Re: Liability (Oxford) Ltd

FALSE POSITIONS: DOUBTFUL DATA, DUBIOUS SCIENCE

8th Sept 2021 10.00-11.00

www.reliabilityoxford.co.uk

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Lark

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Lark Ascending

I've had enough of this COVID lark!

That Biodiversity lark…

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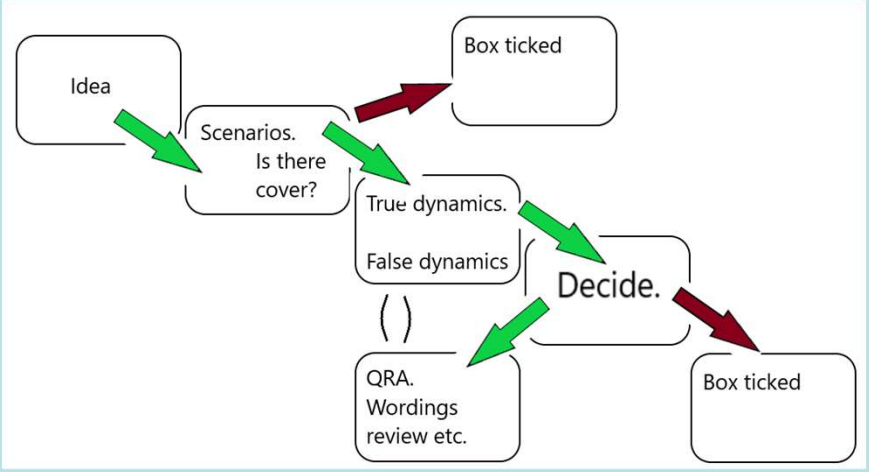
In emerging risks, all positions are valid.



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Surely we want Biodiversity!

Environment Act, long tail liability for biodiversity offsets.

- Ecosystem services, genetic futures, doing the right thing, conservationism, votes…
- But do we also accept natural selection?

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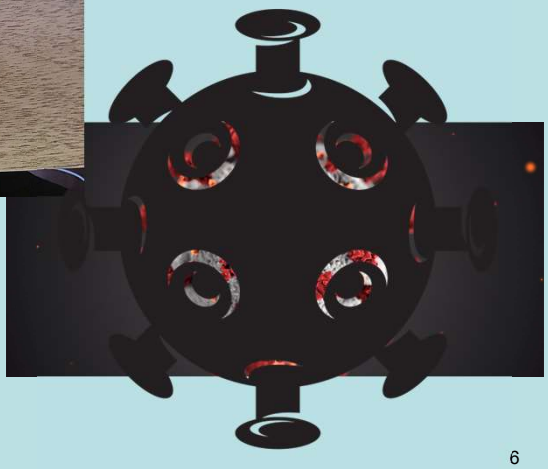
Biodiversity



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Presentation plan

1. Some thoughts on COVID testing.
2. The wrong baseline.
3. The wrong understanding.

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The accurate PCR test

- Billed as 99.7% accurate.
- Test accuracy is defined as the % of TP plus the % of TN.

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Testing…

- For people with symptoms the PCR test has a sensitivity of 95% and a specificity of 99.75%. For those without symptoms these values are unknown.
- Sens = TP/(TP + FN)
- Spec = TN/(TN + FP)
- So the false positive rate is 1 - spec
- Could anyone argue with that?

- For you as an individual you need to know how likely a positive test is to be correct. i.e. TP/(TP + FP)

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Testing

- If the prevalence is 0.1% then the rate of potential false positives is 99.9%.
- So even with a specificity of 99.75% the FP rate is (1-spec) times 99.9% i.e. 0.25%
- TP = sens times 0.1% i.e. 0.095%.
- **TP/(TP + FP) = 27%.** Not even close to the balance of probabilities.

In May 2021 the probability was **3%**.
But 99.75% accurate.

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Epidemiology

RR = Rate of disease in those exposed to hazard
Rate of disease in the control group

Exposed rate > control rate, if the hazard is harmful RR > 1.0. And ER < CR, if the hazard is protective e.g. a vaccine RR < 1.0.

The probable proportion of false positives in the Zen vaccine trial was different between vaccinated (higher proportion of FP) and unvaccinated (lower proportion).

Efficacy was reported as 62% when it was probably 78%.

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Epidemiology

- Pre selection greatly improves the real accuracy of a test.
  - In the COVID example 3% becomes up to 71% using LF test 1st.
- So, study subjects need to be selected at random in both exposed and unexposed groups.
- For campaigners and inexperienced researchers pre selection is an easy route to false claims and sensational findings.

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Epidemiology

- **When the rate of disease is low (<1%) you MUST correct for false positive rate-differential in caseness.**
- **For example**
  - Hypothyroidism in women ranges from 0.2% to 5.3% and in men from 0.02% to 0.5%. Varies with obesity.
  - Testicular cancer in older men 0.005%.
- The lower the rate, the higher the proportion of false positives. OR values for harmful hazards are reduced.

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The wrong baseline 1.0

DF Mackay *et al. NEJM* (2019) DOI: 10.1056/NEJMoa1908483
Neurodegenerative Disease Mortality among Former Professional Soccer Players

Recent accidents and dementia. This myth has been resolved.

What about accelerated dementia spanning decades?

Need to correct for diabetes, vascular health factors, survival bias, drug use, access to health care, diet, sex, recorded brain injury (>15 years), childhood stress, childhood SES and EDUCATION.

Random selection is OK but not a guarantee if any such factor is rare.

You should still measure the known risk factors.

Why? Because if they vary by age then any age-related effect could be a false one.

Dementia is age-related.

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Wrong baseline

- 7,670 FFs and 23,000 controls (male) age-matched, and matched by socio-economic status (based on post code at time of death).
- Death certificates (bias effects not assessed). What did they die of and with?
- Average age of death 68 for both. 15% had died.
- Neurodegenerative disease (NDD) as the primary recorded cause of death was more common among former footballers 1.7%, than controls 0.5%.
- RR was higher for several aetiologically very different NDD types.

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But

- Odds ratios for mortality in dementia studies are very often > 3.0 until corrected for childhood SES / educational attainment / childhood stress.
  - There are countless examples https://jech.bmj.com/content/72/Suppl_1/A47.1.abstract
  - There was no data on these childhood factors.
  - No data on other known risk factors, except CVD.
  - No data on detection bias.
  - No explanation as to how diseases with very different chemistry should all be at elevated levels.
- Cognitive reserve theory maybe?

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The wrong baseline 2.0

- Is the national average the right comparator (baseline)?
- For diseases with a risk of > ~10% the chance of a locally (small sample) significant blip is small.
  - You can compare the interest area with any non-interest area.
- At < 1% statistical variation can be high.
  - You have to measure the actual risk in a large set of similarly bounded areas and generate a probability distribution.

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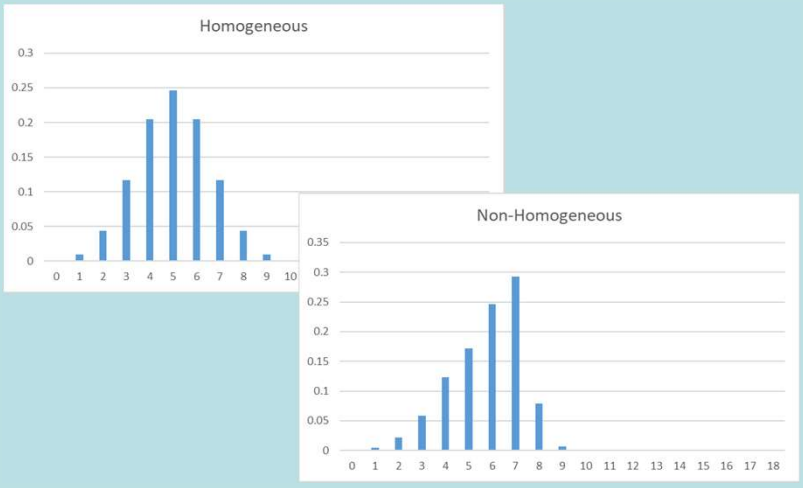
Where causally relevant factors are not homogeneous



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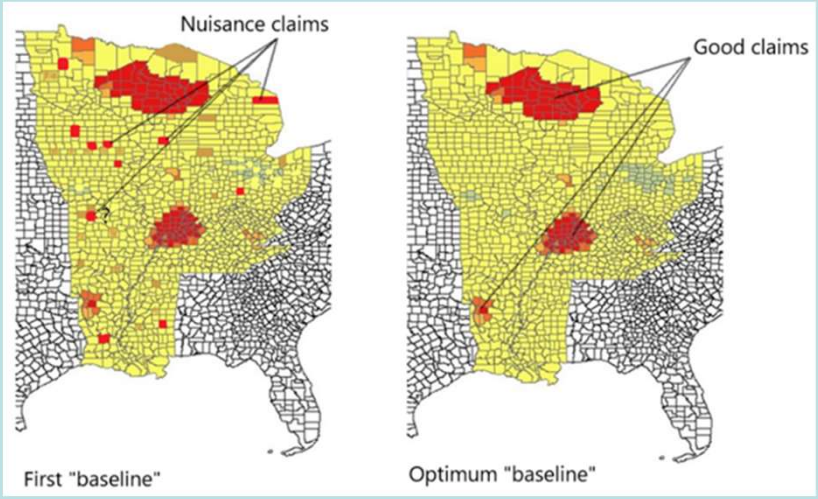
Clusters



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Clusters

- Homogeneity is simply assumed.
- Out of 1,000 'obvious' clusters reported to public health you might see 5 that are not just explained as sampling effects.
- Credible clusters:
  - A disease with a distinct aetiology. A specific cancer.
  - Strong specific overlap between agent map and population map.
    - So the agent and the disease are not generally found
  - The outcome is rare in the observed age group. E.g. new leukaemia in the 20-24 age band.
  - Correspondence between exposure history and latency.

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Model mania

- Sometimes a model drives the understanding and yet is false.
  - Biodiversity is good.
  - A single virus can kill you.
  - There is no safe exposure to a carcinogen.
  - Mesothelioma must be caused by asbestos.
  - Addiction can't be helped.
    - Caffeine is addictive

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Vietnam

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1775687/pdf/amjph00813-0048.pdf

- US servicemen. 20% were heroin addicts.
- 90% of these simply chose to stop being addicts on return to home soil. 98% had withdrawal symptoms. Lasting < 11 days in 89% of cases.
- 9% became re-addicted 43% made recreational use.
- NICE now recommend contingency management (CM)
- https://www.nice.org.uk/guidance/cg51/chapter/appendix-c-contingency-management-key-elements-in-the-delivery-of-a-programme
- The brain disease model of addiction is false. It promotes a 'helpless victim' model.

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BDMA

- See screen

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BDMA explains

- How humans are adapted to form habits.
- How drugs provide rewards. Euphoria. Dopamine.
- How drug taking becomes a habit if repeated soon enough.
- How physiological dependency develops/accumulates but is reversible.
- Bias in conscious actions.
- a human being is an urge-driven slave to sensations and gratification

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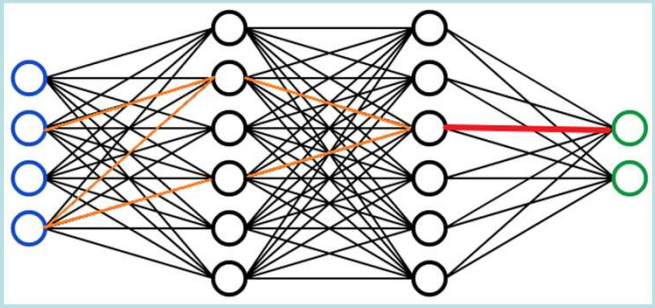
In the mind



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BDMA fails

- To explain how personality persists despite changes in connectivity.
- Free will to stop.
- The ability to reason.
- “addicts” never lose the capacity to make conscious decisions and control their actions.
- normal conscious states have causal efficacy for legal purposes
- The urge to take, is just an opportunity to decide.
- What foreseeably happens after that, is freely chosen.

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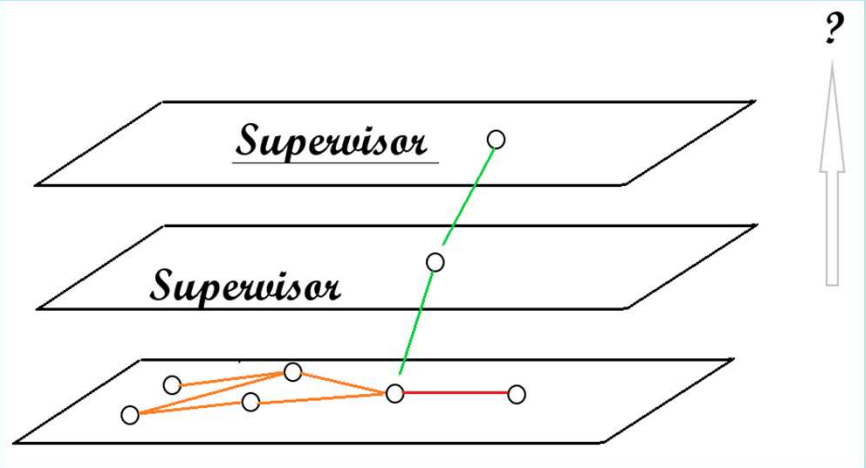
In the mind



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Generative stability

- The personality is not lost provided the topmost layer of supervision is maintained. [there is no pleasure in losing that] But how is the coherence of the ultimate supervisor maintained?
- Integrativity is maintained by supervision, redundancy and degeneracy.
  - Redundancy – several pathways do the same thing
  - Degeneracy – a pathway can be imitated (the act of a supervisory layer)

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Caffeine

- A small effect on an anti dopamine chemical (adenosine) leads to a small increase in dopamine. A slightly euphoric effect building up over some minutes.
- An increase in adenosine production and sensitivity.
- Habituation occurs. Need more caffeine to get the same effect.
- No systems become disabled in the absence of caffeine. There is no dependency.
- Overdose would require an extraordinary effort.
- ‘Cold turkey’ could last two days.

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So what?

- Text mining science articles will be misleading. Useful as a test of reporting fashions and social bias but not as a test of legal causation or emerging risk.
- If you have a concern about any emerging hazard or causation theory, have the evidence assessed for scientific rigour (model, hypothesis, methods, assumptions, validated tools, interpretation…). AI cannot do this.
- Beware of self-evident truths…someone is relying on you to convince yourself. Why do the work for them?
- Decide.

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