such as mortality.

Can predictive models be developed for understanding complex ageing cellular dynamics?

Dr. Kumar Selvarajoo

Living organisms are highly complex dynamical systems that display unpredictable emergent behaviors, such as disease onset and progression. Over several decades, theoretical biologists have developed numerous predictive models for understanding diverse cellular processes based on sound mathematics, physics and computing techniques. Despite much is learned from the models, we are still very far from predicting our own aging process. In my talk, I will present some examples of predictive as well as statistical models based on omics datasets of diverse cellular processes, such as inflammation and cancer dynamics. Moving forward, can such models be expanded and integrated for the creation of holistic digital twin aging platform?

Beyond the Hallmarks: What are we missing in ageing research?

Prof. Matt Kaeberlein

Computational approaches to target ageing

Prof. Morten Scheibye-Knudsen

Insights into ageing, longevity and rejuvenation

Prof. Vadim Gladyshev

Statistical models termed ageing clocks can predict chronological and biological age from the longitudinal time evolution of DNA methylation. In this talk, I will discuss biophysical approaches to understanding the mechanistic basis of epigenetic ageing. Combining tools from theoretical physics and genomics I will show how ageing manifests in how epigenetic states of genome relate to each other and identify a phase separation mechanism driving changes in the epigenome during ageing. I will then discuss the different contributions of stochastic and deterministic components to ageing and explain a general principle underlying how stochastic and deterministic underlying the balance between both contributions in the genome-wide.

Dynamic ways of quantifying the human ageing methylome and exercise rejuvenation

Prof. Nir Eynon

During ageing, the human methylome exhibits both differential (i.e. change in mean) and variable (i.e. change in variance) shifts, along with a rise in entropy. We are building a multi-tissue (18 tissues) ageing methylome atlas (more than 90K samples), and assessing exercise responses in human muscle and blood. In the first part of the Atlas, using > 32,000 human blood methylomes from 56 datasets (age range = 6-101 years), we revealed an unprecedented proportion of the blood methylome that is differentially methylated with age (48% DMPs; FDR< 0.005) and variably methylated with age (37% VMPs; FDR< 0.005), with many sites overlapping between the two groups (59% of DMPs are VMPs). We observed that bivalent and Polycomb regions become increasingly methylated and divergent between individuals, while quiescent regions lose methylation in a more homogeneous manner between individuals (1). We also showed that individuals with higher exercise capacity have younger DNA methylation and transcriptome profiles in human muscle (2). We are currently in the process of analysing DNAm patterns from >14 different tissue to form the full picture of the ageing methylome, offering insights into primary molecular changes during ageing.

Rejuvenating aged oocytes for female reproductive longevity

Dr. Wang Haiyang

Aging-related decline in oocyte quality presents a significant challenge to female reproductive health and longevity, impairing the oocyte's ability to undergo meiotic maturation and successful fertilization. It is known that oocytes undergo two rounds of asymmetric cell divisions, resulting in a large fertilized egg and two small polar bodies. I will discuss how actin-based forces drive spindle migration during oocyte maturation and spindle rotation during fertilization, and how these processes are compromised with reproductive aging. To address age-related defects in oocytes, I have developed the Reconstituted Chimeric Follicle system, a 3D culture model that rejuvenates aged oocytes by replacing the aged follicular somatic microenvironment with a youthful one. My research demonstrates that young follicular somatic cells can restore the developmental competence of aged oocytes by reducing aneuploidy and enhancing mitochondrial function. These works not only advance our understanding of the fundamental mechanisms underlying oocyte aging but also hold promising potential for developing a safe, cell-based therapy to treat age-related infertility.

Understanding Evolution, Ageing, and Repair using Complex Network

Prof. Ee Hou Yong

To effectively intervene in aging, it is critical to understand the mechanisms driving mortality acceleration. Current biological models identify multiple hallmarks of aging, none of which can be considered fundamental. In longevity medicine, these hallmarks are often described as age-related diseases. Drugs targeting them are expected to produce modest lifespan extensions, with metabolic disorder treatments offering the largest benefit of approximately 10 years. This complexity necessitates borrowing approaches from physics, particularly statistical mechanics and complex systems theory, to uncover deeper, universal principles governing the aging process. We present a theoretical framework unifying aging mechanisms through thermodynamics, information theory, and stochastic processes. Analysis of DNA methylation data and longitudinal medical records reveals aging as a process driven by numerous independent, infrequent transitions between metastable states in a vast biological configuration space. These rare, high-energy transitions in a rugged energy landscape lead to effectively irreversible system failures across redundant biological structures. The cumulative impact of these transitions is captured by thermodynamic biological age (tBA), a stochastic variable reflecting entropy production and information loss during aging. This stochastic damage signature provides a theoretical basis for the absolute human lifespan limit, estimated at 120-150 years due to complete physiological resilience loss. Aging appears entropic and largely irreversible, with tBA correlating with physiological drift, reduced resilience, and exponential increases in chronic disease risk. However, we propose a fundamental thermodynamic control variable, the "effective biological temperature," regulating the magnitude of molecular fluctuations across biological pathways. Our analysis, supported by methylation data, identified this variable and demonstrated its influence on initial mortality rates and mortality rate doubling time while remaining independent of maximum lifespan. Notably, reducing the effective temperature could extend the average lifespan from approximately 80 years to 120-150 years, far exceeding the impact of drugs targeting age-related diseases. This work offers the first comprehensive theoretical model of aging, integrating entropic biological aging processes with dynamic resilience loss. It defines a clear path for substantial lifespan extension by targeting thermodynamic and informational drivers of aging, shifting focus from symptom management to the root causes of age-related decline. These insights will be presented at the Eureka Conference on Aging, emphasizing how a deeper understanding of these principles could drive transformative geroprotective interventions capable of suppressing the fundamental aging rate and redefining human longevity potential.

Percolation theory in complex networks

Dr. Feng Ling

Abstract: Percolation theory is a theory that describes critical phase transitions. It has been widely studied in complex networks both in theory and its associated applications. It studies the transitions of a network's overall connectivity, as well as it's resilience against different types of attacks - two facets of the same phenomenon. In this talk, I will cover some basic concepts in percolation theory, the factors that affects the critical threshold and nature of phase transitions, as well as its associated applications ranging from epidemics spreading, social network analysis, and some related aspects on biological networks.

SHORT TALKS

Heritability of human lifespan in the light of the saturated removal model of ageing

Mr. Ben Shenhar

Investigating the interplay between genetics and environment in human lifespan is crucial for understanding aging and for developing potential treatments based on protective genetic functions. While research on twins and family registries has found limited heritability of longevity (10-30%), the underlying genetic components remain poorly understood. Here we present two findings. First, we show that extrinsic mortality masks the heritability of lifespan, in a model-independent way. Twin studies, based on cohorts from roughly 100 years ago with high extrinsic mortality, underestimated the heritability of lifespan. When adjusted for extrinsic mortality, heritability of intrinsic longevity increases to approximately 50% - roughly twice the previous estimates. Second, to explore the biological processes underlying this genetic variation, we employed the saturated removal model - a calibrated mathematical model of aging that describes damage production, removal, and noise, with mortality when damage crosses a threshold ("Xc"). To explain the twin heritability of lifespan we explored which parameters in the model can vary in the population. We find that most parameter variations can be excluded since, at the variation needed to explain twin heritability, they give rise to unrealistic life spans beyond 140. The main parameter that can explain twin variability and not exceed observed lifespans is the threshold parameter Xc, which we estimate varies by about 30% between individuals. This parameter describes biological resilience - suggesting that the most widely varying genetic component of longevity in humans stems from damage tolerance mechanisms. We use this understanding to provide quantitative survival advantages for siblings of long-lived individuals. These findings suggest model-informed approaches for genetic analyses to identify the specific genetic variations influencing human lifespan.

Estimating Uncertainty in Biological Age Prediction: A Fundamental Challenge

Dr. Dmitrii Kriukov

Biological age is an unobservable quantity in nature; thus, its estimation fundamentally depends on its definition and cannot be explicitly validated. If we aim to use biological age as a decision-making tool or a clinical trial endpoint, its reliability must be rigorously assessed. The quantitative reliability of an individual's biological age estimate is best characterized through the concept of uncertainty. However, uncertainty is multifaceted and extends beyond the "average model error on a validation subset," being influenced by multiple other factors. In this talk, I will deconstruct the sources of uncertainty in biological age prediction and discuss key concepts such as aleatoric (arising from internal data randomness) and epistemic (arising from lack of knowledge) uncertainty—both crucial in modern machine learning. I will also demonstrate how uncertainty can be quantified using contemporary machine learning techniques and how these methods apply to common scenarios in geroscience. Furthermore, we will explore extreme cases of biological age prediction, particularly with epigenetic aging clocks. These include multiple efforts to exemplify or validate putative epigenetic age reversal in cellular reprogramming, as well as the so-called "ground zero" event in embryogenesis. While not disputing the biological plausibility of age reversal, we will show that the high uncertainty of aging clock predictions challenges the reliability of reported rejuvenation effects during in vitro reprogramming and throughout embryogenesis. Finally, I will emphasize the broader significance of uncertainty estimation—not only for constructing robust biological age predictors but also for developing any surrogate biomarkers of aging, whether grounded in physical theories or derived from artificial intelligence approaches.

Use of potential gerotherapeutic drugs and mortality of geriatric rehabilitation inpatients

Dr. Weilan Wang

Repurposing drugs for longevity, or gerotherapeutics, may enhance healthspan and lifespan. This retrospective analysis of the RESORT cohort, a longitudinal study of geriatric rehabilitation patients (n=1890, mean age 82.6 years, 56% female), investigated the association of gerotherapeutic drugs (ACEi/ARBs, aspirin, metformin, beta-blockers, bisphosphonates) with physical function, readmission, and mortality. No functional improvement was observed, but the use of metformin, aspirin, and ACEi/ARBs was associated with lower post-discharge mortality risk. Using three or more drugs further reduced mortality. These findings indicate potential benefits of geroprotective drugs, with drug combinations possibly offering greater protective effects.

Dynamical modelling of the frailty index indicates that health reaches a tipping point near age 75

Dr. Glen Pridham

The frailty index (FI) is a health state variable that summarizes an individual's overall health. The FI is an important risk factor for adverse outcomes, such as hospitalization and death. Fluctuations in the FI are also predictive of mortality, and are known to occur due to transient stressor events like acute illness. Yet little is known about the dynamics of the FI. The de facto, damage-promotes-damage paradigm posits that the FI obeys a simple positive feedback (exponential) model. We explored alternative models of the FI and their implications for the overall age-related decline in health, together with possible biological connections. In contrast to the damage-promotes-damage paradigm, we find that the FI has two distinct dynamical phases. This includes a young, healthy phase with low FI and an older, unhealthy phase with high FI. These phases are separated by a tipping point in health at age 75 caused by a critical loss of ability to resist damage (robustness) and repair damage (resilience) with increasing age and FI. A biological mechanism for this behaviour remains elusive, but preliminary results indicate a potential connection to the senescence-associated secretory phenotype. As this phenotype is circulated through the blood, it can account for the observed coherent decline across multiple systems. We discuss the effects of FI dynamics on population-level health, and the potential to identify key aging mechanisms at the individual-level using dynamical models of the FI.

Analysis of individual mouse survival data from large databases – insights and findings

Mr. Kamil Pabis

Although murine lifespan extension remains the gold standard for assessing interventions proposed to impact the biology of aging, there are important limitations to this approach ranging from small sample sizes, over limited genetic diversity of employed strains to unhealthy controls. We aim to address the latter issue to help researchers improve robustness of their studies and analyses. Unhealthy, short-lived animals in the control group may exaggerate the relative efficacy of putative longevity interventions. Moreover, due to the high cost and long timeframes of mouse studies, it is rare that a particular longevity intervention will be independently replicated by multiple groups.
To facilitate identification of successful interventions, we developed an approach particularly suitable for well-characterized inbred strains and HET3 mice. We used the methods of systematic review and meta-analysis, recovery of unpublished raw data and statistical analysis to develop what we term the 900-day rule.
We showed that studies with shorter-lived control animals exaggerate lifespan results for statistical and biological reasons, that commonly used mouse strains live close to 900 days, that lifespans reported in published studies are improving, and slowly converging towards 900 days, over the course of the 20th century. Based on these results we suggest two approaches. First, to identify compounds that extend lifespan compared to historical controls, which are used to define the strain- and species-specific upper limit of lifespan. Second, identify compounds that extend the lifespan compared to long-lived within study controls. Finally, using this 900-day rule we identified several candidate interventions from the literature that merit follow-up studies e.g. spermidine or L-deprenyl. To summarize, in the absence of independent replication, a putative mouse longevity intervention should only be considered with high confidence when control median lifespans are close to 900 days or if the final lifespan of the treated group is considerably above 900 days.

No easy answers - AI-assisted protein design for lifespan extension

Dr. Andrei Tarkhov

Aging is a multifactorial process affecting all levels of organismal organization. The consensus is that human anti-aging therapies would have to target multiple pathways, however, the practical tools for doing so remain limited. Epigenetic reprogramming with the Yamanaka factors (OSKM) has shown some promise in reversing aging phenotypes but leads to loss of cellular identity. These four transcription factors acting together perturb numerous pathways, dramatically reorganizing the cellular state. We attempted to disentangle the rejuvenating and reprogramming effects of these factors by directly modifying their amino acid sequences via AI-assisted protein reengineering. Traditional protein engineering methods like directed evolution and chimerization remain largely constrained due to combinatorial complexity. To make larger edits feasible, we constrained the search space to evolutionarily-plausible sequences by training a protein language model, GPT-4b micro, which can be steered by including co-evolving and interacting sequences in its prompt. We guided the model to generate SOX2 and KLF4 variants, modifying up to ⅔ of the wildtype sequences. We first screened our variants for improved reprogramming efficiency and speed in human dermal fibroblasts. Our re-engineered SOX and KLF4 variants improved over wild type factors by at least two orders of magnitude on day 10 of reprogramming. To evaluate the same variants for their rejuvenation potential and thereby disentangle cell state changes from cell fate changes, we are developing a platform to screen for resilience. Increased stress resilience is used in several model organisms as a proxy for rejuvenation, and loss of resilience is evident across multiple aging phenotypes in humans, thus allowing for a fast predictor for healthy life extension. Initial results suggest that variant performance on reprogramming vs resilience assays is decoupled. Our results highlight the promise of AI-assisted protein design for overcoming critical bottlenecks in cellular reprogramming, and in rational engineering of human healthy life extension.

Critical phenomenon associated with luminogenesis during ovarian follicle development

Dr. Leong Kim Whye

The development of a fluid-filled lumen within ovarian follicles is a critical step in early female reproduction and ovulation. While there exists evidence linking lumen formation to oocyte maturation, the underlying mechanism of luminogenesis and the mechano-chemical functions of fluids in antral follicles remain elusive. In this study, we investigated the spatiotemporal dynamics of lumen growth in mouse follicles, using advanced microscopy, ex vivo platform and biophysical model. In the early phase of lumen formation, we observed temporal changes in cell proliferation and death, accompanied by the emergence of basally polarized rosettes and spatial patterning of cell-cell junctional proteins within the follicles. 3D quantification of lumen size and geometry revealed that the lumen in the pre-antral follicles behave as percolating fluid pockets exhibiting critical behavior. As the follicles grow above a critical size, the fluid network transitions to a phase-separated state characterised by the appearance of a dominant growing lumen. By modelling the granulosa cells and follicular fluids as a binary fluid mixture based on an Kawasaki-type Ising model, we successfully recapitulated the lumen dynamics in various follicle stages. The model further revealed the cell-fluid surface tension as a key parameter regulating the fluid phase transition, which we experimentally validated through pharmacological inhibitions of cell-cell junctions. We further discovered that the self-organized criticality in pre-antral follicle is robustly maintained through the surface tension gradient (as observed experimentally), while a transition to the phase-separated state in antral follicles is triggered by changes in the overall lumen fraction or surface tension gradient during follicle maturation. In the future, we aim to elucidate the functional benefits for mammalian follicles to operate at criticality, and explore its implications in misregulated luminogenesis and ovulation during ovarian aging.

Identifying Novel Geroprotectors through Ageing Network Mapping

Mr. Weihan Huai

In the current aging and longevity field, small molecules, often called geroprotectors, is considered as one of the most clinically applicable approach to combat aging.4 While many methods has been applied in discovering novel geroprotectors, genetic-based drug prediction is gaining attention for its low cost and potential to discover novel class of geroprotectors. However, most of the current genetic-based approaches involves using data of known interventions, which lead to prediction of a conserved list of compounds. Also, these methods assumes that transcriptomic changes in Thus, there is an urgent need to find tools to expand the number of potential geroprotector candidates and to discover novel classes of geroprotectors. We proposed a novel method to identify longevity-associated gene signatures for genetic-based drug prediction. We firstly modelled aging transcriptome of C.elegans as a network to distil complex aging transcriptome into modules with genes co-expressed during aging. We next used meta-analysis on public datasets with various interventions to associate module expression with longevity to get longevity-associated modules. We further used hub genes within longevity-associated modules to query the Connectivity Map (CMap) to identify potential geroprotector candidates and selected 15 of them to test their ability to extend lifespan in C.elegans. Among the 15 drugs we tested, 10 of them significantly extended lifespan of C.elegans, with 6 of them being completely novel. Among these compounds, compound B (masked for IP application), a mucolytic drug, and compound G, a natural product from traditional Chinese medicine, appeared to have strongest lifespan extension. This study provides a new method to deconvolute the aging transcriptomics and identified critical longevity-associated processes that can be altered upon interventions. We successfully identified novel potential geroprotectors with low toxicity, which have significant clinical applicability and could produce significant impact in wide populations.

Methylation and network entropy measurements during ageing

Dr. Csaba Kerepesi

What is aging? How do aging and entropy relate to each other? Can different entropy measurements be suitable to evaluate aging and rejuvenation? Entropy is often linked to the aging process as it can be defined as a measure of disorder in an aging system, in which death is the maximum disorder. An approach for measuring entropy during aging is methylation entropy which is the average Shannon entropy of all individual CpG sites of the sample. Previously we observed a steady increase in methylation entropy during the adult life of naked mole-rats pointing to the possibility of naked mole-rat aging (Kerepesi et al. 2022, NatComm). Later we observed organ-specific differences in mean methylation entropy between low- and high-capacity running (LCR and HCR) rats, and the direction of these differences was the opposite compared to the age-related changes in the rat blood (Kawamura, Kerepesi et al. 2024, bioRxiv). Another entropy approach can be the measurement of entropy (i.e. diversity) of the microbiome. Calculating the Shannon entropy of the relative abundances of taxa at seven taxonomic levels, we revealed the link between accelerated epigenetic aging an increased entropy in the gut microbiome of physically active middle-aged/old individuals (Torma, Kerepesi et al. 2024, Aging Cell). Most recently, we investigated the aging of biological networks (such as protein-protein interaction networks, and brain graphs) focusing on the changes of network entropy. I will present interesting unpublished results.

Metabolic drivers of species ageing

Dr. Peter J Mullen

Humans are living longer, and new strategies are required to extend healthy aging. Many current strategies involved manipulating metabolism, but it is not known how metabolism changes across and within different species. Answering this could reveal new mechanisms of aging and age-targeted therapeutics. We approach this by using a systems biology approach to identify age-associated metabolic changes in multiple species, organs and both sexes. We described distinct patterns of metabolic aging that are differentially affected by sex, and identify common metabolic changes, such as decreased hydroxyproline levels. We also developed a metabolic aging clock that predicts biological age and identified alpha-ketoglutarate, previously shown to extend lifespan in mice, as a key predictor of age. Our results reveal fundamental insights into the aging process and identify new therapeutic targets to maintain organ health.

A Network Perspective on Comparative Lipidomics and Lifespan Regulation

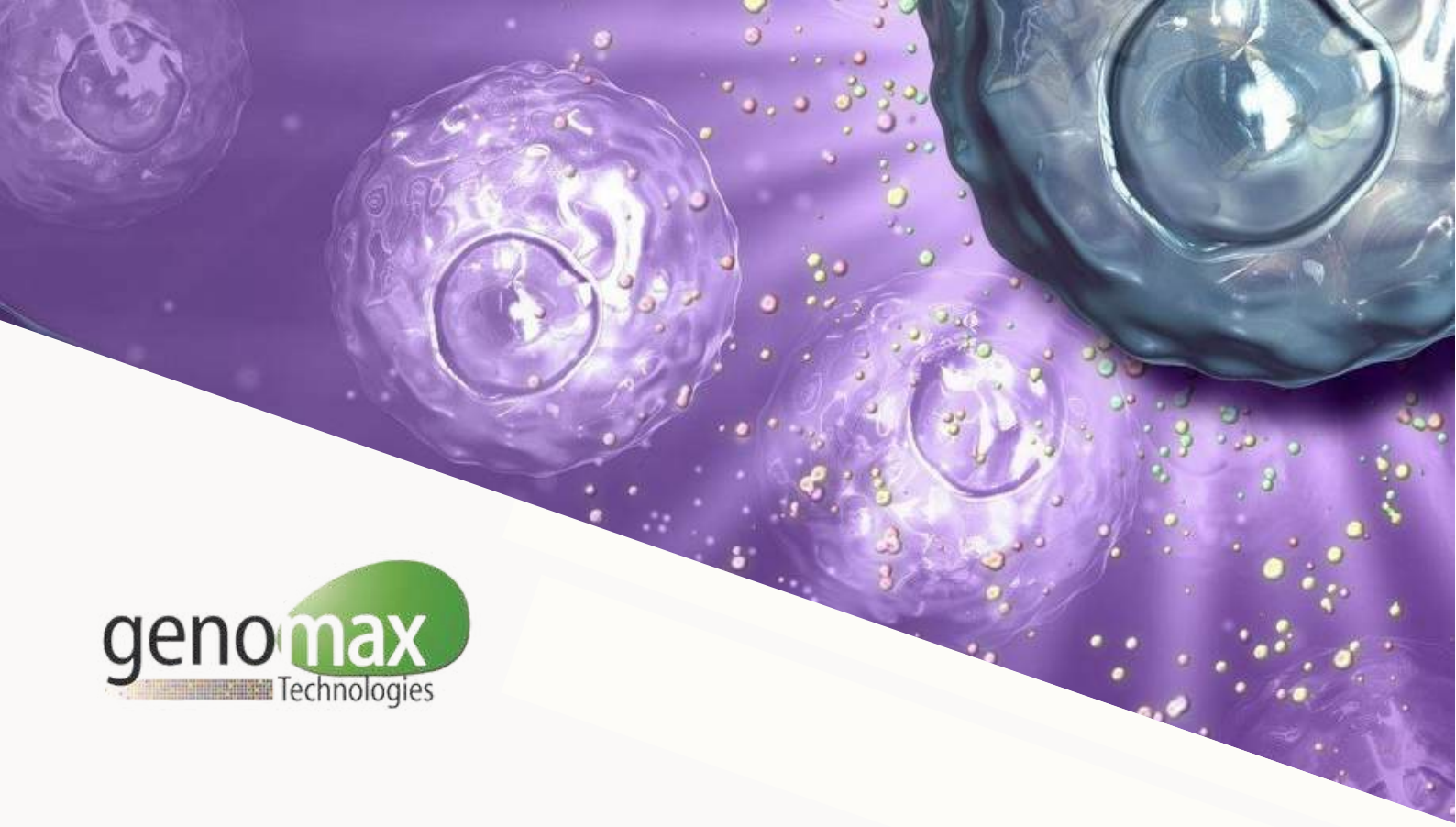
Dr. Max Unfried

Lipids are key regulators of cellular aging, impacting membrane integrity, energy metabolism, and inflammation. While lipid composition is known to shift with age, the relationship between species-specific lipidomes and lifespan remains unclear. Here, we use a comparative lipidomics approach to investigate evolutionary lipid signatures of longevity. By analyzing lipidomic profiles from short-lived and long-lived mammals, we identify lipid pathways enriched in species with exceptional lifespans. Our results suggest that longevity is encoded in lipidome architecture, providing new targets for interventions aimed at extending healthy lifespan.

Age-dependent remodeling of ribosomes in skeletal muscle: Structural and functional changes

Dr. Yumi Kim

Ribosome heterogeneity is believed to reflect their specialized regulatory roles. Using the turquoise killifish, the shortest-living vertebrate model, we investigated age-dependent differences in ribosomal composition and function. We observed significant changes in the mRNA and protein expression levels of ribosomal proteins (RPs) and found that two distinct regions in 18S and 28S rRNAs were uniquely protected by RPs in young versus aged skeletal muscle. Structural and dynamic analyses of ribosomes further confirmed notable differences between young and aged muscle ribosomes. Interestingly, these age-related changes had minimal impact on the translation of foreign mRNA. However, the interaction between Ltn1 and uL24 in the large subunit was markedly increased in aged ribosomes, while RACK1 in the small subunit exhibited an extended conformation. Collectively, our findings suggest that the structural and functional remodeling of ribosomes in aged muscle enhances their role in ribosome-associated quality control of newly synthesized proteins.



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P1 Ms Sharah Mae Capinpin Elucidating the role of circadian rhythm in neurogenesis during ageing using cellular models of ageing
P2 Mr Evgeniy Efimov Benchmarking epigenetic aging clocks: towards a standardized and clinically relevant methodology
P3 Ms Ekaterina Kuzmina Modeling Age-Associated Neural Oscillations to Advance Adaptive Therapies in Parkinson's Disease
P4 Dr Shi Yan Ng Telomere shortening induces aging-associated phenotypes in hiPSC-derived neurons and astrocytes
P5 Dr Zi Xiang Lim The impact of aerobic exercise intensity and age on muscle oxygenation recovery kinetics in exercising and non-exercising skeletal muscle
P6 Asst Prof Chii Chan Investigating the mechanobiological principles underlying mammalian egg development during ovarian aging using advanced biophotonics and biophysical approaches
P7 Dr Marie Cutiongco Revealing nanoscale nuclear envelope deterioration in cellular aging via nanopillar-guided nuclear deformation
P8 Ms Ankita Nitin Nayak Investigating the impact of topographical cues on mechanical cell competition
P9 Ms Sandra Jose Development of Targeted Proteolysis Approaches to Promote Healthy Longevity
P10 Assoc Prof Oliver Dreesen Molecular functions of the nuclear lamina in premature cell aging & senescence
P11 Dr Zongmin Liu Evaluating FDA-Approved Drugs for Longevity Potential Using Large Language Models: A Rigorous, Multi-Platform Analysis
P12 Ms SiYi Liu Mfsd7c (FLVCR2) as a promising drug target to enhance memory via the choline transport pathway in Alzheimer's and aging subjects
P13 Ms Viktoriia Palagina Optimizing Methylation Signatures for Age-Accelerated Diseases Determination
P14 Dr Ravi Kumar Chaudhary Regenerative Strategies for Age-Related Diabetes and Cardiovascular Diseases: A Transdifferentiation Approach
P15 Mr Vlad Fedotov Evaluating the Impact of Somatic Mutations on Aging: A Dynamical Systems Approach
P16 Ms Trina Tan A novel molecule to boost NAD+
P17 Dr Han Dong Limiting cap-dependent translation increases 20S proteasomal degradation and protects the proteomic integrity in autophagy-deficient skeletal muscle
P18 Dr EngHock Chia Running and Sprinting as Geroprotectors: Exploring the New Frontier in Biophysics of the Human Body

P19 Ms Mingtong Gao Targeting AMPK Deficiency: Geroprotective Interventions to Reverse Cellular Aging

P20 Prof Jean-Jacques TEMPRADO Age-related changes in coordination dynamics within and between brain, muscular and behavioral levels: a dynamical systems and complexity theories approach

P21 Ms Jessica Lu Digital biomarkers of ageing for monitoring physiological systems in community-dwelling adults

P22 Ms Liyan Huang Intake of dietary carotenoids and flavonoid at midlife and risk of physical frailty at late life: the Singapore Chinese Health Study

P23 Mr Boon Heng Ng Investigating the role of theca cell mechanosensing during mammalian ovarian follicle development

P24 Mr Naveh Raz Comparative biology using the saturating removal model

P25 Mr Djakim Djael Latumalea DoliClock: A Lipid-Based Aging Clock Reveals Accelerated Aging in Neurological Disorders

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P34 Ms Grace Keh TFEB Activator Screen Identified Sertindole, an Anti-psychotic Drug, as a Novel TFEB Activator for Geroprotection

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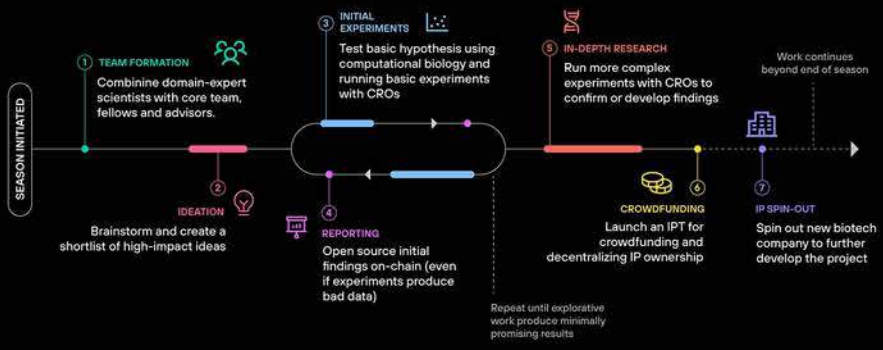

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