Memoir Of My Career

Mentors and distinguished medical scientists

Choi Young-kil sits with his former professor Edmund B. Flink at a restaurant in Seoul during the American doctor's visit to Korea in 1971. Courtesy of Choi Young-kil

Roger Guillemin, a Nobel Prize laureate in physiology and medicine in 1977, visits Kyung Hee Medical Center as a special lecturer in 1982. Courtesy of Choi Young-kil

Prominent scientist and professor of medicine in cortisol metabolism and kinetics

Emile Eugene Werk Jr., 93, end...
portcitydaily.com

Publications on cortisol metabolism and kinetics

Personal Publications
(Related to cortisol metabolism and kinetics)

1) Ubiquitin specific peptidase 19 involved in transcriptional repression of retinoic acid receptor by stabilizing coro2A. Oncotarget April 25, 2016

2) Cushing's Syndrome With Dual Pituitary Adrenal Control. Arch Intern Med..1970;125(6)1045-1049

3) Interference in the effect of Dexamethasone by Diphenylhydantoin. N Engl J Med 1969;281;32-34

4) The measurement, excretion, and source of urinary 6-hydroxycortisol in humans. Steroids, Volume 14, Issue 5, November 1969, Pages 455-468

5) Interference of heparin containing benzyl alcohol in the fluorometric determination of plasma corticosteroids. J Clin Endocrinol Metab. 1967 Sep;27(9);1350-2

6) Failure of Metyrapone To Inhibit 11-Hydroxylation of 11-Deoxycortisol During Drug Therapy. J Clin Endocrinol Metab. Volume 27, Issue 9, 1 September 1967, Pages 1358-1360

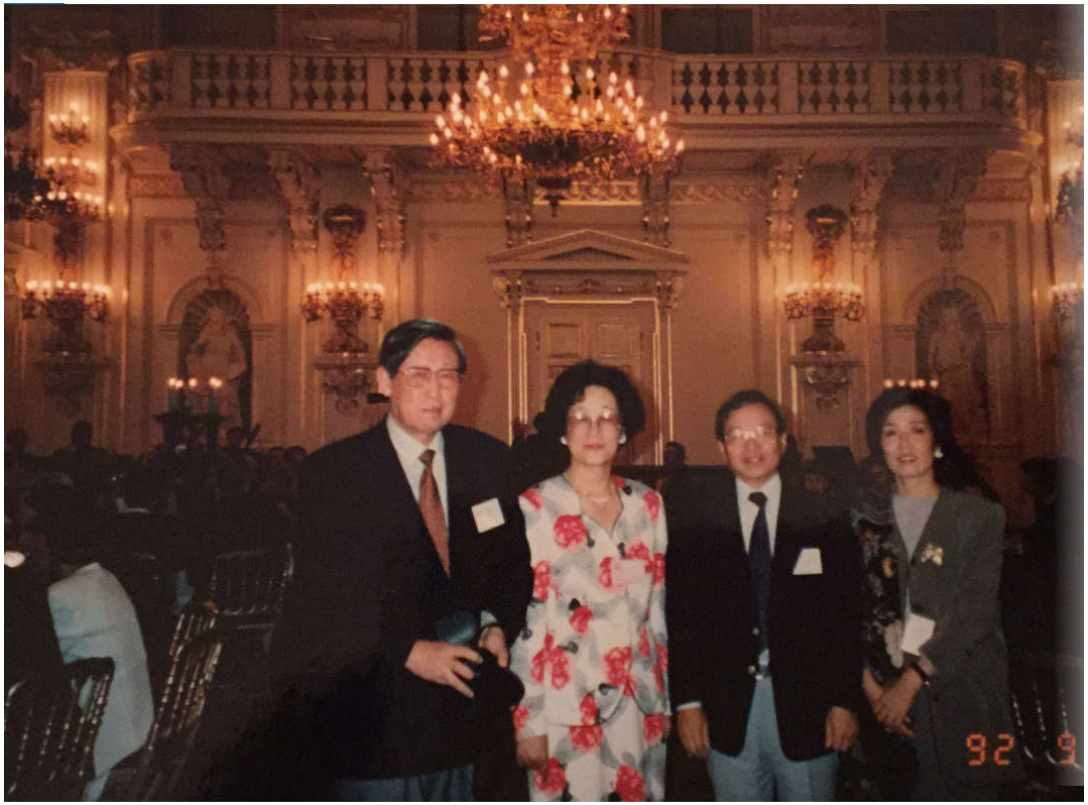
7) Effect of diphenylhydantoin on cortisol kinetics in humans. Journal of pharmacology and experimental therapeutics.Vol.176,no1, sep20,1970 page27-34

第一次 韓日糖尿病SYMPOSIUM記念

1981. 10. 31

Korea Japan symposium on diabetes mellitus


THE 3 RD KOREA-JAPAN SYMPOSIUM ON DIABETES MELLITUS
SEPTEMBER 6-7, 1985 SEOUL KOREA
KOREAN DIABETES ASSOCIATION


92 9

1976년 제9차 IDF 대회에 참석한 최영길, 민병석, 김응진, 이태희, 이상용 교수가 비엔나의 베토벤 동상 앞에서 기념촬영을 하였다.

1979년 오스트리아 비엔나에서 열린 제10차 IDF 대회에 참석한 민병석, 이상용, 김응진, 이태희, 최영길, 윤영길 교수의 기념촬영 사진이다.

Perspective on the progress of diabetes research

- Precision Medicine

- Sasp(senility associate secretory pattern)

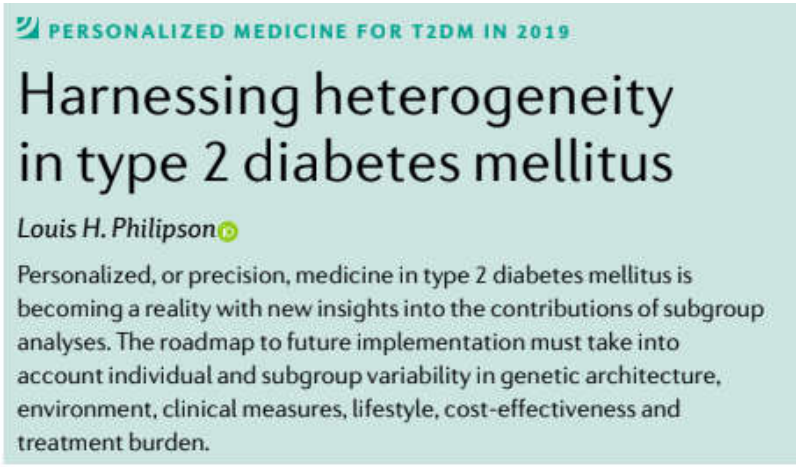
Harnessing heterogeneity in type 2 diabetes mellitus

Louis H. Philipson

Nature Reviews Endocrinology **16**, 79–80 (2020) | Cite this article

Personalized, or precision, medicine in type 2 diabetes mellitus is becoming a reality with new insights into the contributions of subgroup analyses. The roadmap to future implementations must take into account individual and subgroup variability in genetic architecture, environment, clinical measures, lifestyle, cost-effectiveness and treatment burden.

Precision medicine Vs idiopathic hyperglycemia



PERSONALIZED MEDICINE FOR T2DM IN 2019

Harnessing heterogeneity in type 2 diabetes mellitus

Louis H. Philipson

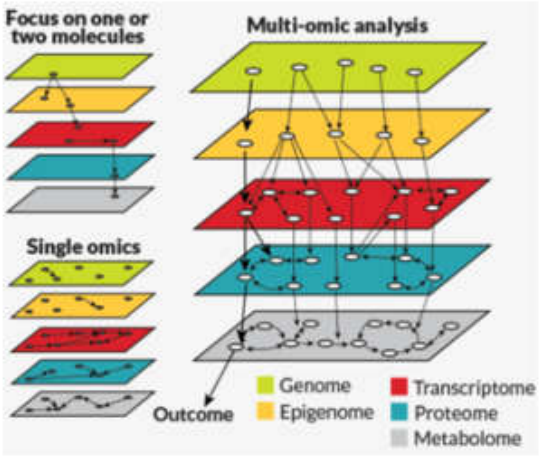
Personalized, or precision, medicine in type 2 diabetes mellitus is becoming a reality with new insights into the contributions of subgroup analyses. The roadmap to future implementation must take into account individual and subgroup variability in genetic architecture, environment, clinical measures, lifestyle, cost-effectiveness and treatment burden.

Cluster formation and Precision medicine

Key advances

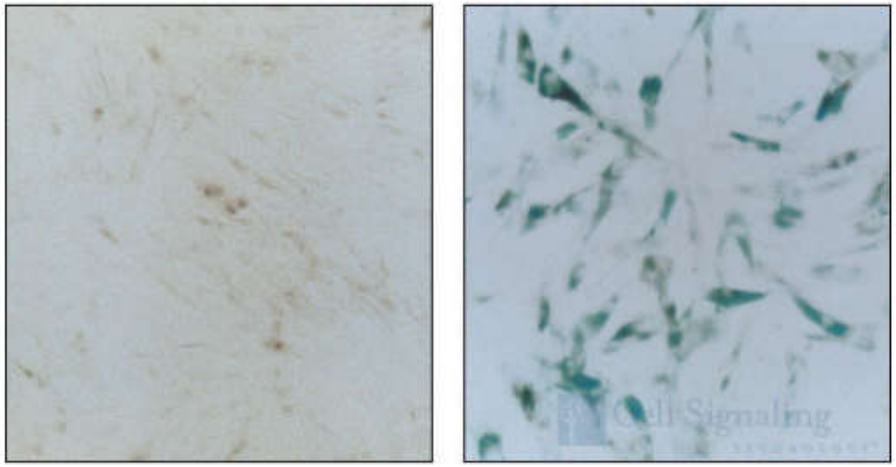
- Subgroup analyses have divided patients with type 2 diabetes mellitus (T2DM) into at least five clusters that differ with regards to genetics, insulin secretion, disease progression and disease complications
- Specific phenotypic measures of readily measured continuous clinical features can help predict specific outcomes such as fatty liver disease or neuropathy.
- Autoimmunity screening might be beneficial in all patients with T2DM[7].
- Deep longitudinal omics profiling can lead to prediction models of insulin resistance with increased acceptance of diet and exercise changes in research participants[8].
- Individuals with biomarkers of insulin resistance can benefit from targeted treatments with PPAR γ agonists as opposed to sulfonylureas to address cardiovascular protection[9].
- Models based on multi-criteria decision analyses that include disease outcomes, patient preferences and medication characteristics can improve personalized treatments.

Seeing a Forest and Not a Tree

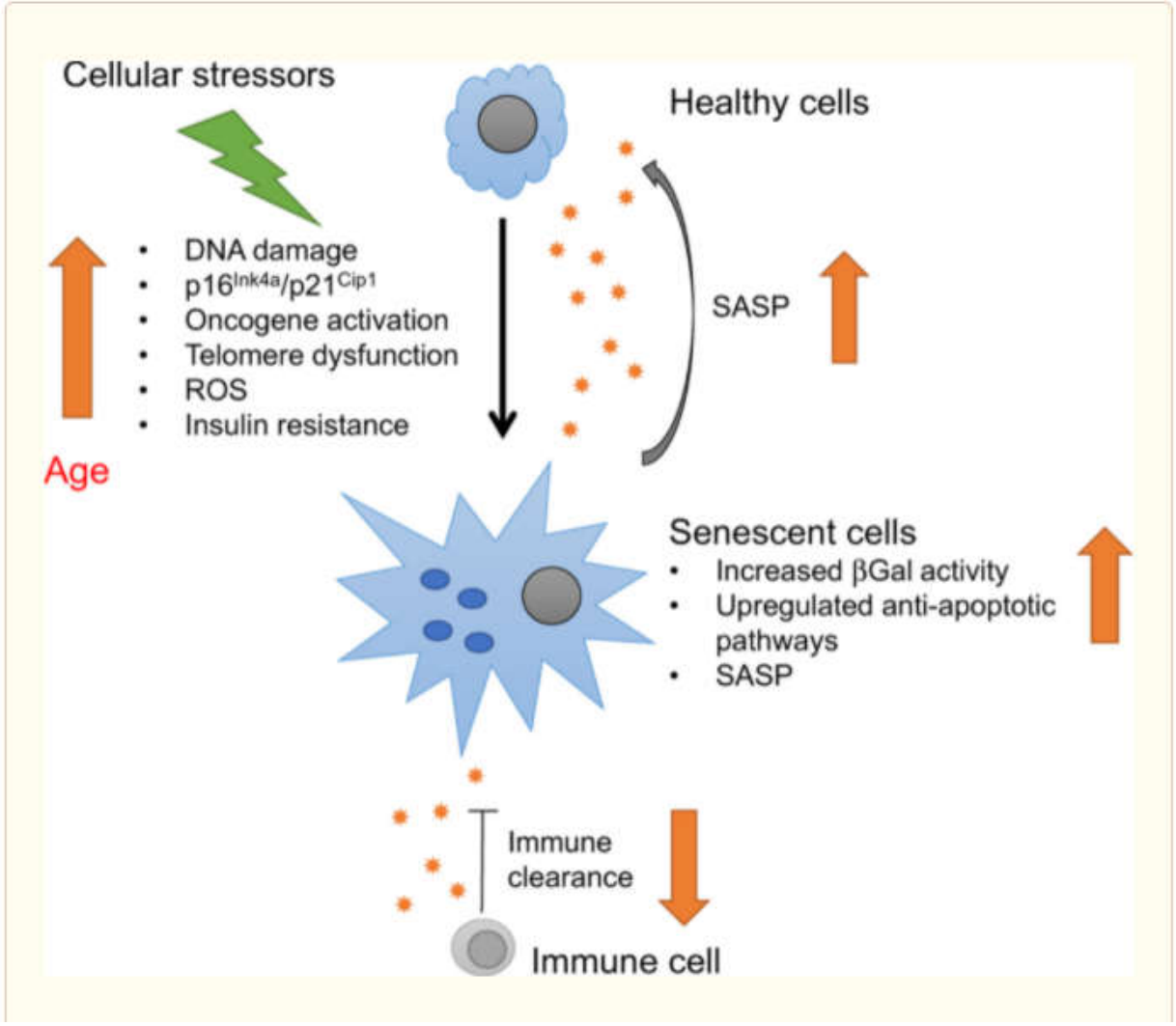


Source: Katsuyuki Yugi et al / Trends in Biotech. 2016

Beta galactosidase staining Sasp cell

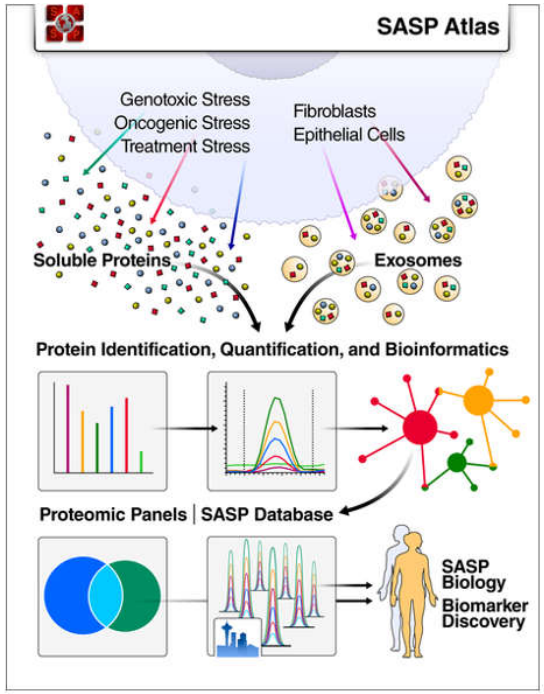


β-Galactosidase staining at pH 6 on normal WI38 cells at population doubling 29 (left) and senescent WI38 cells at population doubling 36 (right).



β Cell Aging Markers Have Heterogeneous Distribution and Are Induced by Insulin Resistance. [Cell Metab. 2017]

CCL2,1L1A,TNF ALFA,IL6 ,EXOSOME



Basisty N, Kale A, Jeon OH, Kuehnemann C, Payne T, et al. (2020) A proteomic atlas of senescence-associated secretomes for aging biomarker development. PLOS Biology 18(1): e3000599. https://doi.org/10.1371/journal.pbio.3000599
https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.3000599

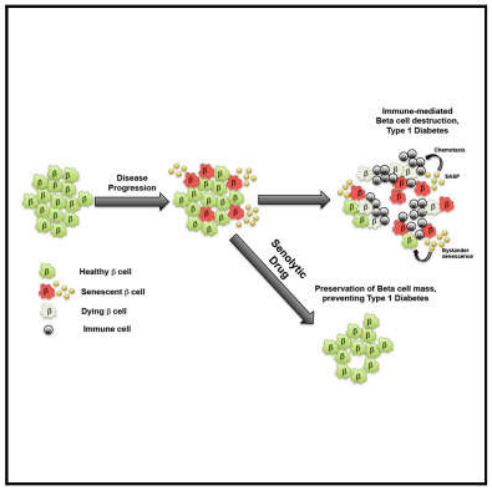
Senolytics and Type 1 diabetes

Article

Cell Metabolism

Targeted Elimination of Senescent Beta Cells Prevents Type 1 Diabetes

Graphical Abstract



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In Brief

Pancreatic beta cells are usually thought as passive victims of immune cell attack in type 1 diabetes (T1D). Thompson et al. now demonstrate that a subpopulation of beta cells becomes senescent and actively promotes the immune-mediated destruction process. Clearance of senescent beta cells with small-molecule inhibitors prevents T1D.

Highlights

- Beta cells in NOD mice and human T1D become senescent
- Senescent beta cells acquire a senescence-associated secretory phenotype
- Senescent beta cells upregulate Bcl-2 and senolytic drugs induce their apoptosis
- Clearance of senescent beta cells preserves beta cell mass and prevents diabetes

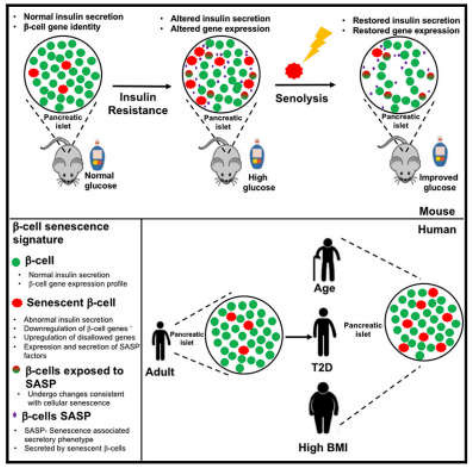
Senolytic drugs and insulin resistance

Article

Cell Metabolism

Acceleration of β Cell Aging Determines Diabetes and Senolysis Improves Disease Outcomes

Graphical Abstract



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In Brief

Aguayo-Mazzucato et al. identify the signature of senescent pancreatic β cells and show that the population of senescent β cells is increased by insulin resistance but is partially reversible. Removing senescent cells improves insulin secretion, genetic identity, and glucose homeostasis. These findings provide insight into how β cell senescence contributes to type 2 diabetes, opening new therapeutic targets.

Highlights

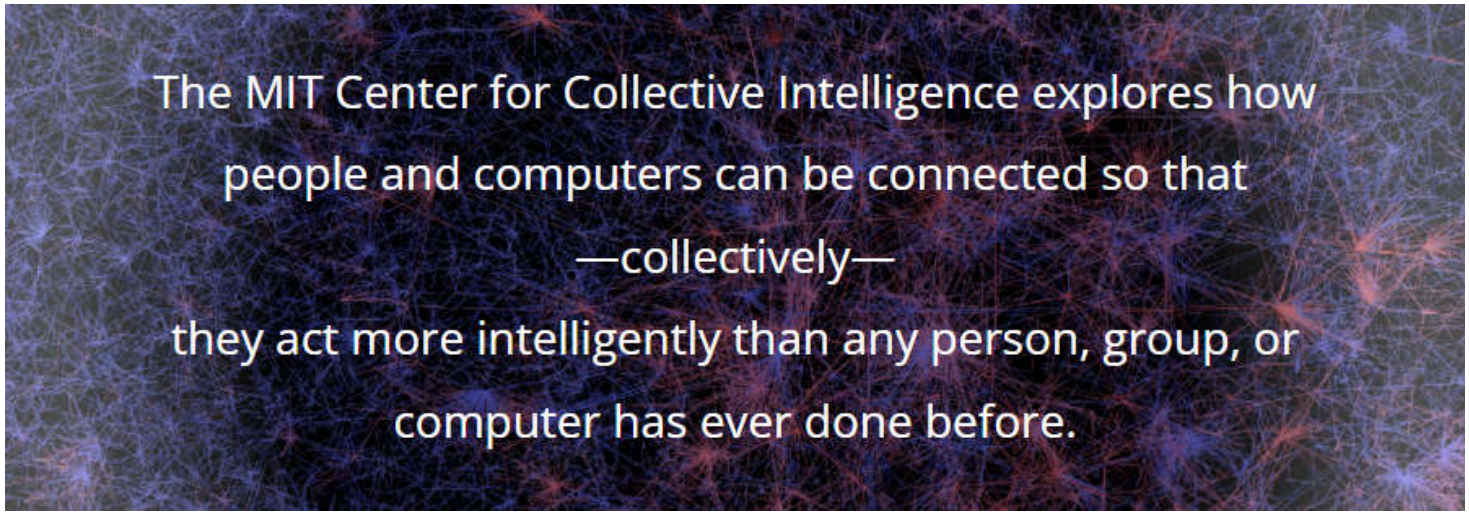
- β cell senescence signature reveals loss of identity and upregulation of SASP factors
- Insulin resistance accelerates the appearance of senescent β cells
- Clearance of senescent cells improves glucose levels, β cell function, and identity
- In humans, β cell senescence increases with type 2 diabetes, age, and BMI

Vision and New Leadership

Spread of Current Breakthrough

- Has no Historical Precedent
- Is Evolving at an Exponential rather than at a Linear Pace
- Penetrating Vision
- Vigilant Readiness

Collective intelligence, Wall breaking and Artificial information



Law of Diminishing Returns

- Winner Takes All (Priority of Occupancy)
- Basic Science Advancement
- Information Technology (Big Data and AI)
- Increasing Returns Principle VS Law of Diminishing Returns

Tiger or cat

Charles Darwin

- 새로운 환경에 적응하고 선택된 Species만 생존한다.
  Leadership with Solid Vision and Integrity (Actionable)
- Negative Regulation
- Homo Sapiens VS Homo Deus(superhuman)

노인과 어른의 차이, 의학계의 큰 스승

전종휘(全鍾暉)

국내 최초 내분비 연구소 설립
임상에서 방사면역 측정 표준화
및 유전체 분석 등 활용
임상 의학에서 분자생물학의
개념 도입 및 확산
선진의학의 기초 확립