1B – Implications to Retirement Planning

SOA Antitrust Disclaimer
SOA Presentation Disclaimer

LINKING RETIREMENT AGES TO LIFE EXPECTANCY DOES NOT LESSEN THE CONSEQUENCES OF UNEQUAL LIFESPANS

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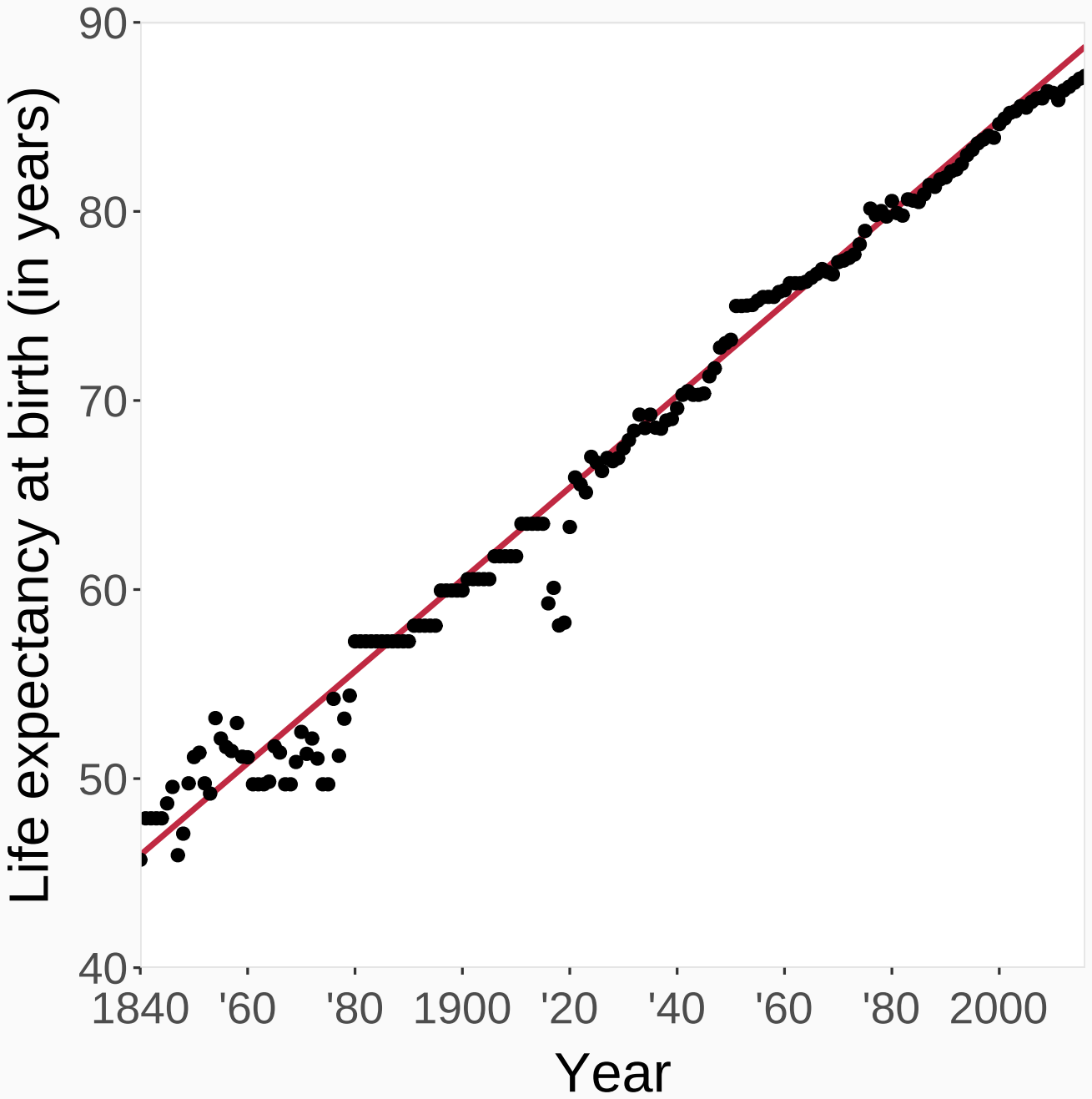
BEST PRACTICE LIFE EXPECTANCY AT BIRTH



so what?

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- Put preassure on **national finances**.

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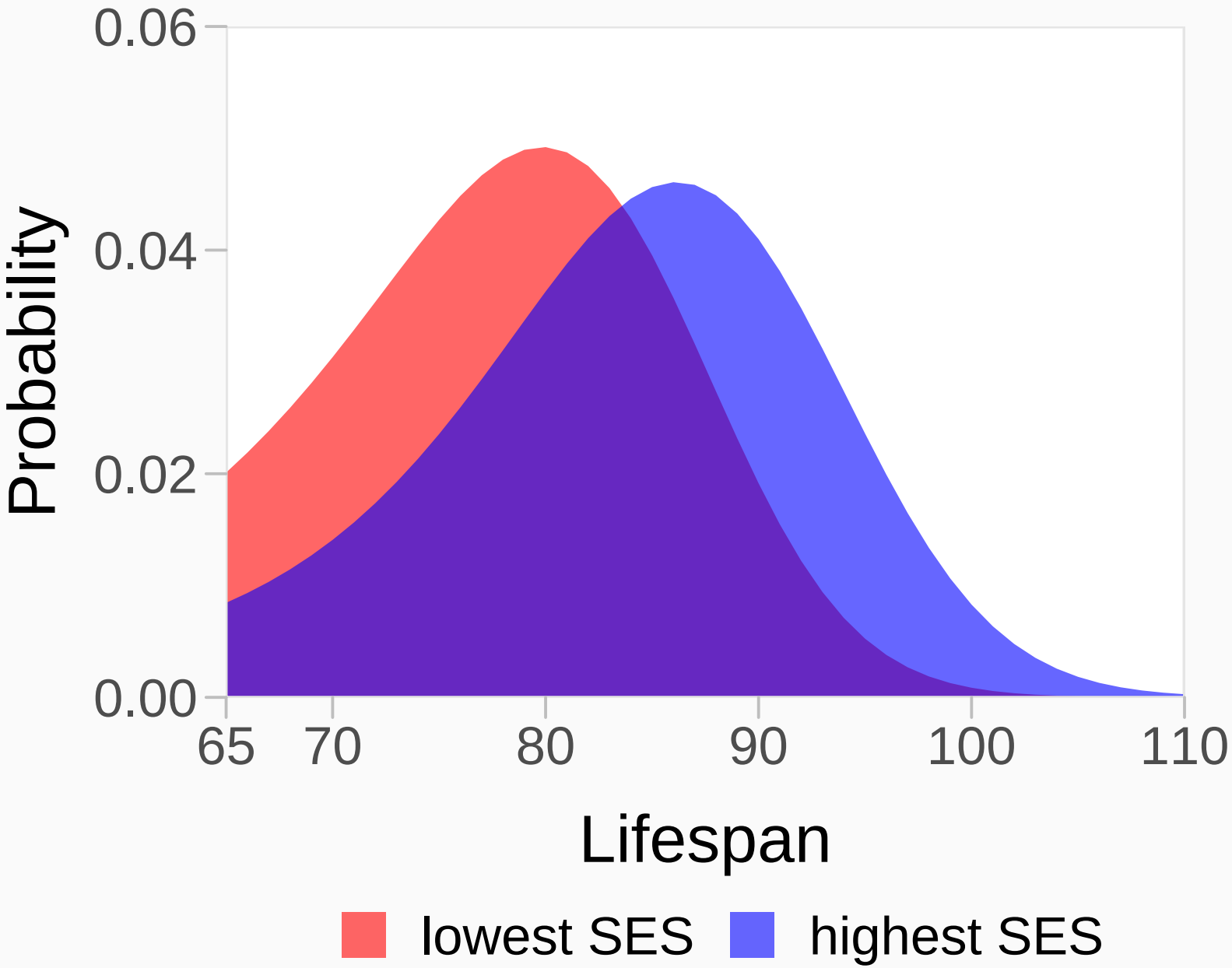
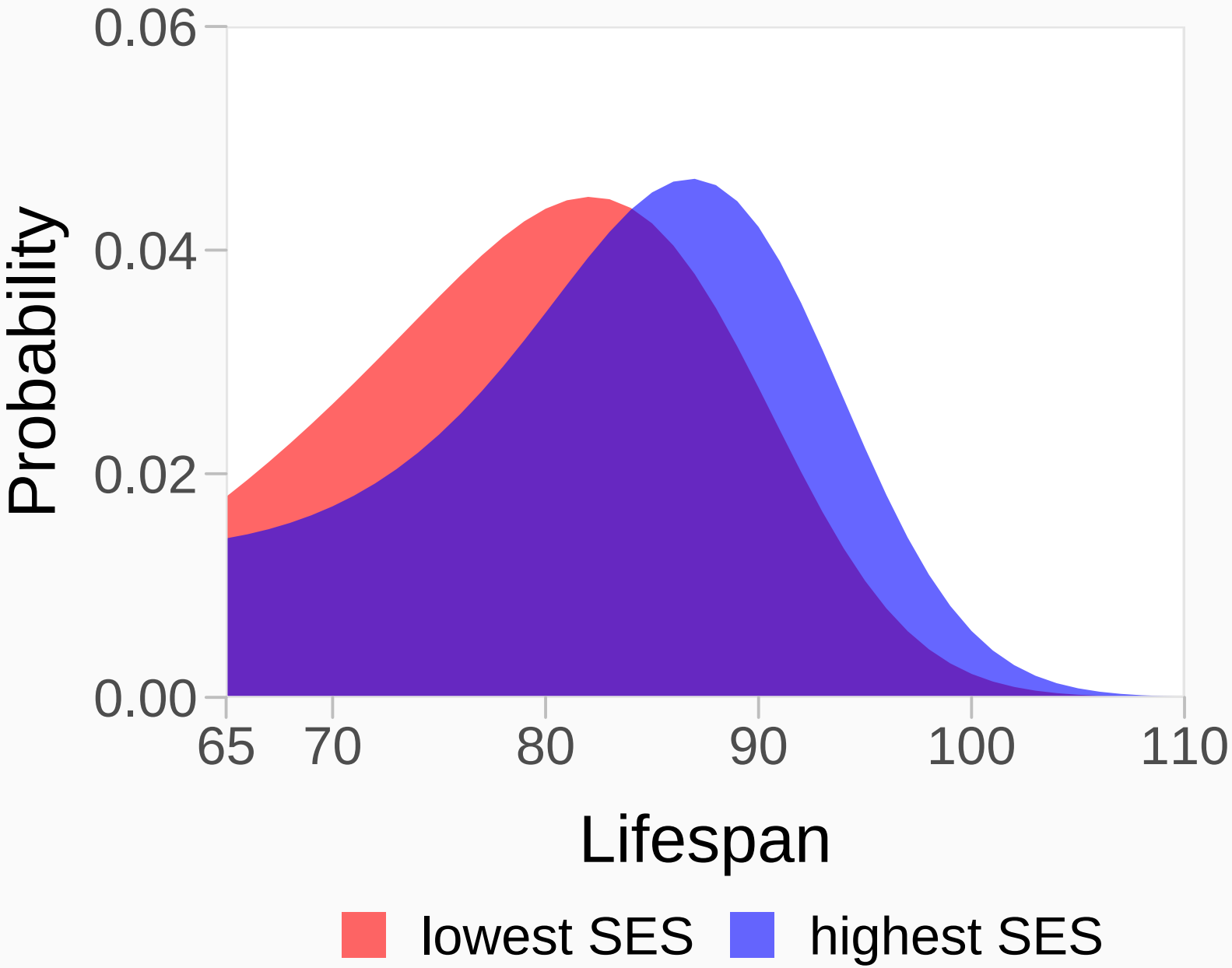
in the long run, it is expected that the indexation rule will **alleviate the burden of increased longevity.**

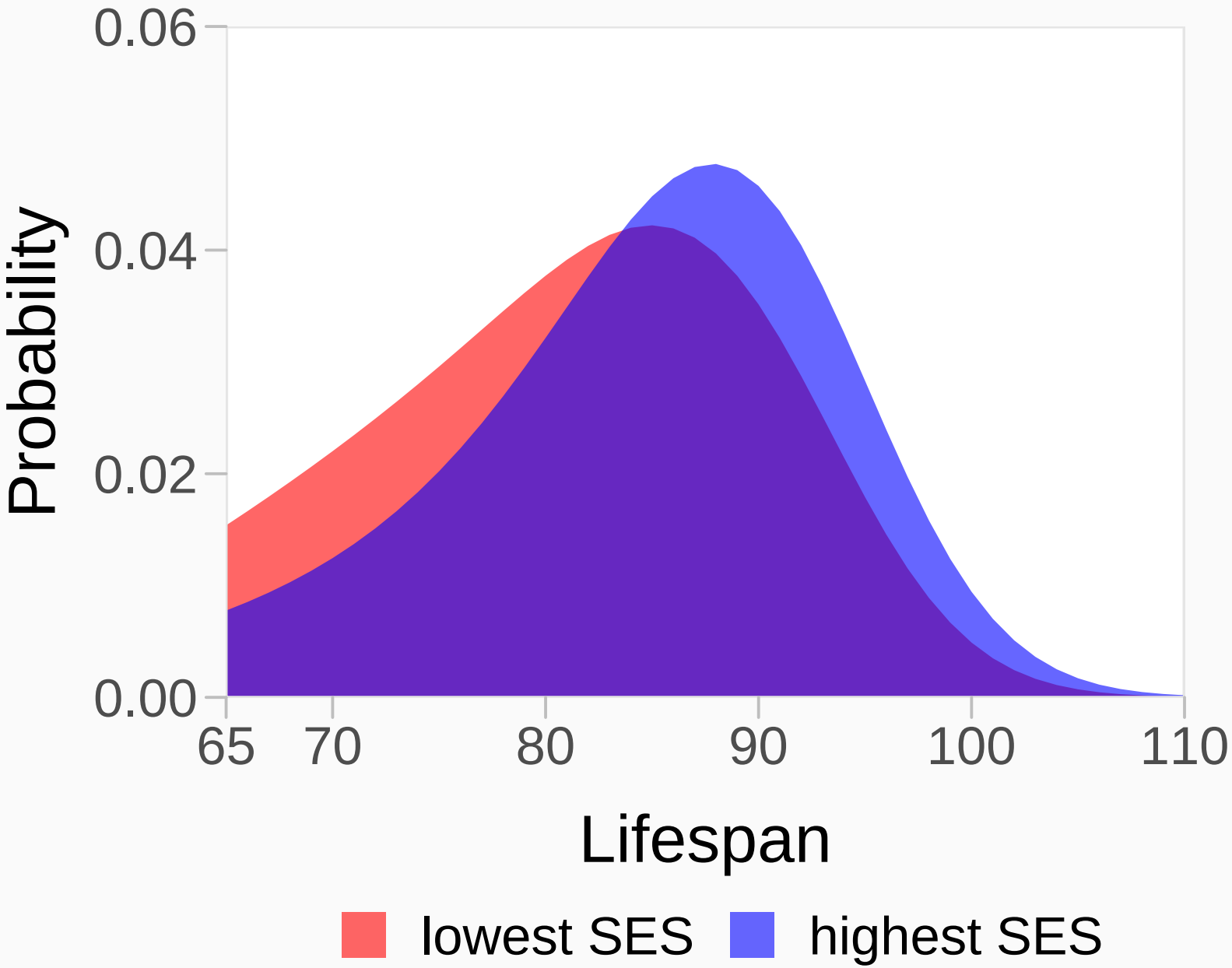
DISTRIBUTION OF LIFESPANS FOR DANISH FEMALES, 1985



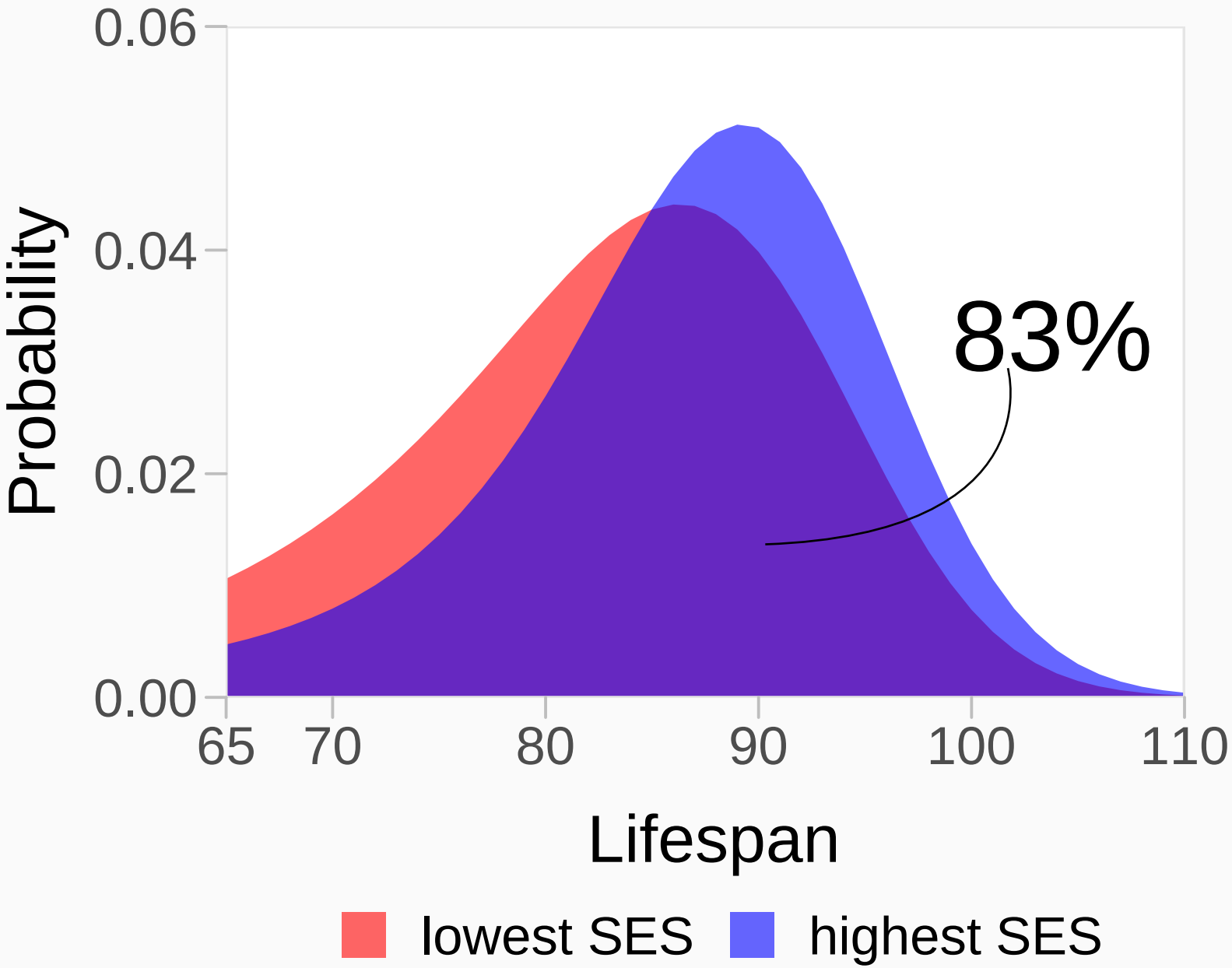
DISTRIBUTION OF LIFESPANS FOR DANISH FEMALES, 1995



DISTRIBUTION OF LIFESPANS FOR DANISH FEMALES, 2005



DISTRIBUTION OF LIFESPANS FOR DANISH FEMALES, 2016



LIFESPAN INEQUALITY AFTER RETIREMENT - MICRO LONGEVITY RISK

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- **Reduce the exposure to macro-longevity risk**: It is expected that individuals spend similar average times in retirement, regardless of the year in which retire.
- **Increase lifespan inequality $\rightarrow$ increases the exposure to micro-longevity risk**. This could be magnified for those socio-economic groups that exhibit a higher degree of lifespan inequality.

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- Compare both settings **retrospectively**.

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  - 5 equally sized groups (quintiles) over time.

Life expectancy

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If $H(x) = 0.4$, then a uniform reduction of one percent in the force of mortality at all ages above x will yield an increase of 0.4 percent in $e(x)$.

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If $\delta = 0$, then $\bar{a}(x) = e(x)$ and $\bar{H}(x, \delta = 0) = H(x)$

DECOMPOSITION OF DIFFERENCES OVER TIME IN H BETWEEN SES

The relative derivative of $\bar{H}(x, \delta)$ with respect to time:

$$\frac{\dot{\bar{H}}(x, \delta)}{\bar{H}(x, \delta)} = \frac{\dot{\bar{a}}^{\dagger}(x)}{\bar{a}^{\dagger}(x)} - \frac{\dot{\bar{a}}(x)}{\bar{a}(x)}.$$

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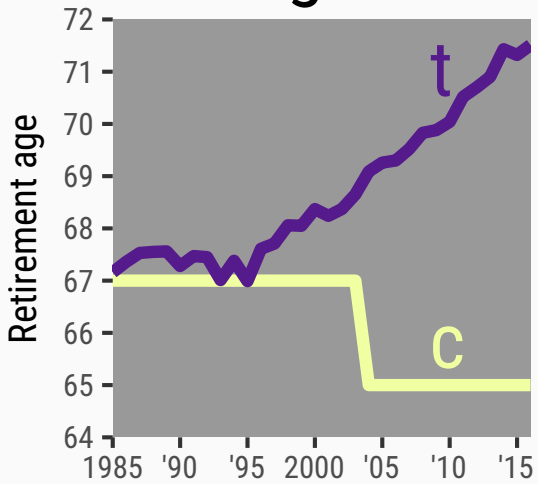
Translation effect: Differences due to **translation in the mean value of the life annuity factor**.

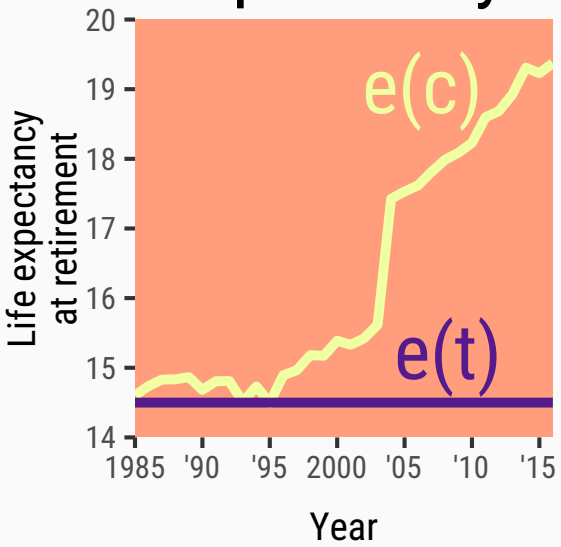
Demographic panorama after retirement, $\delta = 0\%$

DEMOGRAPHIC PANORAMA AFTER RETIREMENT

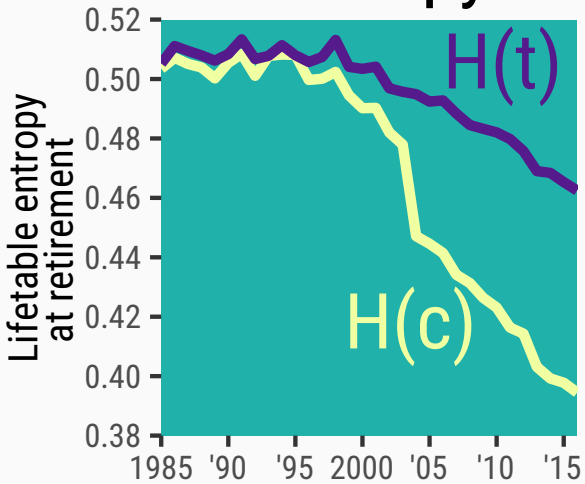
Retirement age



Life expectancy



Lifetable entropy

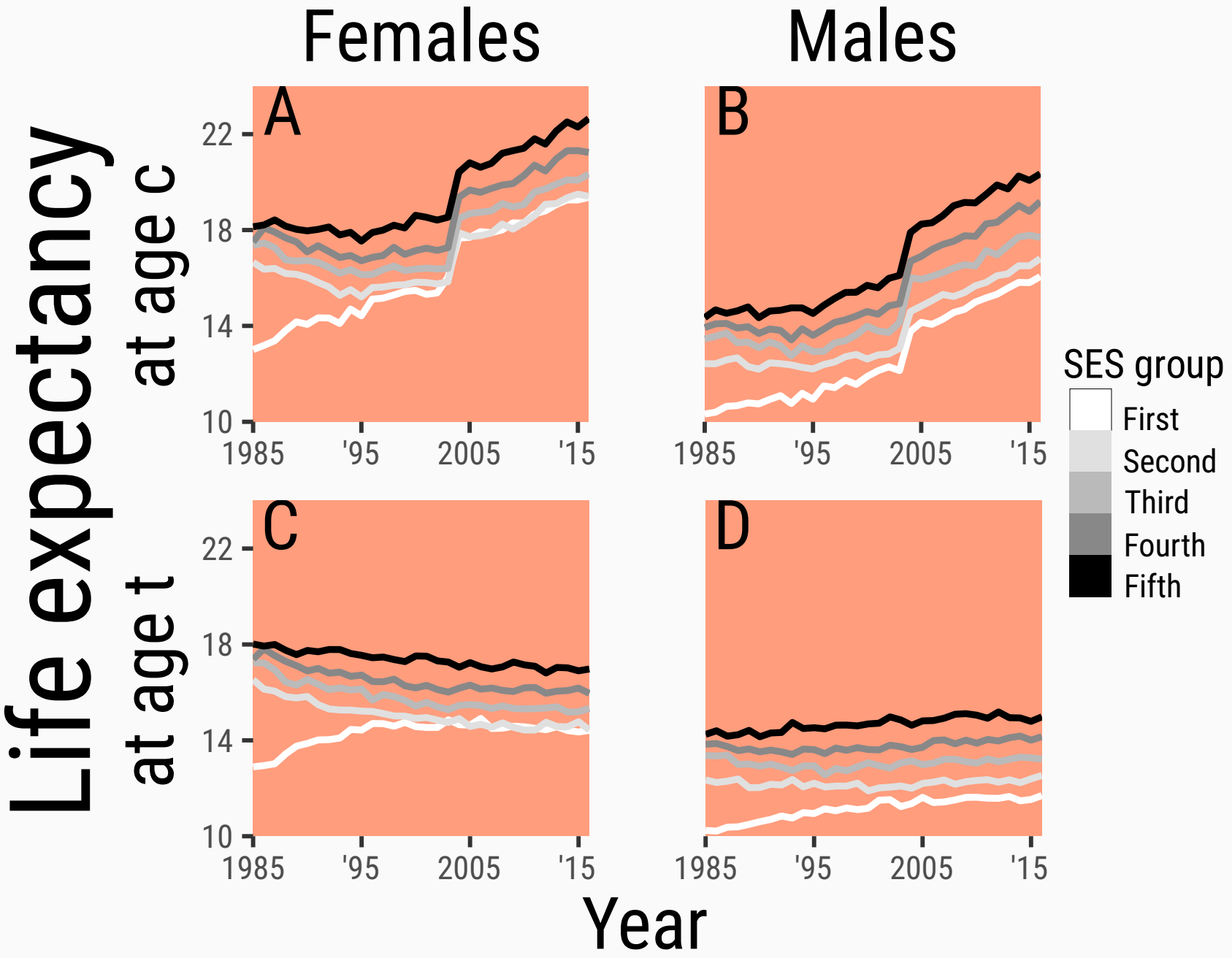


c: current retirement age

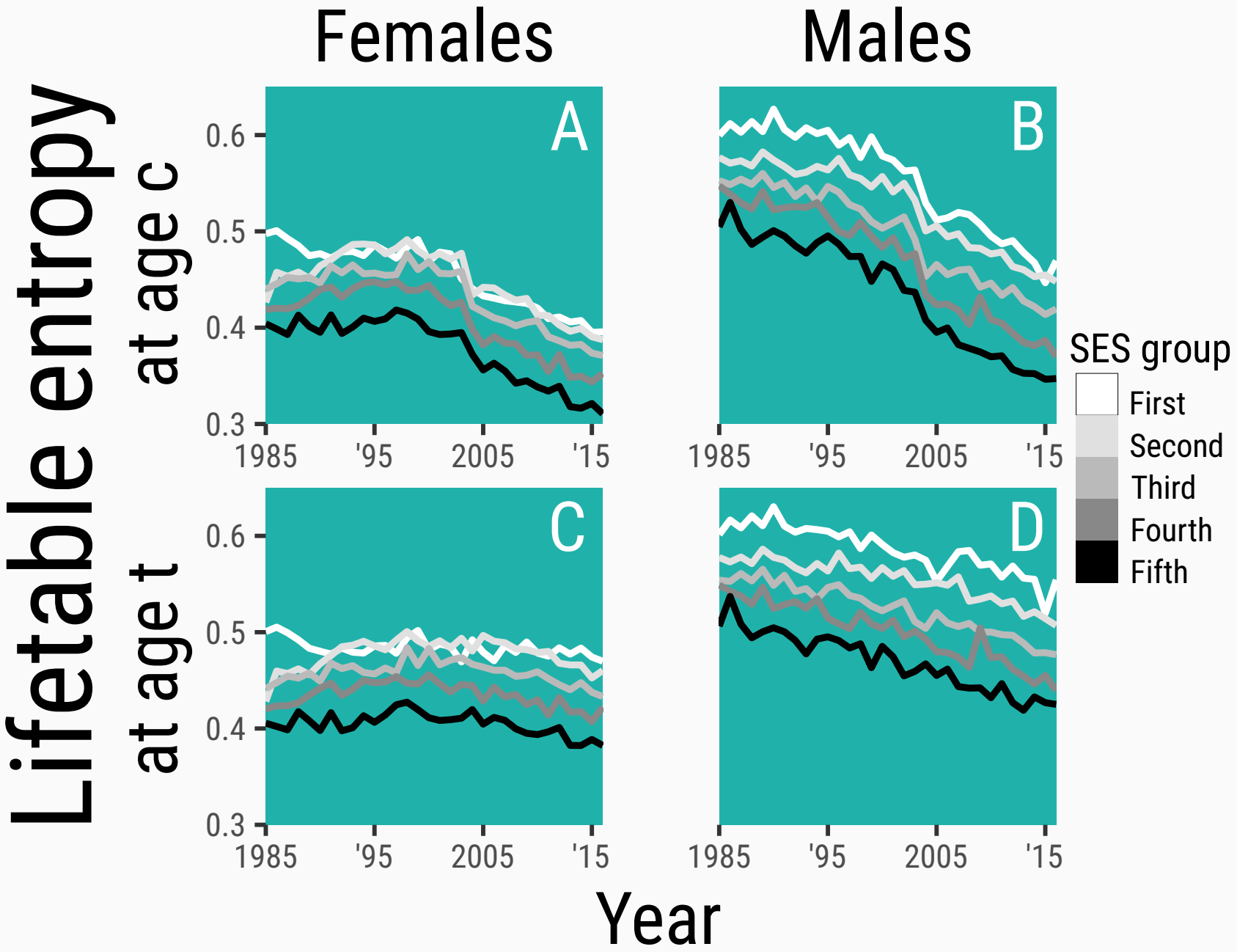
t: target retirement age

Demographic panorama by socio-economic groups

LIFE EXPECTANCY BY SOCIO-ECONOMIC GROUPS

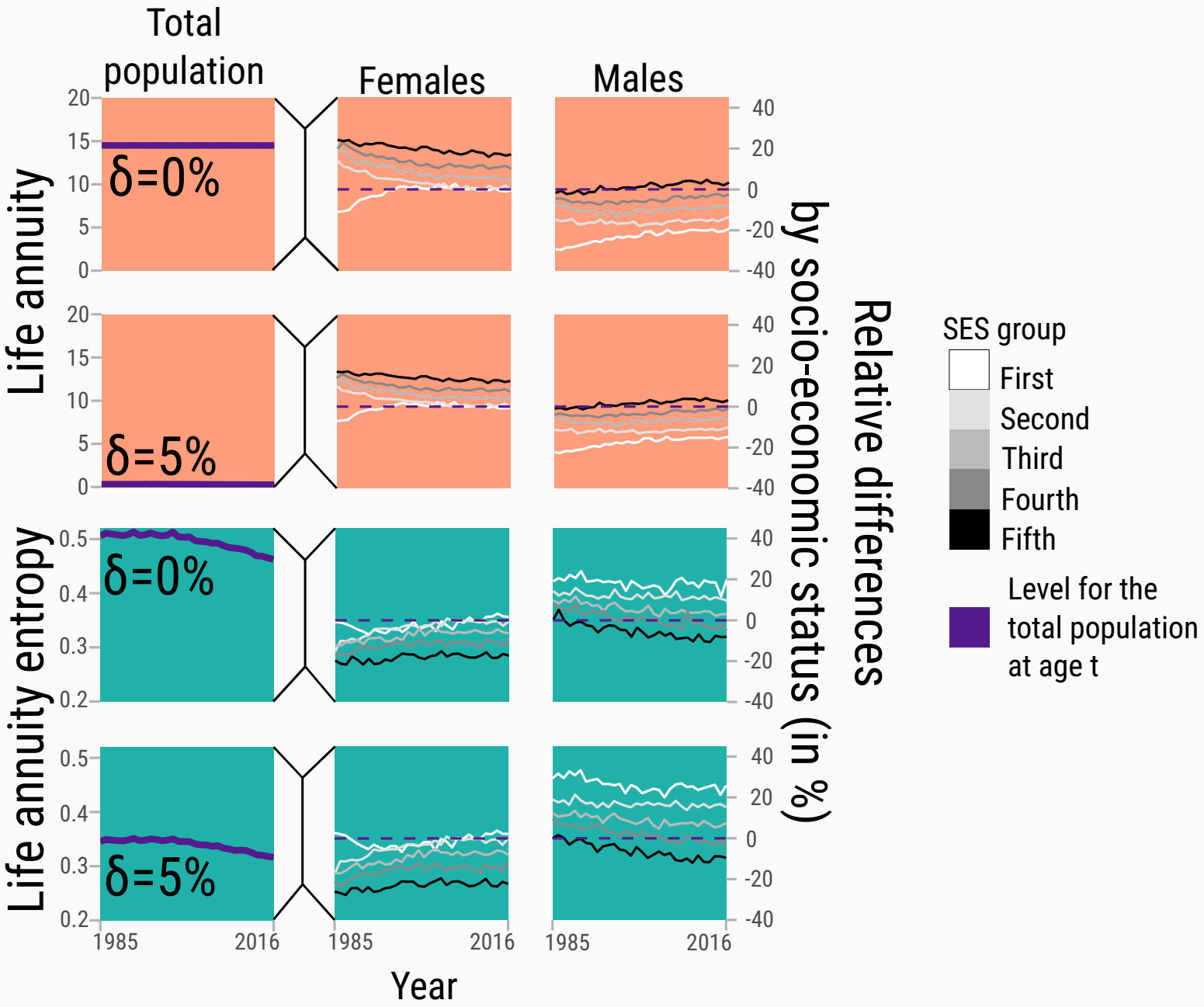


LIFETABLE ENTROPY BY SOCIO-ECONOMIC GROUPS

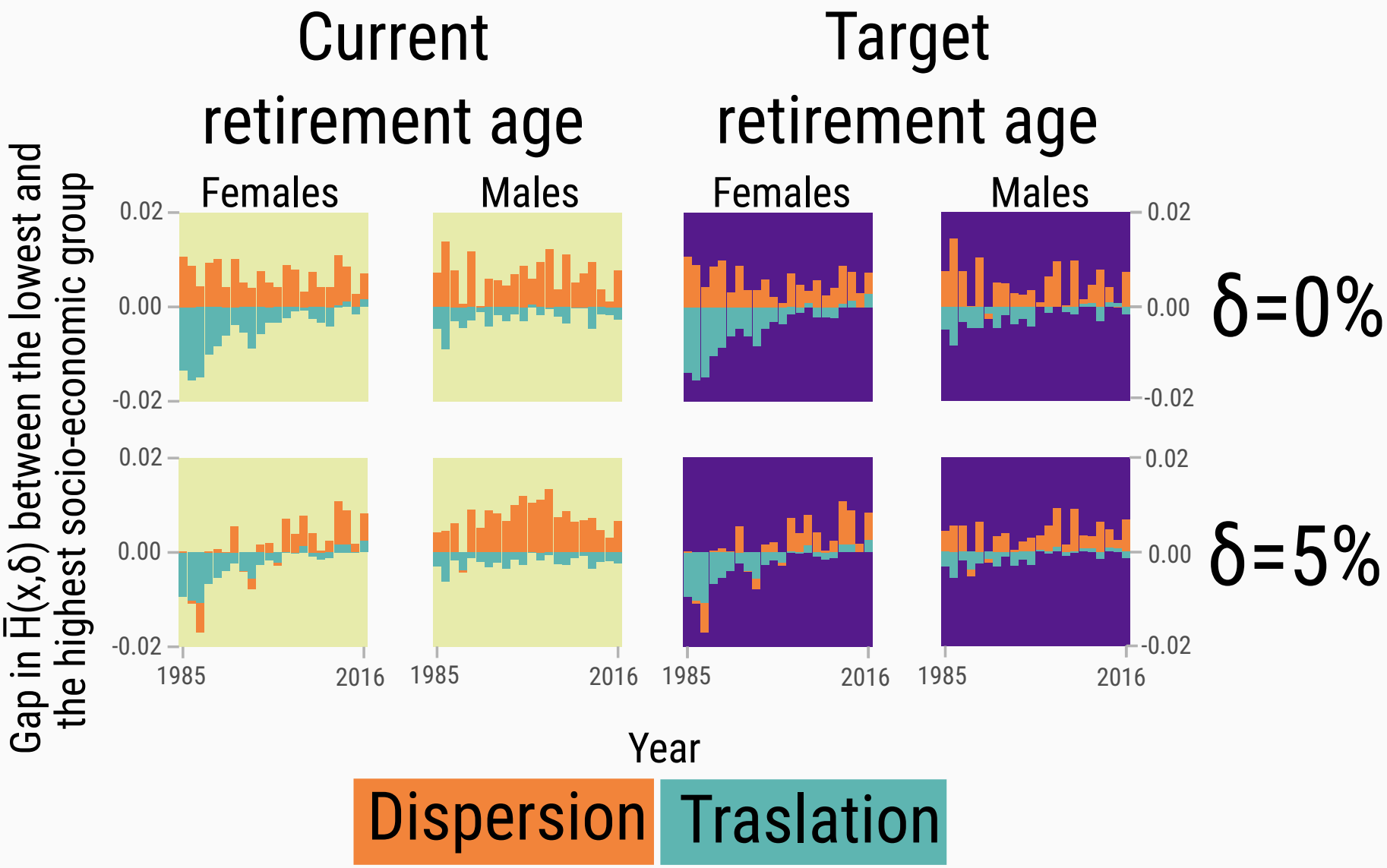


Actuarial perspective on socio-economic differences, $\delta > 0\%$

ACTUARIAL PERSPECTIVE AT THE TARGET RETIREMENT AGE



DECOMPOSITION OF DIFFERENCES BETWEEN THE HIGHEST AND LOWEST SES



To sum up

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Lifespan inequality increases with **life annuities becoming more sensitive to changes in mortality**.

- The exposure of pensions to **micro-longevity risk is higher**.

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Life annuities backed up with **riskier financial products** (higher interest rates) are crucial for **reducing** the exposure to **micro-longevity risk** (high lifespan inequality).

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Life annuities backed up with **riskier financial products** (higher interest rates) are crucial for **reducing** the exposure to **micro-longevity risk** (high lifespan inequality).

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- Such guarantees have been eliminated, allowing for investment of **pension wealth in riskier assets**, thereby yielding **higher returns** (Balter et al., 2018).

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- Such guarantees have been eliminated, allowing for investment of **pension wealth in riskier assets**, thereby yielding **higher returns** (Balter et al., 2018).

Even in this case, individuals will deal with **uncertain pension payments** under the target pension scheme, either through **higher micro longevity** risk or through **higher financial risk.**

IMPLICATIONS OF $e(t) = 14.5$ FOR SOCIO-ECONOMIC GROUPS

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Disadvantages are **magnified** when **retirement age is linked to life expectancy**.

Linking retirement age to life expectancy has detrimental implications for lower socio-economic groups.

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Retirement ages should be defined as a **trade-off between constant life expectancies and low lifespan inequality.**

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Implications to Retirement Planning – a discussion

January 13, 2020

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What I'll cover

- Brief overall comments
- Subramanyan paper
  - A few general comments
  - Sources of health risks
- Álvarez et al. paper
  - A few general comments
  - Inequality

Brief overall comments

- At first, these two papers don't seem related
- But both deal with implications of differences among people
  - Backgrounds and characteristics
  - Risks
  - Outcomes
  - "The average" person does not exist
- Both raise important issues that an actuary should consider

Subramanyan paper

- What is important for retirement planning
  - One size does not fit all, and never will
    - In terms of needs and mortality/morbidity risks
  - I agree that knowledge of personal risks is important for planning, but possibly more important is what an individual can do about it
  - Savings a function of
    - What a person has to begin with
    - Environment and subsequent human actions – societal and personal lifestyle
- The genetic makeup of a country's population does not change quickly
  - So, although these factors may be important for an individual, not so much for society

Subramanyan paper (2)

- Big data/artificial intelligence have potential
  - Data and resulting findings are only as good as data quality/availability
  - AI is only as good as its initial algorithm and assumptions
  - Privacy concerns are growing
- I agree with several points made in the paper, e.g.,
  - Biological age is more important than chronological age
    - But for retirement policy, only the latter is available
  - Distinction of what can be changed and what cannot
    - Family history, educational attainment, predisposition to certain diseases, genetic knowledge can be important but may be accompanied by possible anti-selection
      - Sound laws relating to use of genetic tests are needed
    - People who were given DNA-based information concerning their disease risks made little to no changes to their health behaviors – difficult to change behaviors
- Paper needs further editing and review

Causes of health risk

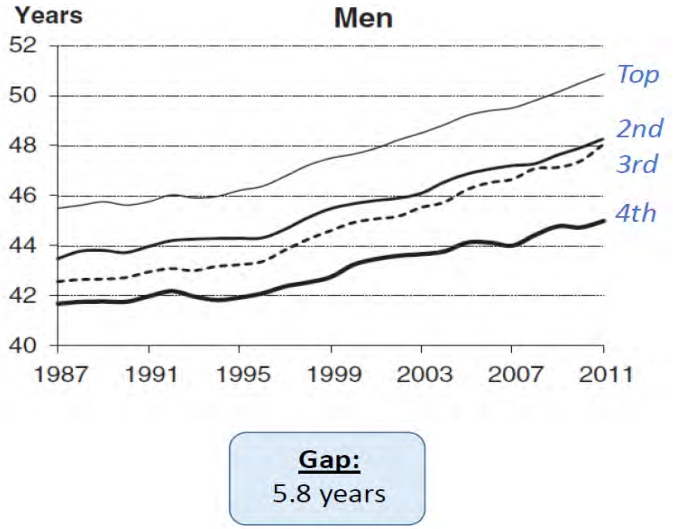
- Genetic
  - Evidenced through family history or genes
  - Although certainly relevant, the paper may have exaggerated its importance
  - Often, genes only indicate a predisposition to a condition
  - In most cases, more than a single gene is involved
    - The basis for life is rarely simple
- Environmental
  - Result of culture, past and current societal actions and medical developments/practices
- Personal behavior
  - Lifestyle factors, e.g., smoking, diet, physical activity
  - Individuals can only lever through personal action

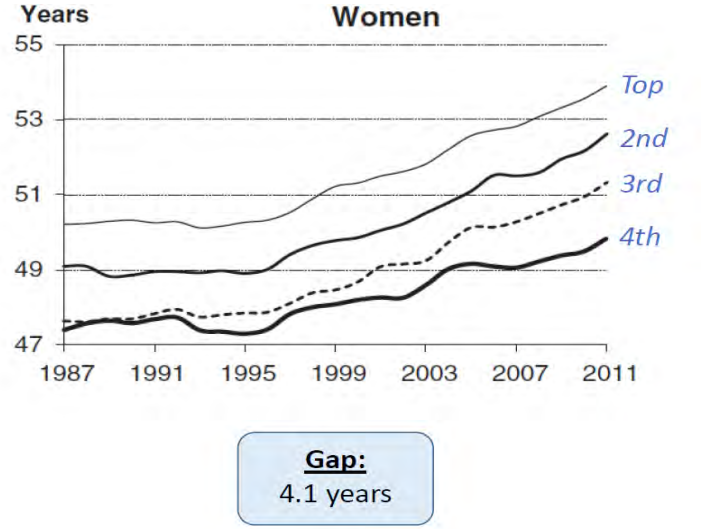
Álvarez et al. paper

- Overall, quite a good well-written paper
- Discusses issues associated with raising the retirement age
  - Often a huge political issue
  - Need to objectively address fairness between socioeconomic and sociodemographic groups
  - As pointed out in an IAA paper*, need to address treatment of those with lower income or disabled
- Interesting focus of this paper is that distributions (through longevity inequality) should be reviewed, not just expected values

*IAA Population Issues Working Group discussion paper (2016): "Determination of Retirement and Eligibility Ages: Actuarial, Social and Economic Impacts"

Interesting study of Danish mortality by educational attainment – Life expectancy at age 30





Bronnum-Hansen, H., M. Baadsgaard (2012). "Widening social inequality in life expectancy in Denmark. A register-based study on social composition and mortality trends for the Danish population". *BMC Public Health*. 2012 Nov 17;12:994.

Inequality

- Inequality aspects*
  - Inequality is inevitable
  - Opportunity (ex-ante) versus Outcomes (ex-post)
  - Actuaries typically estimate expected costs associated with ex-ante (expectations of future longevity) based on results (ex-post)
  - Need to consider a wide range of risks, characteristics and stakeholders

*IAA Population Issues Working Group discussion paper (forthcoming). “Actuarial Perspective on Inequality”

Álvarez et al. paper

- Agree with conclusion that financial planning needs to be conducted with respect to the particular condition of the individual, rather than just averages
- Program design should address the needs of a range of individuals
- Agree that linking retirement age to life expectancy enhances intergenerational equity, but unless modifications in plan design occur, intragenerational socioeconomic and sociodemographic differences can have detrimental implications for lower socio-economic groups
- Inequalities / heterogeneity will always exist
  - But will change, as conditions will change
- But need to understand why, rather than just what

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2020 Living to 100 Symposium

N.V.SUBRAMANYAN

1B – Implication to Retirement Planning

A Study into the Impact of Pre-Programmed Genetic Health Risks in Retirement Planning

Date: 13th Jan' 2020

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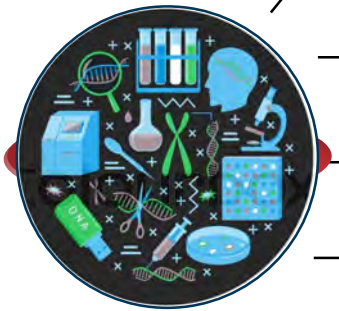

- Synopsis
- Executive Summary
- DNA Based Pre-Programmed Health Risk
- Impact, scope and potential for retirement planning
- Regulatory impact and interventions
- Opportunities for Society, Institutions & Individuals
- Conclusion

Synopsis

1. Human life expectancy and prognosis of health conditions is possible based upon the genetic profile of people
2. With developments in genetic research, it is possible to reasonably identify pre-programmed health risk depending on a persons' DNA.
3. It can be reasonably identified how long and how healthily might one expect to live with DNA study through Genetic Scores, Disease Association Analysis, Polygenic Score Analysis and other methodology.
4. Impact of family history with respect to longevity of persons is there and possible to be identified.
5. There is need to have a framework of regulations to handle the situations arising from these developments.

Executive Summary

1. Genetic testing and the consumer wellness genomics market is anticipated to reach USD$4.6 billion by 2025.
2. Several genetic variants have been discovered including the greater risk they confer for various diseases
3. Research shows that humans are pre-programmed in DNA to develop certain diseases due to DNA mutations and targeted treatment is possible:

    a) innate carcinogenesis through co-occurrence of metastases caused by Quantum Entanglement Entropy
    b) Heterochromatin Protein (HP1) acts as epigenetic marker for colon cancer and CRISPR-Cas9 enzymes act like molecular scissors or genetic scalpels to delete defective genetic code and swap a replacement.
    c) Pre-programmed propensity to diseases is not unique to Colon cancer alone but applies to other cancers and diseases too.
    d) Evidence links aging to genetic and epigenetic alterations and with the reversible nature of epigenetic mechanisms, they provide promising avenues for prediction and intervention against aging decline and disease.

        i. Manipulations of genes have been found to increase longevity in C'elegans and replicated in some invertebrates and some few higher life forms.
        ii. DNA methylation age of blood can predict all-cause mortality in later life and studies have identified biomarkers of chronological age based on DNA methylation levels.

Executive Summary (Contd)

1. Epigenetics offers the most promise in old age management through:
   a) Gene control: Epigenetics determines cell specialization and, through environmental stimuli, can cause genes to be turned on or off
   b) Universality: Diet, lifestyle, sleep pattern, exercise etc. can lead to modifications around genes to turn them on or off to predict cancer, Alzheimer's etc.
   c) Inheritance: Enables to understand ourselves and indications are some epigenetic changes can be inherited.
   d) Reversibility: With over 20,000 genes, we could theoretically address most old age issues viz. control cancer, slow aging, stop obesity etc.
2. Epigenetic changes are dictated by environmental stimuli and nutrient availability that alter intracellular metabolite concentrations.
3. Genetically tractable models can be used to investigate both replicative lifespan and chronological lifespan
4. If we consider the combination of DNA methylation & histone modification patterns, with the possibility of paramutation, it would be possible for epigenetics to unravel complexity of genetics of common diseases.

DNA Based Pre-Programmed Health Risk

1. Role of DNA is more than the way one resembles family members and can be a predictive tool.
2. Genes and their mutations are responsible for health and how long one could live.
3. Diseases related to heritable gene mutations impact lifespan like cystic fibrosis, sickle cell anemia, Tay-Sachs and Huntington's.
4. Mutations could be harmful or beneficial. Eg. Huntington's and Sickle cell v/s mosquito vector borne diseases.

Theories of Aging

1. Accumulation of damage:
   a) Wear and tear theory of aging
   b) Rate of living theory of aging
   c) Protein cross-linking theory of aging
   d) Free radical theory of aging
   e) Somatic mutation theory of aging
2. Programmed longevity:
   a) Programmed longevity - sequential switching of genes
   b) Endocrine theory of aging
   c) Immunological theory of aging
3. Telomere lengths
4. Longevity genes: specific genes associated with living longer eg. SIRT1 and SIRT2 polymorphisms associated in disease related phenotypes like diabetes, obesity, cholesterol metabolism, cardiovascular disease etc.
5. Stem cells - these are immature cells which can potentially become many types of cell which can be used to further longevity as stem cells are only a small number of the cells present in the body.
6. Epigenetics - refers to gene expression within the constraints of genetics to protect or predispose to disease.

Genetics explains ~35% of lifespan conditions. Aging is a multifactorial process including human behavior, exposures and random chance.

Main uses of genetic testing

1. Get a brief overview of the current state of human disease mapping to provide foundational knowledge for genetic-based disease prediction
2. Describe process of disease prediction in a simple probabilistic framework
3. Provide overview of the basic classes of genetic-based prediction models and measures of prognostic utility
4. Illustrate application of genetic-based predictive models to data from biobanks and prospective cohorts.
5. Ideal genetic-based predictive model for clinical applications should:
   a) Markedly modify posterior probability of traits vis-à-vis existing clinical assessment
   b) Impact majority of individuals and provide improved outcomes.
   c) Allow broad applicability that defines an archetypal genetic-based predictive model.

Predictive Models

1. Regression methods for prediction modelling
2. Use of Bayesian networks has promise to obtain posterior probabilities: the posterior probability of the disease trait as: $PPD^n = P(D||\cap^{ni=1} G^i) = P(\cap^{ni=1} G^i | D)P(D) / P(\cap^{ni=1} G^i)$
   - where D denotes a random variable for the disease trait and n genetic markers are used in the prediction. Under the conditional independence assumption of naive Bayes, we can completely factorize the product and, for a binary trait (D = 1 to denote disease and D = 0 for non-disease), the PPD is:
     - $PPD^n = P(D=1)\prod^{ni=1} P(G^i|D=1)P(D=1)\prod^{ni=1} P(G^i|D=1) + P(D=0)\prod^{ni=1} P(G^i|D=0)$.

Predictive Models

1. Prognosis of disease traits with genetic information are classical problems of classification and clustering within machine learning.
2. Need of large computational power to crunch the huge amounts of data is very much there in the current stage. Machine learning and Artificial Intelligence (AI) would help to do this efficiently.

Analysis of Individuals DNA

1. Most diseases are linked not to multiple mutations each effecting the odds of getting a disease
2. Genetic research has determined that all persons, no matter the geographic origin, are about 99.5 percent the same genetically. Studies of twins have shown that approximately 25 percent to 50 percent of morbidity and mortality differences from person to person are due to genetic variation.
3. DNA analysis for risk prediction could be through risk scores or regression analysis.
    a. Risk scores method is like numerical rating method of underwriting which constructs the predictive model based on sum of predisposing genotypes each individual carries, unweighted or weighted by the effect size of specific predisposing genotypes. Basic approach is to take weighted sum of risk alleles, choose risk alleles based on those found to be genome-wide significant in a recent meta-analysis.
    b. Gene-based multiple regression association testing is more for combined examination of common and low frequency variants in quantitative trait analysis using multi-marker methods.
4. With respect to individual mortality, biological age perhaps is more important than chronological age as patients diagnosed with diabetes or hypertension are biologically older than healthy peers and the difference translates into a lifespan change.
5. Summary of Kaplan-Meier survival curves, stratified into the high- and the low- risk groups according to the difference between the estimated biological age of an individual and the averaged estimated age of gender- and age-matched peers gives a clear understanding of the differences.

Analysis of Individuals DNA

- Kriging, a geostatistical interpolation technique, can also be used in predicting genotypes and phenotypes and overall genetic values in predicting individual mortality values. Predictions of genetic predispositions to human diseases should be useful for preventive and personalized medicine. The kriging approach consists of:
  - Prediction of the values of the regionalized variables by performing a best linear unbiased prediction, under the auxiliary assumption that the parameter values and hidden variables estimated in the first step are the true ones
  - If the study group has q individuals with family history information, n of them being genotyped and having phenotype measurements of a certain quantitative trait, the overall model could be: $y_i = w^T_i\beta + z^T_i u + g(x_i) + e_i$ for i =1 to n
    - with y_i as a measurement of the phenotype for individual i, β is a function of nuisance location parameter not of immediate interest but useful later, x_i is a p-vector of dummy SNP instance variates (genotype) observed on individual i, and g is a random function as a Gaussian random field. In matrix notation, the statistical model reduces to: $y = W\beta + Zu + g(x) + e$.
- A single method may not always be adequate, and a combination of methods would be needed

Impact of family history

- It is known that while we can reduce our risk of disease with healthy diet, exercise and not smoking, our family history is one of the strongest influences on risk
- We cannot change genetic make-up but family history helps identify inherent risks. With respect to family history, the primary factor of DNA is that it:
  - is unique to everyone
  - has predictive powers
  - may affect relatives
  - may be used to discriminate
  - emotionally affects persons
- Key red-flags in family history that may increase risk are diseases:
  - occurring at an earlier age than expected (10 to 20 years before most people get the disease)
  - in more than one close relative
  - that do not usually affect a certain gender (for example, breast cancer in a male) and
  - certain combinations occur within a family (for example, breast and ovarian cancer, or heart disease and diabetes)

Impact of family history

1. If a family has one or more adverse or even positive features, it holds important clues about risk.
2. People with family history of disease gain the most from lifestyle changes and screening tests.
3. Adopting a healthier lifestyle and screening tests can reduce one's risk for hereditary diseases. can detect diseases and help address:
   a) Major medical conditions and causes of death
   b) Age of disease onset and age at death
   c) Ethnic background
4. Various approaches to analyze the polygenic effect of family history on the prediction accuracy can be devised, for example the polygenic effect "α" (or a polygenic score) of a child can be calculated as: ½ * ($\alpha_{Father} + \alpha_{Mother}$) + m, where m is its Mendelian sampling term drawn from a normal distribution n from a normal distribution N (0; 0:25*(2-($F_{mother} + F_{father}$))*σ^2_{poly}) with F_{mother} and F_{father} being the inbreeding coefficients of the corresponding mother and father.

Impact of family history

As per the study by the National Cancer Centre Singapore, over 400 hereditary cancer susceptibility syndromes have been described, most of which feature an autosomal dominant inheritance pattern. Although many of these are rare syndromes, they account for at least 5–10 percent of all cancer incidences.

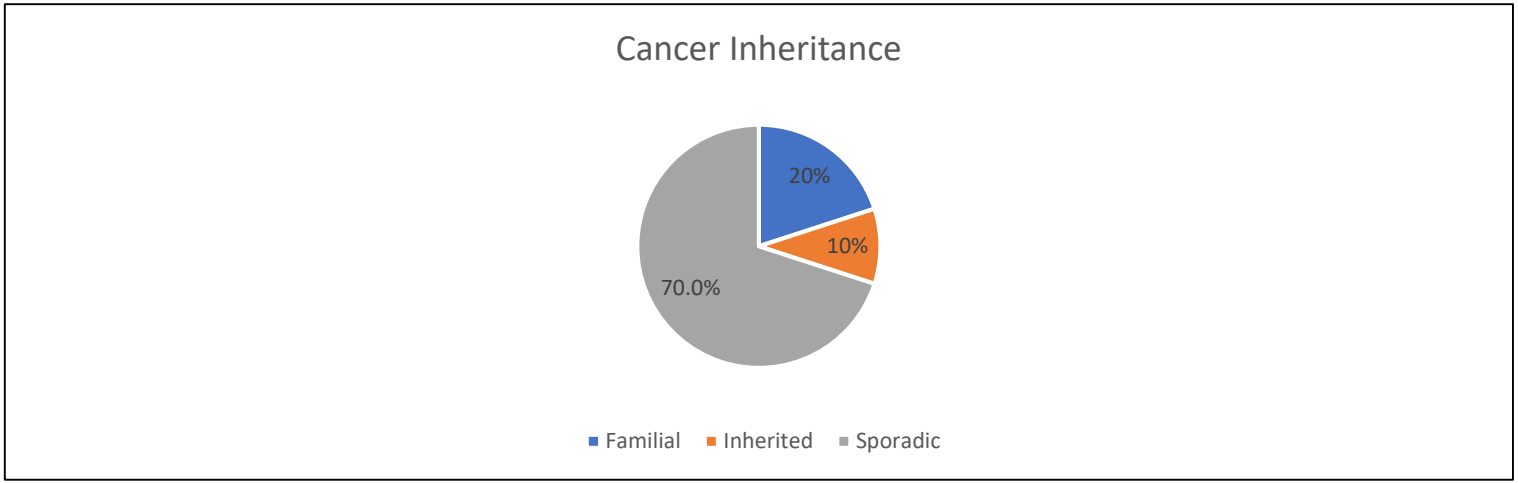

Figure : Inheritability experience of Cancer in Singapore

Impact of family history

On further study, an inherited cancer susceptibility syndrome is usually suspected in families with the following characteristics:

a. Two or more relatives with the same type of cancer on the same side of the family – this likely aggravates the risk.
b. Several generations affected – more the number of generations affected, more serious is the risk.
c. Earlier age of cancer diagnosis than typically seen for that cancer type
d. Individuals with multiple primary cancers
e. The occurrence of cancers in one family, which are known to be genetically related, such as breast and ovarian cancer, or colon and uterine cancer and
f. The occurrence of non-malignant conditions and cancer in the same person and/or family

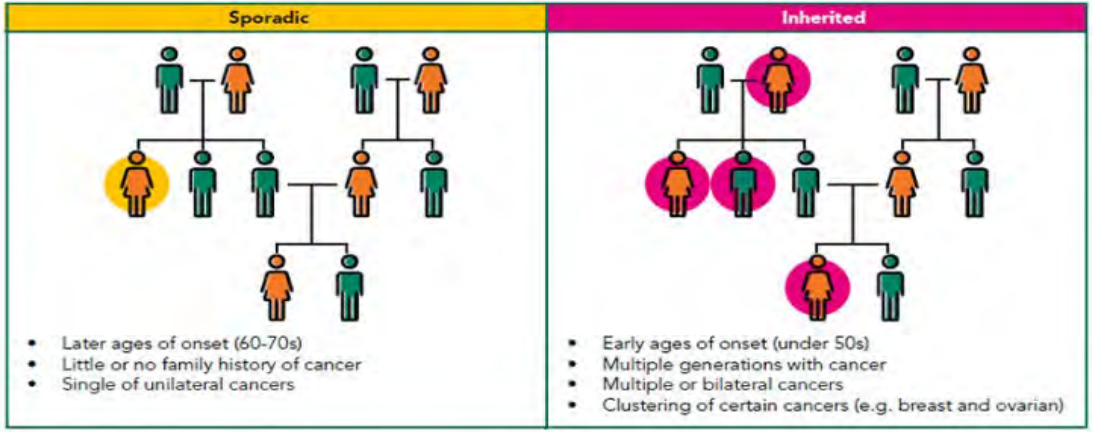

Figure : Characteristics of Sporadic Vs. Inherited Cancer in Singapore

Impact of family history

Markov models provide promise on studying impact of genetic testing. A sample age-wise model can be defined for population at time zero, for different family history. A simple example is shown above with three sub-populations:

(a) persons with no family history and therefore not at risk of a genetic disease (i = 1)

(b) persons at risk because of family history but do not in fact have the mutation (i = 2) and

(c) persons at risk because of family history and have a mutation (i = 3).

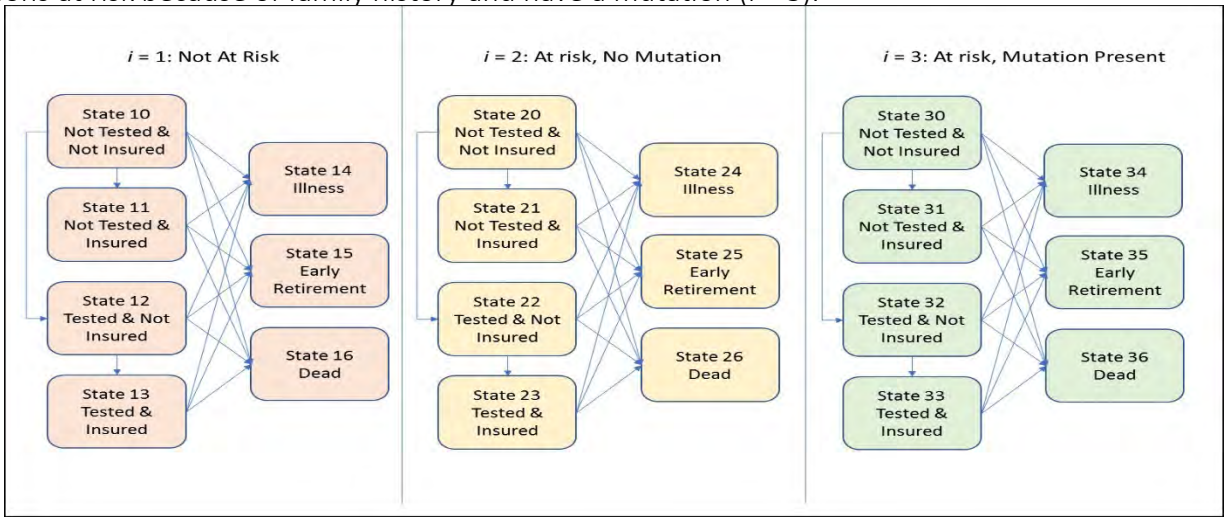

Figure : Markov model for transition with insurance including Retirement plan

Potential for retirement planning

1. DNA testing should allow be a useful tool for retirement planning also by answering the question *“Will I outlive my money?”* or *“How much longer can I work”* or *“What will be the status of my health post 70?”*. Knowledge about future health and longevity would be very helpful for retirement planning.
2. With advances in genetics and computing, tools are available for planning. After considering heredity, lifestyle, DNA testing would allow to predict future health and elder care costs. Financial plan can be adjusted between life-insurance saving and retirement planning more efficiently.
3. Insurance might be used to mitigate some financial risks. Better understanding of how genetic testing impacts mortality should help identify potential implications for insurance and provide useful information in shaping policy. Mostly only the applicant is aware of genetic results and family history and exposure of insurer to these is minimal. So, impact on preferred classification and change in policyholder behavior should be addressed. This aspect mainly affects:
    a) Long Term Care insurance
    b) Disability insurance,
    c) Critical illness insurance

Regulatory impact and interventions

As new testing becomes more affordable and more widespread, life and health insurance companies need to monitor the emerging trends in genetic science closely. Working with regulators, medical professionals, industry groups and genetic councilors to agree on reasonable self-regulation in the field of genetics may be a prudent approach to staving off unwanted restrictive regulation.

Figure : Present Regulatory scenario in major economies

Opportunities for Society, Institutions and Individuals

- Genetic testing to be used wisely to improve the standard of life, price accurately & better financial planning
- Synergy can be generated and channelized between the health industry, testing laboratories & insurers
- Genetic testing can help to avoid unnecessary clinical investigations, help choose suitable therapy and allow financial planning:
  - HSBC Insurance (HK) provides health screening (ONEdna) to obtain better information about their health status risks to allow accurate formulation of an optimal diet and sensitivities to many common medications.
  - AIA (HK) offers “Smart Elite Ultra” critical illness protection solution with an AIA Vitality Selected Insurance Product to get “fitlife Health Coaching Program and Genetic Test” to tailor their health program.
  - Prudential Singapore offers myDNA, Singapore's first health & wellness program, to help make better lifestyle choices
  - Thailand’s Muang Thai Life Assurance: through ‘My Thaidna’ and ‘SmileCare’, initially, consumers get a blood test, and are encouraged to do so in 6-month intervals; if their glucose levels improve, their next premium is reduced. The data is the property of Muang Thai & uses it for pricing.

Opportunities for Society, Institutions and Individuals

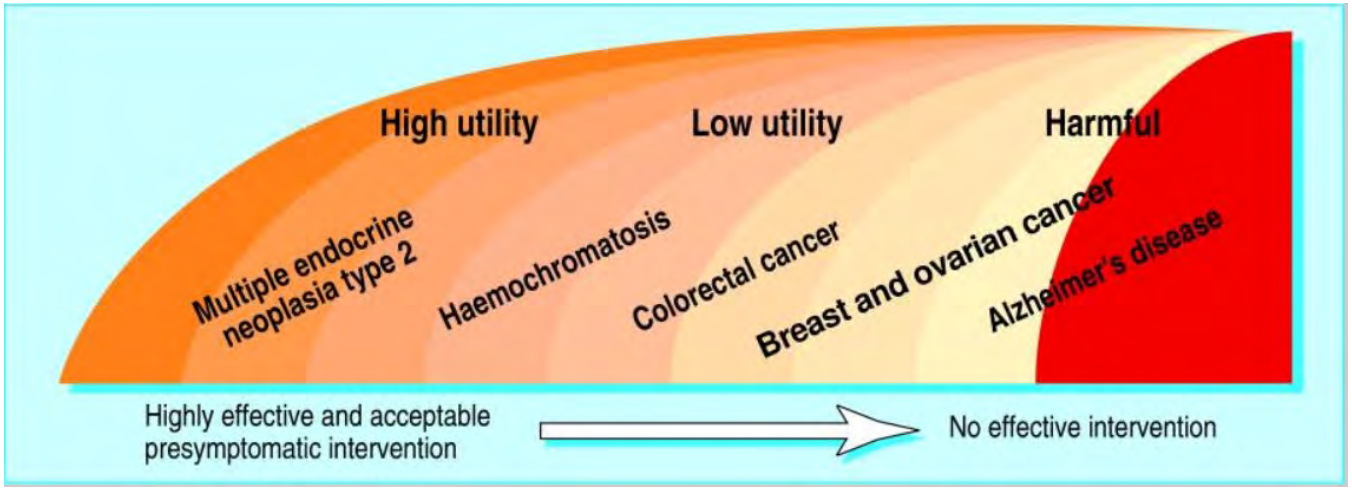

Figure 6: Utility of predictive genetic testing for different diseases

Source: The complexities of predictive genetic testing

Conclusion

1. Much of the impact of DNA testing is in handling the uncertainty it brings, and assumptions are needed in two categories: impact on consumer behavior, and impact on mortality and morbidity outcomes.
2. Genomics, genetic testing, and precision medicine will play a rapidly increasing role in patient care and disease prognostication and ultimately lead to improvements in morbidity and mortality.
3. But is absolute change of human behavior vis-à-vis the results of a genetic test or medical advice a universal fact? Available evidence does not to support this view; at least not wholly.
4. Access to genetic testing can improve patient care and could be incorporated into insurance products for policyholder benefit.
5. Extra-genetic factors play a bigger role on human health and longevity
6. While an immediate concern for the industry may not be there, it helps to research more into the area to be aware of the impact it entails

Thank you!