Is the EAT-Lancet commission report alone enough for dietary recommendations?

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Today focus on following aspects of the derivation of the report’s Universal Healthy Reference Diet (Reference diet or REF) health projections (Section 1)

- **Evidence basis**
  - Methodology lacks transparency: e.g. dietary selection process not well documented
  - Not a systematic approach, not following accepted guidelines. Based on expert opinion.
  - No evidence quality assessment
  - Reference diet established based on prevented mortality, afterward adjusted for nutritional content and evaluated *a posteriori* for sustainability

- **Modeling methodology**
  - Causality assumption
  - Wrong RRs
  - Incorrect and incomplete uncertainty analysis
  - Comparing REF diet with fixed, perfect intake against status-quo diet caloric intake

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The EAT–Lancet Commission's Dietary Composition May Not Prevent Noncommunicable Disease Mortality

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ABSTRACT

The recently published EAT–Lancet Commission report on dietary impacts on noncommunicable disease (NCD) burden has been criticized for methodological limitations. Aims: To critically evaluate the dietary recommendations of the EAT–Lancet Commission report. Methods: The EAT–Lancet Commission report was critically evaluated for its dietary recommendations. Results: The EAT–Lancet Commission report concluded that dietary composition should be changed to prevent NCD burden. However, the evidence base to support this recommendation is weak. Conclusion: The EAT–Lancet Commission report’s dietary recommendations for preventing NCD burden are not strongly supported by the evidence base. [https://academic.oup.com/jn/article/150/5/985/5736577]
Proposed reference diet based only on “health”, measured through prevented mortality -> not an optimization

Reference diet established based on prevented mortality, afterward adjusted for nutritional content and evaluated a posteriori for sustainability

Prevented mortality estimates come from three publications with overlapping co-authors

BUT, significantly departs from other established, health-based diets (e.g. US Dietary Guidelines and UK Eatwell guide)

WHY?
Evidence selection

No systematic review approach used, despite Lancet publication requirements

- **PRISMA** for systematic reviews and meta-analyses
- **GATHER** for studies involving global health estimates

Systematic approach also advocated by NAS in 2017 “Redesigning the Process for Establishing the Dietary Guidelines for Americans, Chapter 4: Strengthening Analyses and Advancing Methods Used”

Lack of systematic approach can result in:

- **biased evidence, particularly for food groups with conflictive literature (e.g. animal proteins)**
- Ignore other equally optimal diets outside of those evaluated by authors

Examples:

- Some existing SRs were cited, but selection process undefined e.g.
  - why use Chan et al (2011) vs five other newer meta-analysis on CRC and red meat?
  - why include protective effect of nuts against T2DM but not dairy and CRC?
  - Poultry 29(0-58) gr/day. But studies cited in report, even higher amounts not associated with negative health outcomes AND protective effect against CRC and cardiovascular disease

- No article inclusion/exclusion criteria presented, nor database of articles screened
Strength of evidence and inconsistencies

- No evaluation of quality of evidence e.g. GRADE e.g. Johnston et al.(2019) and red meats
- Diet risk factors for health outcomes RRs small and from observational studies assume causal relationship, and are independent from each other

Discrepancies on total red meat RRs:
- Total red meat RR for stroke Chen et al.19 was 1.15 (1.05-1.25), but Springmann et al used 1.1 (1.05-1.15)
- Total red meat RR for T2DM Feskens et al.18 was 1.13 (95% CI 1.03-1.23) but Springmann et al. used 1.15 (1.07-1.24),
- REF diet uses RR for total red meat, should be only unprocessed (as diet excludes processed red meat) -> T2DM should then be excluded, lower for CRC and stroke
Non-Med diets (DASH and Japanese studies)

Mediterranean diets

REF diet vs references cited

Grains
Potato
Legume
Veg
Fruit
Total Red Meat
Fish
Eggs
Dairy
Edible fats

0 100 200 300 400 500 600 700 800 900 1000 g/day
- Wide range of consumption with no effect
- Amount does not correspond to any one source, including Med diet
The Population Impact Fraction (PIF) general approach used for many other disease-risk factor relationships outside of diet
- Requires causality assumption
- Model additive only when risk factors are independent (diet risk factors are not)

Linear dose response for all health impacts of red meat and legume consumption, but non-linear for protective effects of other dietary components
- Based on observational studies at highest consumption, but extrapolated down when no effect observed

Choose the Risk Factors:
1) Red Meat
2) Fruits
3) Vegetables
4) Nuts and Seeds
5) Legumes
6) Fish
7) Underweight
8) Overweight
9) Obesity

Identify the Health Impacts:
1) Coronary Heart Disease
2) Type 2 Diabetes Mellitus
3) Stroke
4) Colorectal Cancer
5) Total Cancer
6) Other

Quantify the relationship:
Select relative risk (RR) for each Risk Factor/Health Impact combination (either matched to the doses or from a dose-response)

Calculate the Impact Fraction:
How much of the total health impact is from the risk factor?

Combine impact fractions:
What is the total percent effect of the diet on the Health impact?

Calculate the prevented deaths/illnesses:
What is the net effect (in numbers of people or years of life) of all of the risk factors based on diet?

Per Country:
Quantify the exposure:
How much does the population eat, and how many of them don’t eat any?

Sum of all country results
Total worldwide prevented deaths/illnesses
Potential Corrections to the analysis

Used total stroke and red meat RR when red meat is only significantly associated with ischemic stroke

No uncertainty in:
- amount of food consumed
- prevalence of consumers
- mortality rates
- prevalence underweight, overweight, and obesity

Incorrect RRs red meats

Ref diet contains no processed meat, but the total red meat RR was used (should be only unprocessed)

Assumes disease cases prevented = deaths prevented

Inconsistent meta-analysis sources for RRs

Per Country:

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Total worldwide prevented deaths/illnesses

Sum of all country results
Reduction of **weight-related risk factors alone** impacts mortality

Diet maintaining recommended calorie intake (i.e. perfect adherence resulting in no underweight, and no overweight/obese) will prevent mortality

We separated the weight-related risk factors from the overall mortality estimation (weight + dietary components) to answer:

**Is there additional reduction in mortalities from the change in dietary components alone after we adjust for weight-related deaths?**

Allows us to know if changing from status-quo to the reference diet prevents deaths, or mortality reduction could be achieved with any diet that keeps everyone at a healthy weight

Roughly 50% prevention due to calorie control

From Springmann et al, 2018
Avoided mortality after adjustments – United States

The graph shows the distribution of avoided mortalities in the United States, with different categories such as corrected avoided mortality, original reported avoided mortality, and weight only.

Avoided mortalities (in thousands)

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REF diet calorie intake assumed fixed -> underweight, overweight, and obesity eliminated in the US population. This assumption is responsible for **75% (63-94%)** of the predicted total mortalities avoided in the USA.

“After adjusting for the omitted uncertainty and RR errors, and beyond the impact of changing energy consumption to a fixed and ideal level with the REF diet, there may not be statistically significant changes in deaths from switching from the current diet composition to the REF one.”
Conclusions

The EAT-Lancet Universal Healthy Reference Diet is NOT:

- A health-optimal diet
- Systematically derived, or using standard reporting guidelines
- Based on causal evidence
- Consistent with several other guidelines or studies

But even accepting all of the above, reference diet mortality prevention NOT statistically different from status-quo diet, after adjusting for ideal caloric intake adherence (for the US)

So although the principle has merits, the methodology must be independently replicated, tested and potentially improved by others before it’s accepted as sound evidence for policy decisions.
Thank you!

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