Session 134 PD - Reducing Risk of Anti-selective Behavior in Underwriting Today and in the Future

Moderator:
Richard C. Pretty, FSA, MAAA

Presenters:
Peter Banthorpe
Maria Govorun
Reducing Risk of Anti-selective Behavior in Underwriting Today and in the Future:

Anti-selection risk from advances in statistical Genetics

Peter Banthorpe
SVP, Global Head of Research and Data Analytics

October 2017
Agenda

- Use of Genetics in Insurance
- Scientific Background
- Genetic Risk to Disease and Polygenic Risk Scores
- Genetics and Risks of Anti-selection
- Conclusions
Use of Genetics in Insurance
Genetics has always elicited a varied set of views across stakeholders

**DNA and Insurance, Fate and Risk**

**Introduction**

As costs for DNA sequencing drop, hundreds of thousands of Americans are undergoing the procedure to see if they are at risk for inherited diseases. But while federal law bars employers and health insurers from seeking the results, insurers can still use them in all but three states when considering applications for life, disability and long-term care coverage.

Should insurance companies be barred from seeing genetic information when considering those policies so people can get the tests without fear that the results would be used against them?

**Debaters**

- **Risks Are Too Small for Insurers to Worry**
  
  Angus S. MacDonald, Professor of Actuarial Mathematics
  
  Only the rarest hereditary disorders would create a major cost burden for insurers. They should agree to ignore genetic tests, and avoid a legal ban.

- **Guarantee Privacy to Ensure Proper Treatment**
  
  Jeremy Grudziak, Council for Responsible Genetics
  
  If the promise of the genetic revolution is to be fulfilled, the public must know that genetic testing will not endanger their economic security.

- **Questions Remain; Some Rules Should Be Clear**
  
  Francis S. Collins, National Institutes of Health
  
  Even without barring insurers from seeing genetic tests, such tests should not be demanded of anyone. And research data must be kept private.

- **It’s Yet to Be Shown That Discrimination Exists**
  
  Bartha Maria Knoppers, McGill University
  
  Only rare conditions can be predicted with certainty, and insurers can already access a variety of hereditary information about applicants.

- **Let Insurers Have Data and Trust to Get It Right**
  
  Sarah Hanson, American Council of Life Insurers
  
  Advances in medicine have made it possible for insurers to offer coverage to more people, not fewer.

- **Test Results Are Not Always What They Seem**
  
  Joy Larsen Haddad, National Society of Genetic Counselors
  
  Even if insurers are allowed to consider the tests, they need to ensure they fully understand what results do and do not reveal.

Increasing levels of interest in Genetics and Genomics* for medical applications

High degree of promise

- Prevention of disease manifestation
- Motivate Lifestyle modification
- Precision medicine
  - Pharmacogenetics
  - Cancer treatment
- Prenatal and Newborns screening
- Accurate diagnosis of rare disease
- More accurate disease prognosis
- Disease recurrence detection
- Everything!

Falling costs and increased availability

- The first human genome took $2.7 billion and almost 15 years to complete.
- Now it costs about $1,000 and the sequencing can be done in a few days.
- In a few years it may only cost $100.
- Multiiple providers of D2C testing

- Genetics is the study of inherited traits and genes. (simplistic view)
- Genomics is the study of how a set of genes behave. (complex view)
Increasing levels of interest in Genetics and Genomics* from governments and regulators

- Genetics is the study of inherited traits and genes. (simplistic view)
- Genomics is the study of how a set of genes behave. (complex view)

**Council of Europe Recommendation**
October 2017

**State of New York**
Jan 2017

**Canadian Genetic Non-discrimination Act**
May 2017

**England CMO Annual Report**
July 2017
Scientific Background
Genetics 101

DNA

Base pairs

- Adenine
- Thymine
- Cytosine
- Guanine
- Phosphate backbone

SNP

Chromosome

Gene
Genome Wide Association Studies (‘GWASes’)  

- A GWAS compares SNPs across thousands of people with and without a particular disease / phenotype.

- By tallying which SNPs are common to those people who have the disease, researchers can determine which sections of the genome are associated with the disease and how much increased risk is associated with each SNP.

- The P-value for significance is corrected for multiple testing issues (sometimes millions of SNPs are tested). The conventional threshold is \( P < 5 \times 10^{-8} \).
Disease prediction using GWAS results

- GWASes have been highly successful at identifying genetic variants associated with disease.

- The first GWAS, conducted in 2005, compared 96 patients with age-related macular degeneration with 50 healthy controls. It identified two SNPs with significantly altered allele frequency between the two groups.

- Since the first landmark GWASes, sample sizes have increased (some in the range of 200,000 individuals!). This means SNPs with smaller odds ratios and lower frequency can be identified.

The National Human Genome Research Institute (NHGRI) Catalog of Published GWAS provides a publicly available manually curated collection of published GWAS assaying over 38,000 SNP-trait associations from more than 2,800 publications as of May 2017.
Prevalence vs. Penetrancc
Genetic Risk to Disease and Polygenic Risk Scores (PRS)
Polygenic Risk Scores (PRS)

- A central point of debate on GWASes is that most SNPs are associated with only a small increased risk of the disease, and have only a small predictive value (especially when compared to classical risk factors such as family history or cholesterol).

- The finding that multiple DNA variants are associated with common disorders is leading to disorders being thought of in quantitative terms.

- As multiple DNA variants are identified, they can be aggregated into composites that represent the polygenic liability that underlies common disorders.

- Polygenic risk scores (PRS) capture much more information by looking at a much larger number of variants genome wide (not just the highly significant SNPs).
Calculating PRS

- PRS are based on the selection of SNPs which, individually, do not achieve significance in large-scale GWAS.

- The score is typically calculated by adding the number of risk alleles (0, 1, or 2) carried by each individual weighted by the effect size ($\beta$) of the SNP-trait association:

  $$PRS = \beta_1 \cdot snp_1 + \beta_2 \cdot snp_2 + \cdots \beta_n \cdot snpn$$

- Since even large GWASes achieve only marginal evidence for association for many causal variants, PRS are usually calculated for a set of P-value thresholds (e.g., $P = 1 \times 10^{-5}, 1 \times 10^{-4}, \ldots, 0.05, 0.1, \ldots, 0.5$).
PRS has been shown to increase predictive power of existing risk scores for Coronary Heart Disease

- This study tested the clinical utility of a PRS for coronary heart disease (CHD), in terms of lifetime CHD risk and relative to traditional clinical risk.

- PRS tested in independent cohorts (combined n = 16,802 with 1,344 incident CHD events) and contrasted with the Framingham Risk Score (FRS).

- The HR for CHD from the PRS was 1.74 and 1.28 for the FRS. Further, the PRS was largely unchanged by adjustment for known risk factors, including family history.

- Integration of the PRS with the FRS significantly improved 10 year risk prediction.
## Sample of PRS in literature (1)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Genetic Variants</th>
<th>Difference in Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary Artery Disease</td>
<td>60</td>
<td>2x (top to bottom 20%)</td>
</tr>
<tr>
<td></td>
<td>(explain 50% of heritability)</td>
<td></td>
</tr>
<tr>
<td>Coronary Heart Disease</td>
<td>49,310</td>
<td>1.8 to 4.5x (top to bottom 20%; depending on cohort tested in)</td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
<td>1000</td>
<td>3.5x (top to bottom 20%; after adjustment for standard risk factors)</td>
</tr>
<tr>
<td>Ischemic Stroke</td>
<td>10</td>
<td>1.2x to 2x (top to bottom 20%)</td>
</tr>
<tr>
<td>Breast Cancer</td>
<td>77 (from 1 PRS)</td>
<td>3x (top to bottom 20%)</td>
</tr>
<tr>
<td>Breast Cancer (in women of East Asian ancestry)</td>
<td>44 (from 1 PRS)</td>
<td>2.9x (top to bottom 20%) – impressive given majority of SNPs associated with breast cancer risk have been conducted with European descendants</td>
</tr>
<tr>
<td>Prostate Cancer</td>
<td>77 (from 1 PRS)</td>
<td>4x (top to bottom 20%)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>38</td>
<td>4.6x (top to bottom 25%)</td>
</tr>
</tbody>
</table>
## Sample of PRS in literature (2)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Genetic Variants</th>
<th>Difference in Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sporadic early-onset Alzheimer’s disease</td>
<td>21 (not including APOE alleles)</td>
<td>2.27 [6.44 when including APOE alleles] (top to bottom tertiles)</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>31 (not including APOE alleles)</td>
<td>3.34 (top to bottom deciles; in normal APOE [ε3/3] individuals)</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>356,033</td>
<td>AUC = 78.2% (logistic regression model with APOE, the polygenic score, sex and age as predictors)</td>
</tr>
<tr>
<td>IBD</td>
<td>2,986</td>
<td>5.69 for Crohn’s disease and 3.35 for Ulcerative Colities [top to bottom deciles]</td>
</tr>
<tr>
<td>Colorectal cancer (in Japanese men)</td>
<td>6</td>
<td>Including PSR significantly improved c-stat for classification from 0.6 to 0.66</td>
</tr>
<tr>
<td>Alcohol problems</td>
<td>1,115,557</td>
<td>Higher polygenic scores predicted a greater number of alcohol problems (range of Pearson partial correlations 0.07–0.08, all p-values ≤ 0.01).</td>
</tr>
<tr>
<td>Migraine</td>
<td>21</td>
<td>Odds ratio equal to 1.6x (case vs. control; 2x for migraine without aura)</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>16</td>
<td>12.3x (top to bottom 25%)</td>
</tr>
<tr>
<td>Cardiovascular mortality in patients with CAD</td>
<td>32</td>
<td>Hazard ratio of 1.5 (top to bottom 50%), after adjustment for classical risk factors</td>
</tr>
<tr>
<td>Recurrent cardiovascular events in patients with CAD</td>
<td>45</td>
<td>Hazard ratio of 1.5 (top to bottom 50%)</td>
</tr>
<tr>
<td>Venous thromboembolism</td>
<td>16</td>
<td>1.5x (top to bottom tertile)</td>
</tr>
<tr>
<td>Melanoma risk</td>
<td>15</td>
<td>2.6x (top to bottom quintile)</td>
</tr>
</tbody>
</table>
How could PRS be adopted into Clinical Medicine

- Screening, Individuals with the highest 1% or 5% of PRS values, might be offered
  - regular screening,
  - encouraged to participate in lifestyle modifications, or
  - prescribed therapeutic interventions

- E.g. in the UK, mammogram screening is initiated at age 47, based on a 10-year risk of breast cancer in the average woman.
  - Women in the top 5% of PRS risk reach this level of risk at age 37
  - Those with the lowest 20% of PRS will never reach it

Source: Prospects for using risk scores in polygenic medicine. Forthcoming. Cathryn M. Lewis, Evangelos Vassos
How do PRS interact with lifestyle?

- A genetic predisposition to coronary artery disease is not deterministic but attenuated by a favorable lifestyle.
PRS demonstrate correlations between lifestyle factors and health outcomes

- Individuals with more education tend to live longer and genetic variants have been discovered that predict educational attainment.

Source: Genetic variants linked to education predict longevity. Marioni et al. PNAS November 22, 2016 vol. 113 no. 47 13366-13371
Genetics and Risks of Anti-selection
Considered 13 genetic conditions with known impact on mortality

Concluded mortality experience in the long-run would increase by:
- 35% for Males
- 60% for Females

January 16 paper considered 6 conditions impacting Critical Illness – showed lower impact
Canadian Institute of Actuaries Report, July 2014

Assumptions

Genetic Risk Assumptions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence</th>
<th>Penetrance</th>
<th>Rating</th>
<th>Predicted</th>
<th>Tested</th>
<th>Male</th>
<th>Standard</th>
<th>Grading</th>
</tr>
</thead>
<tbody>
<tr>
<td>BRCA1 or 2</td>
<td>500</td>
<td>25%</td>
<td>200%</td>
<td>50%</td>
<td>30</td>
<td>0%</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>HTCM</td>
<td>500</td>
<td>69%</td>
<td>0.01</td>
<td>50%</td>
<td>25</td>
<td>50%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>DCM</td>
<td>2700</td>
<td>75%</td>
<td>0.04</td>
<td>25%</td>
<td>35</td>
<td>50%</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>ARVCM</td>
<td>1250</td>
<td>75%</td>
<td>0.023</td>
<td>25%</td>
<td>25</td>
<td>50%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Long QT</td>
<td>3000</td>
<td>50%</td>
<td>0.001</td>
<td>25%</td>
<td>20</td>
<td>50%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Brugada</td>
<td>2000</td>
<td>75%</td>
<td>0.015</td>
<td>25%</td>
<td>30</td>
<td>50%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Huntington</td>
<td>20000</td>
<td>90%</td>
<td>1000%</td>
<td>50%</td>
<td>25</td>
<td>50%</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>PKD</td>
<td>10000</td>
<td>100%</td>
<td>50%</td>
<td>75%</td>
<td>30</td>
<td>50%</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>DM1 or 2</td>
<td>8000</td>
<td>75%</td>
<td>500%</td>
<td>50%</td>
<td>25</td>
<td>50%</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>ADO</td>
<td>2427</td>
<td>100%</td>
<td>1000%</td>
<td>50%</td>
<td>30</td>
<td>50%</td>
<td>15</td>
<td>10</td>
</tr>
<tr>
<td>HNPCC</td>
<td>500</td>
<td>50%</td>
<td>300%</td>
<td>50%</td>
<td>30</td>
<td>50%</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Marfan</td>
<td>5000</td>
<td>50%</td>
<td>500%</td>
<td>50%</td>
<td>20</td>
<td>50%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CPVT</td>
<td>10000</td>
<td>75%</td>
<td>1000%</td>
<td>25%</td>
<td>20</td>
<td>50%</td>
<td>0</td>
<td>5</td>
</tr>
</tbody>
</table>

Insurance Assumptions

- Testing Rate: 1/30 p.a.
- Seeking insurance: 75%
- Declined (due to other conditions): 5%
- Face amount: $900,000
- Lapse: 0.5% or 3% p.a.
- Conversion: 50%-100%
- Policy modelled: Convertible Term to 65

Policies Purchased = Population * Prevalence * Tested % * Not declined * (1 – Predicted)

Source: Genetic Testing Model: If Underwriters Had No Access to Known Results. Robert Howard. Canadian Institute of Actuaries, July 2014
Thinking about Life Insurance through a genetic lens, May 2017

- Discussed the concept of polygenic risk scores
- Considered Trauma (Serious Illness) Insurance
- Allowed for purchasing behavior ahead of genetic testing
- Model considered 3 conditions
- Only presented as “illustrative”
- Impact of 1.8% on claims costs
  - Does not appear to consider larger insured pool to offset
- Noted many the current research findings are based on studies of Europeans

Source: Thinking about life insurance through a genetic lens. Dr Damjan Vukcevic & Jessica Chen. May 2017
Assumptions

**Genetic Risk Assumptions**

- **Proportion tested**: 0.5%
- **Increase in risk**: 11%

**Insurance assumptions**

- **Insured already**: 8%
- **Low Risk Policy Lapses**: 20% (+5% to base)
- **Purchasing insurance prior to test**: Everyone
- **Keep insurance post test**: Only high risk
- **Face amount (implicit)**: Average

Source: Thinking about life insurance through a genetic lens. Dr Damjan Vukcevic & Jessica Chen. May 2017
Predicting impact of PRS is still early

- Genetic loci associated with disease will continue to be found and could confer additional predictive power
- Correlations with other health and lifestyle factors could be more significant than high penetrance genes
- Correlations between PRS for different conditions
- Risk of developing a disease may be correlated with severity of disease
- Preventative or mitigating actions, such as:
  - Screening programs based on PRS may limit mortality impact
  - Impact of preventative lifestyle actions unknown
  - Pharmcogenomics etc
- Application of PRS to non caucasian populations
Potential for CAD PRS Anti-selection – Input Data

- Based on Khera, NEJM, 2016
- 50 SNP PRS for CAD
  - Inter quintile range between 1.75 – 1.98
- 4 Lifestyle factors
  - Smoking
  - Healthy BMI
  - Physical Activity once a week
  - Healthy Diet
- End points
  - MI, Revascularization, Death from CHD

Source: Genetic Risk, Adherence to a Healthy Lifestyle, and Coronary Disease. Khera et al, November 2016, DOI: 10.1056/NEJMoA1605086
## Potential for CAD PRS Anti-selection – Simple Modelling

<table>
<thead>
<tr>
<th>Environmental risk</th>
<th>Assumed Proportion in Standard Insured Population</th>
<th>Relative Risk across group</th>
</tr>
</thead>
<tbody>
<tr>
<td>High (Score = 0-1)</td>
<td>0%</td>
<td>n/a</td>
</tr>
<tr>
<td>Intermediate (Score =2)</td>
<td>70%</td>
<td>1.6</td>
</tr>
<tr>
<td>Low (Score ≥ 3)</td>
<td>30%</td>
<td>1.38</td>
</tr>
<tr>
<td>Low genetic risk (lowest 20%)</td>
<td>100%</td>
<td>1.54</td>
</tr>
<tr>
<td>Intermediate genetic risk (mid 60%)</td>
<td>1.64 (+6.5%)</td>
<td></td>
</tr>
<tr>
<td>High genetic risk (highest 20%)</td>
<td>1.64 (+6.5%)</td>
<td></td>
</tr>
</tbody>
</table>

Twice as likely to buy

---

Relative Risk Data Source: Genetic Risk, Adherence to a Healthy Lifestyle, and Coronary Disease. Khera et al, November 2016, DOI: 10.1056/NEJMoa1605086
Conclusions
Conclusions

- Huge ongoing interest in Genomic and Genetics
- Insurance industry benefits society and in a non-compulsory market needs to limit information asymmetry to remain viable
- Wide spread adoption of polygenic risk scores would increase anti-selection risk over consideration of high penetrance genes only, if insurers were not able to assess the same genetic information
- The commensurate increase in premiums might be in the range 3%-10% based on very simple modelling and accepting the large degree of uncertainty in how PRS will emerge into clinical usage
- Additional research is needed to understand both the science and the interaction with insurance buying behavior
Reducing Risk of Anti-selective Behavior in Underwriting
Smoker Predictor Models

October 2017
Integrated Analytics

Munich RE
Agenda

• Opportunities and risks
• Smoker attributes
• Smoker predictor models
• Implementation
  - Process
  - Data
  - Performance
  - Mortality impact
• Conclusions
Opportunities and risks of accelerated underwriting

**Opportunities**

- **Better customer experience**
  - Less intrusive – no fluid testing
  - Less hassle – no test scheduling
  - Faster

- **Better producer experience**
  - Less administration
  - Faster

- **Insurer benefits**
  - Redesigned apps and reflexive questions – better risk assessment
  - Higher sales, cross-sells
  - Lower underwriting costs
  - Access to new/underinsured markets

**Risks**

- **Higher loss costs**
  - Elimination of data may result in less accurate risk assessment
  - Elimination of fluids may miss some health conditions
  - Misrepresentation / anti-selection

- Age
- Gender
- Income
- County

- Smoking prevalence varies significantly by county
- Counties in Utah and other Western states have the lowest rate of smoking
- Counties in the South have the highest rate of smoking as well as those with locations where tobacco can be purchased tax-free.
Smoker predictor models

**Models**
- Custom
- Third party
- Smokers vs smoker-liars

**Data**
- Application
- Geospatial
- Lifestyle & behavioral
- Social media

**Compliance**
- FCRA
- Route to FUW
Implementation: process

- Insurance History
- Prescriptions
- Driving Record
- Credit
- Electronic health records
- Lifestyle / Social
- Wearables

Predictive models:
1) screen
2) risk selection
3) smoker

Rules-based Automated UW

Manual UW

Risk Class

traditional data:
- Medical Lab Results
- Attending Physician Statement
- Income & financial info
## Implementation: data

### Demographics
- Age / Date of Birth
- Gender
- Marital Status
- Face Amount
- Occupation
- Income
- Education

### App & Tele-interview Questions
- Self-reported BMI
- Personal Health History, e.g. Cancer, Heart Disease?
- Drug and Alcohol Use
- Mental Health, e.g. Anxiety, Depression, or Bi-Polar Disorder?
- Family Health History
- Tobacco Usage
- Insurable Interest

### Lifestyle
- Aviation
- Risky Sports
- Survey
- Tobacco Tax
- Purchases
- Vacations
- Home Value
- Household Size

### Geospatial
- Population density
- Unemployment Rate
- Tobacco Tax
- Household Composition

### Third Party
- Rx
- MIB
- MVR

Factors listed here are illustrative, not exhaustive.
Implementation: performance

<table>
<thead>
<tr>
<th>Area</th>
<th>Predictor</th>
<th>Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affluence</td>
<td>Face amount</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Home value</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Client issue age - older</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Investment assets</td>
<td>▼</td>
</tr>
<tr>
<td>Lifestyle</td>
<td>Catalog stationery buyer</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Healthy behavior change index</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>Survey xxxxx music</td>
<td>▲</td>
</tr>
<tr>
<td></td>
<td>Survey xxxxx vacations</td>
<td>▲</td>
</tr>
<tr>
<td></td>
<td>Survey lotteries or sweepstakes</td>
<td>▲</td>
</tr>
<tr>
<td></td>
<td>Survey xxxxx diet</td>
<td>▼</td>
</tr>
<tr>
<td></td>
<td>xxxxx pet ownership</td>
<td>▲</td>
</tr>
<tr>
<td>Location</td>
<td>Counties, grouped</td>
<td>▼</td>
</tr>
</tbody>
</table>

Smoker Rate by Decile

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Implementation: smoker-liars vs. all smokers

Two-Model Approach:

Model Smoker-Liar (SL): *Of self-declared Nonsmokers (NS), predict likelihood of being a smoker liar*

- Based on all information in the app, incl. self-reported smoking history and medical drill down questions.
- Standard approach.
- Assume likelihood of being a smoker for self-reported smokers is 100%.
- Doesn’t take into account future changes in self-reported NS rates, which will change the AUW environment.

Model Smoker-All (SA): *Of all applicants, predict likelihood of being a smoker*

- Ignores all self-reported smoking questions (current smoker, previous tobacco use).
- Heavily relies on public data sources such as US Census.
Implementation: mortality impact

Currently all applicants are sent for fluid tests; extra mortality is 0%
- When fluids are eliminated without routing likely smokers for tests, mortality will increase
- At current self-disclosure, Model SL minimizes extra mortality cost (slightly)
- As non-disclosure increases, Model SA minimizes extra mortality cost

*Calculated as smoker liar rate * 200% (mortality multiplier for smokers)

<table>
<thead>
<tr>
<th>Fluid Test</th>
<th>Non-disclosure</th>
<th>Extra Mortality*</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>25%</td>
<td>0%</td>
</tr>
<tr>
<td>None</td>
<td>25%</td>
<td>12%</td>
</tr>
<tr>
<td>None</td>
<td>50%</td>
<td>21%</td>
</tr>
<tr>
<td>None</td>
<td>100%</td>
<td>40%</td>
</tr>
</tbody>
</table>

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1. If 80% of business is written without fluid testing
   • 8% extra mortality under current self-disclosure
   • 12% extra mortality under 50% no self-disclosure
   • 23% extra mortality under 100% no self-disclosure

2. Employ two tools: Smoker All and Smoker Liar models
   • Currently, Model SL minimizes extra mortality cost
   • Once current self-disclosure rates start to increase, Model SA should be used
   • Assuming 50% scenario, targeted testing using models reduces incremental mortality from 21% to 12%.

3. Use Random hold-out
   • Test random 10% of applicants
   • Track self-disclosure rates to select SA or SL Model
Conclusions

• Start simple
• Add data over time
• Integrate with other tools
  ▪ Facial analysis
  ▪ Wearables
• Measure smoker prediction value with customized CBA
Reducing Risk of Anti-selective Behavior in Underwriting

Smoker Predictor Models

Thank you!

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