Towards healthcare sustainability

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Markers of unsustainability
Societal vs Individual Risk of Death in the United Kingdom

Societal Risk is represented as the risk of death per million total population. Individual risk is represented as the risk of death per million exposed to that hazard. Bubble size represents the relative risk to an individual. By way of example, the bubbles representing deaths due to preventable medical injuries in hospitals and military personnel in Iraq/Afghanistan are a similar size because the risk of death to a patient in a UK hospital is similar to that for a soldier deployed to a war zone. Medical injury poses a greater risk to society simply because vastly more citizens are exposed to that risk and hence die. Note: Log scales.

Individual Risk: Fatalities per 1 million people exposed to risk (Log scale)

Sources: Variety of UK Government and NGO databases, reports, officials and expert advisers.
2012 © Juderon Associates, juderon@gmail.com
Commissioned by Alliance for Natural Health International (www.anhinternational.org)
Funding by Neal’s Yard Remedies (www.nealsyardremedies.com)
Conclusions: In this large French cohort of healthy adults, adherence to healthy lifestyle factors was associated with a lower incidence of hypertension. In terms of public health, active promotion of healthy lifestyle factors at population level is key to combating the hypertension epidemic.
CONCLUSION The combined intervention of paleolithic diet with exercise elicits favorable metabolic and cardiovascular profiles in those that characterize with MetS. The type 2 diabetes epidemic and associated risk factors may be improved from an evolutionary approach in diet and exercise as a preventable and reversible treatment.
• Background
• Experience
• Belief system
• Science
Environment  Economic  Social

Sustainable health & care system
A scenario for one disease (e.g. T2D)

- Tackle diseases before they emerge
- Mechanisms of disease causation and prevention
- Select approaches/interventions/policies that will be most effective for disease prevention in relevant sub-populations
- Determine which elements of the program can be prioritised given prevailing social, economic, environmental and political factors

Options

1
6
4 x 5
20
Total 2,400
How do we reduce uncertainty over how best to create a sustainable system for managing and optimising human health?
1. Re-evaluate ‘the system’
2. Re-evaluate ‘health’ and ‘care’
3. Re-evaluate the concept of ‘patient’
4. Re-evaluate the lens through which we view human/environmental interactions
5. Re-evaluate multi-layered system of disease causation and perpetuation

- One intervention can have multiple health effects
- Multiple interventions can work synergistically on the same effect
Need to build knowledge base to understand this systems model

Courtesy: van Ommen, B: TNO, Netherlands.
6. Re-evaluate the scientific methods we use to select ‘effectiveness’ and healthcare decision-making

Brian Brown, Paul Crawford, Carolyn Hicks
Open University Press, 2003
7. Re-evaluate our priorities and desired outcomes
8. Re-evaluate our society’s respect for human dignity, freedom and democracy
1. Re-evaluate ‘the system’
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Human metabolism: 135 + metabolic pathways, 7439 reactions, 2626 metabolites

http://vmh.uni.lu/#mapnavigator
Simplified systemic view of the models to be developed within MISSION-T2D and the interdependencies between them

Visualization of the white adipose tissue health reference network with disease-associated gene sets

Chronic diseases linked to xenobiotic exposure:
Comparative Toxicogenomics Database [CTD] [www.ctdbase.org]

Connect. Compare.

CTD is a robust, publicly available database that aims to advance understanding about how environmental exposures affect human health. More...

Discover.

1. What human diseases are associated with a gene/protein? (Example)
2. What human diseases are associated with a chemical? (Example)
3. What genes/proteins interact with a chemical? (Example)
4. What chemicals interact with a gene/protein? (Example)
5. What references report a chemical–gene/protein interaction? (Example)
6. What cellular functions (GO terms) are affected by a chemical? (Example)

News

- May, 2017
  Enhanced exposure science search features! Exposure Details Exposure Studies
- June 8, 2017
  New data available!
- All changes...

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Email address Subscribe
<table>
<thead>
<tr>
<th>CTD chemical category</th>
<th>Top interacting genes</th>
<th>Examples of strongly inferred chemical/human chronic disease relationships [no. genes associated]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amino acids, peptides, and proteins</td>
<td>CASP3, TNF, GSTP1, IL6, CXCL8, IL1B, MAPK3, ABCB1, MAPK1, HMOX1</td>
<td>Glutathione/prostatic neoplasms [74 genes]</td>
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<tr>
<td></td>
<td></td>
<td>Bleomycin/pulmonary fibrosis [35 genes]</td>
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<td></td>
<td></td>
<td>Cyclosporine/obesity [96]</td>
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<tr>
<td>Biological factors</td>
<td>TNF, IL6, IL1B, NOS2, PTGS2, IFNG, HMOX1, RELA, CXCL8, MAPK3</td>
<td>Lipopolysaccharides/inflammation [79 genes]</td>
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<td></td>
<td></td>
<td>Mycotoxins/inflammation [15 genes]</td>
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<td></td>
<td></td>
<td>Aflatoxins/liver neoplasms [2 genes]</td>
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<tr>
<td>Carbohydrates</td>
<td>TNF, NOS2, IL1B, IL6, PTGS2, INS, RELA, IFNG, CASP3, NFKBIA</td>
<td>Lipopolysaccharides/liver cirrhosis [117 genes]</td>
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<td></td>
<td></td>
<td>Fructose/diabetes mellitus [46 genes]</td>
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<td></td>
<td></td>
<td>Glucose/carcinoma [59 genes]</td>
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<tr>
<td>Chemical actions and uses</td>
<td>MGEA5, CYP19A1, TNF, IL1B, AR, CASP3, IL6, MAPK1, ACHE, ESR1</td>
<td>Estrogens/carcinoma (hepatocellular) [36 genes]</td>
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<td></td>
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<td>Air pollutants/breast neoplasms [58 genes]</td>
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<td></td>
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<td>Water pollutant chemicals/breast neoplasms [51 genes]</td>
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<tr>
<td>Complex mixtures</td>
<td>TNF, IL6, CXCL8, IL1B, NFE2L2, PTGS2, CYP1A1, HMOX1, NOS2, CAT</td>
<td>Tobacco smoke pollution/stomach neoplasms [102 genes]</td>
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<td></td>
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<td>Smoke/breast neoplasms [101 genes]</td>
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<td></td>
<td></td>
<td>Particulate matter [lung neoplasms] [79 genes]</td>
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<td></td>
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<td>Chinese herbal drugs/carcinoma (hepatocellular) [55 genes]</td>
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<td></td>
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<td>Vehicle emissions/breast neoplasms [250 genes]</td>
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<td>Petroleum/prostatic neoplasms [26 genes]</td>
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<td>Particulate matter/autoimmune diseases [18 genes]</td>
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<tr>
<td>Enzymes and coenzymes</td>
<td>POR, SLC5A6, AKR1B8, CAT, PPARA, CASP3, GAPDH, CYP3A4, NQO1, NQO2</td>
<td>NAD/obesity [8 genes]</td>
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<tr>
<td></td>
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<td>Thiocotic acid/hypertension [41 genes]</td>
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<td>Leucovorin/heart diseases [2 genes]</td>
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6. Re-evaluate the scientific methods we use to select ‘effectiveness’ and healthcare decision-making

E.g.
• Case controls from SMAs vs conventional consultations
• Comparative effectiveness Research
• Big data
We should not dismiss that there are other considerations to contemplate in today’s “modern medicine,” including the atavistic-sounding patient-centric approach and the use of comparative effectiveness research to guide therapy. The founders of evidence-based medicine, as physicians, comprehended this from the start and appreciated that an evidence foundation is just one component of an evidence-based, patient-centered, decision-making process. There was no intent to eradicate the art of medicine, but to amplify it.

7. Re-evaluate our priorities and desired outcomes
8. Re-evaluate society’s respect for human dignity, freedom, and democracy
Thank you.