Rory Robertson (+61 414 703 471)
January 2019

Expression of Concern regarding misrepresented mouse-diet results in high-profile University of Sydney paper

Dear Editorial Board of Cell Metabolism journal (and independent observers, including journalists),
https://www.cell.com/cell-metabolism/editorial-board

Happy New Year. I am writing to express concern about the misrepresentation of longevity results in a high-profile study in your journal. The main longevity findings sit obscured and undiscussed in a “Supplemental” table: Table S2 shows that the median lifespan of mice on none of 25 diets exceeded 140 weeks, let alone 150 weeks. Yet Figure 2 in the main text (chart below) suggests median lifespans beyond 150 weeks; Figure 2B shows a Kaplan-Meier curve featuring the oldest mice (outliers >150 weeks) while obscuring the range of median lifespans (all <140 weeks) over the 30-diet experiment.

Critically, the authors claim falsely that “Median lifespan was greatest” on diets “low in protein and high in carbohydrate”. You can see (Table S2) that median lifespan was greatest on a diet high in protein (42%) and low in carbohydrate (29%): 139 weeks is 10% better than the next-best median, also from a high-protein diet. Alas, in Figure 2A the authors carefully suppressed any possible sign of the two best diets (median lifespan 126-139 weeks). The public is being deceived. Why?

Table S2, related to Figure 2. Survival analysis by dietary composition.

<table>
<thead>
<tr>
<th>Energy Density</th>
<th>Protein (%)</th>
<th>Carb (%)</th>
<th>Fat (%)</th>
<th>Protein:Carb ratio</th>
<th>Median Lifespan (weeks)</th>
<th>Maximum Lifespan (weeks)</th>
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https://www.cell.com/cmss/10.1016/j.cmet.2014.02.009/attachment/e2d00ae0-845a-4f9e-99a4-a831d55dd569/mmc1.pdf

Figure 2 in main text: p. 5 of 13 at https://www.cell.com/action/showPdf?pii=S1550-4131%2814%2900065-5

Norman and Streiner in PDQ Statistics (3’rd Edition, 2003) explain that authors have a responsibility to convey to readers “an accurate impression” of what the full dataset looks like “before beginning the statistical shenanigans. Any paper that doesn’t do this should be viewed from the outset with considerable suspicion” (p.12). Our troubled paper is shown on p.3.

Table 3 (on p.6, below) confirms that the authors have skilfully misrepresented their 30-diet longevity results, including by obscuring 100+ dead mice on five low-protein diets. In my opinion, their faulty paper should be retracted, then rewritten under competent and honest supervision to ensure the 30 diets’ median lifespans are reliably presented and discussed.
Prof uses 1000 mice to expose food folly

THE key to good health is a balance between protein, carbohydrates and fat, says an expert on obesity, diabetes and cardiovascular disease.

Clifford Fram, AAP National Medical Writer

BELIEF that single nutrients such as omega-3s, sugar or salt can cure or cause all ills is folly, says a leading health scientist.

The key, Professor Stephen Simpson says, is for people to think about food as food and to seek a healthy balance between protein, carbohydrates and fat.

Too much of one for too long can make you fat and unhealthy, or even thin and unhealthy, says Prof Simpson, academic director of the new $500 million Charles Perkins centre set up at the University of Sydney to fight obesity, diabetes and cardiovascular disease.

"The balance really matters," he told colleagues at an Australian Society for Medical Research conference in Victoria.

His team conducted a study in which 1000 mice were fed 30 different diets with different ratios of protein, carbohydrates and fat.

"If you want to lose weight as a mouse, you go onto a high-protein diet. But if you stay on that too long you will have poor circulating insulin and glucose tolerance.

"If you go too low on protein, you will drive over-consumption and be prone to obesity."

A good balance for a mouse is about 20 per cent protein, about 60 per cent carbohydrates and about 20 per cent fat.

"And mice are not that different from humans," he said.

An interesting finding was that a low-protein diet coupled with high carbohydrates led to obesity. But these mice lived longest and had a healthy balance in their gut.

Prof Simpson said he was concerned about the emphasis on micronutrients such as vitamins, sugar and salt.

"It is unhelpful when people argue everything is the fault of sugar or fat or salt or whatever when what we are dealing with is a balancing problem."

The best type of carbohydrates and fat is limited amounts of sugar and complex, low GI, hard-to-digest foods.

Prof Simpson said healthy fats such as omega-3 were also important.

Originally published as Prof uses 1000 mice to expose food folly
This unreliable paper is one of the highest-profile papers ever written in Australia. The University of Sydney promotes it in full-page advertisements in weekend newspapers (p.4). The authors’ deceptive false claims from the mouse paper quickly became diet advice for humans (p.5) and were used to justify public funding of mouse-diet research into dementia (p.10).

It’s thus worth taking the time to understand exactly what has been done. For starters, around 1,000 C57BL/6 (standard laboratory) mice were put on 30 diets, consisting of various parts protein, fat and carbohydrate, each with three energy levels. Along the way, five killer 5%-protein diets (and 100+ dead mice) were buried in the Supplemental material (below).
Epic fail in University of Sydney's quality control: False mouse-diet claims promoted as “research excellence”
Table 3 overleaf collates information from Supplemental materials to reliably present the median lifespans from the 30-diet experiment. Is this story for mice - "The healthiest diets were the ones that had the lowest protein, 5 to 10 to 15 per cent protein, the highest amount of carbohydrate, so 60, 70, 75 per cent..." and less than 20% fat - robust or bogus?

**Low-carb diet may make you unhealthy, shorten your life: study**

By Sarah Dingle

Updated 5 Mar 2014, 4:54pm

**Eating a high-protein, low-carb diet could actually make you unhealthy and more likely to die younger, a landmark Australian study has found.**

The three-year study by the University of Sydney's Charles Perkins Centre found that while high-protein diets might make you slimmer and feel more attractive, the best diet for longevity is one low in protein and high in carbohydrates.

Professor of geriatric medicine David Le Couteur from Sydney's Anzac Research Institute was part of the team which modified the diets of 900 mice with dramatic results.

"If you're interested in a longer life span and late-life health, then a diet that is low in protein, high in carbohydrate and low in fat is preferable," he said.

"You can eat as much of that as you like.

"You don't have to be hungry, you don't have to reduce your calorie intake, you can just let your body decide what the right amount of food is."

The team put mice on 25 different diets, altering the proportions of protein, carbohydrates and fat.

The mice were allowed to eat as much food as they wanted to more closely replicate the food choices humans make.

"The healthiest diets were the ones that had the lowest protein, 5 to 10 to 15 per cent protein, the highest amount of carbohydrate, so 60, 70, 75 per cent carbohydrate, and a reasonably low fat content, so less than 20 per cent," Professor Le Couteur said.

Charles Perkins put ~1,000 mice on 30 diets then misrepresented median-lifespan story re 858 mice on 25 diets

Table 3 below presents the published lifespan results from the 30-diet experiment, facts retrieved from Supplemental information, including Table S2 and details on the five killer low-protein diets (100+ dead mice) quietly abandoned. The authors use their high-profile mouse paper to claim important longevity benefits from low-protein, high-carbohydrate diets:

- “Median lifespan was greatest for animals whose intakes were low in protein and high in carbohydrate...” (p.421)
- “Median lifespan increased from about 95 to 125 weeks (approximately 30%; Table S2) as the protein-to-carbohydrate ratio decreased” (p.421) (Table S2 is reproduced on p.1 earlier; its results are reproduced below.)
- “If you’re interested in a longer lifespan...then a diet that is low in protein, high in carbohydrate...is preferable” &
- “The healthiest diets were the ones that had the lowest protein, 5 to 10 to 15 per cent protein...” (previous page)

Table 3 shows that those claims are false. Indeed, they blatantly misrepresent the paper’s actual longevity results:

- Median lifespan was greatest - by far - for animals on a diet high in protein (42%) and low in carbohydrate (29%);
- That diet’s median mouse lived for 139 weeks, 10% longer than the median mouse on the second-best diet;
- Four of the eight diets on which the median mouse lived for at least 120 weeks are high-protein diets;
- Only eight of the 18 diets on which the median mouse lived for 100 weeks or more are low in protein; while
- Seven of the 12 diets on which mice struggled - the median was dead before 100 weeks - are low in protein.

In summary, Table 3 falsifies the authors’ specific claims above. Again, median lifespan was not greatest on low-protein, high-carbohydrate diets: that oldest median mouse lived on that chunky 1.45 protein-to-carbs ratio for a big 139 weeks.

High-profile false claims mislead. People are dying (pp.8-10). Shouldn’t the paper be formally retracted, then rewritten under competent and honest supervision to ensure the 30 diets’ median lifespans are reliably presented and discussed?

<table>
<thead>
<tr>
<th>Table 3</th>
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<tr>
<td><strong>Mouse diets ranked by median longevity (weeks) of mice on 30 diets</strong></td>
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<tr>
<td>DIET</td>
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<td><strong>RANKING</strong></td>
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*Diets of mice euthanised because they “failed to thrive” are included in analysis above

Source: The paper’s “Supplemental” information, including Table S2 (reproduced on p. 1, earlier).
Bad animal model: C57BL/6 mice profoundly unlike humans with respect to metabolism of carbohydrate and fat

The Charles Perkins Centre’s mouse-diet studies use C57BL/6 mice. That’s fine, as their use is pretty standard in mouse studies in laboratories across the western world: [https://en.wikipedia.org/wiki/C57BL/6](https://en.wikipedia.org/wiki/C57BL/6)

Importantly, when you buy these C57BL/6 mice for laboratory use, you are told that “fed a high-fat [low-carbohydrate] diet”, they “develop obesity, mild to moderate hyperglycemia, and hyperinsulinemia”: [https://www.jax.org/strain/000664](https://www.jax.org/strain/000664)

While it’s widely known that standard lab mice get fat and sick on low-carb diets, Professor Stephen Simpson – Academic Director of the palatial Charles Perkins Centre at the University of Sydney – saw mere confirmation of that as important:

[https://www.abc.net.au/radionational/programs/healthreport/high-protein2c-low-carbohydrate-diet/5309616#transcript](https://www.abc.net.au/radionational/programs/healthreport/high-protein2c-low-carbohydrate-diet/5309616#transcript)

But that was not an important finding, unless all 18 researchers failed to read the instructions on their new box of lab mice. More important is the readily available 2012 paper (below) that explains to science careerists new to working with mice that the C57BL/6 mouse is a bad animal model for humans when the critical issues for discussion include obesity, type 2 diabetes, cardiovascular disease (CVD) and longevity. Again, these lab mice are problematic when the issues for investigation include diet and health, insulin resistance (aka Metabolic Syndrome) and longevity in humans. That’s because the metabolic responses of standard lab mice and humans are profoundly different; in particular, C57BL/6 mice put on low-carbohydrate diets typically become fat and sick - via insulin resistance - whereas humans tend to thrive.

Professor Simpson and his co-authors should have known that mouse and human responses to low-carbohydrate (high-fat) diets tend to be profoundly different; they should be aware that sugary low-protein, high-carb mouse diets tend to harm humans. Tragically, many Australians are dying early via type 2 diabetes and CVD as a result of eating exactly the sort of sugary low-protein, high-carb mouse diets promoted by the Charles Perkins Centre as excellent for human longevity. Compare and contrast the sugary mouse diet on pages 2 and 3 with the sugary diet harming humans, overleaf.

The following pages tell a tragic story of Group of Eight university science gone wrong.
The mobs Charlie Perkins cared about struggle and die early in droves on low-protein, 60%-carb mouse diet

Abstract

Objective: To describe the nutritional quality of community-level diets in remote northern Australian communities.

Design, setting and participants: A multisite 12-month assessment (July 2010 to June 2011) of community-level diet in three remote Aboriginal communities in the Northern Territory, linking data from food outlets and food services to the Australian Food and Nutrient Database.

Main outcome measures: Contribution of food groups to total food expenditure; macronutrient contribution to energy and nutrient density relative to requirements; and food sources of key nutrients.

Results: One-quarter (24.8%; SD, 1.4%) of total food expenditure was on non-alcoholic beverages; 15.6% (SD, 1.2%) was on sugar-sweetened drinks. 2.2% (SD, 0.2%) was spent on fruit and 5.4% (SD, 0.4%) on vegetables. Sugars contributed 25.7%–34.3% of dietary energy, 71% of which was table sugar and sugar-sweetened beverages. Dietary protein contributed 12.5%–14.1% of energy, lower than the recommended 15%–25% optimum. Furthermore, white bread was a major source of energy and most nutrients in all three communities. Mean: 61% carbs, including 24% refined sugar.

Conclusion: Very poor dietary quality continues to be a characteristic of remote Aboriginal community nutrition profiles since the earliest studies almost three decades ago. Significant proportions of key nutrients are provided from poor-quality nutrient-fortified processed foods. Further evidence regarding the impact of the cost of food on food purchasing in this context is urgently needed and should include cost–benefit analysis of improved dietary intake on health outcomes.

Dietary improvement for Indigenous Australians is a priority strategy for reducing the health gap between Indigenous and non-Indigenous Australians. Poor-quality diet among the indigenous population is a significant risk factor for three of the major causes of premature death — cardiovascular disease, cancer and type 2 diabetes. The 26% of Indigenous Australians living in remote areas experience 40% of the health gap of Indigenous Australians overall. Much of this burden of disease is due to extremely poor nutrition throughout life.

2 Estimated energy availability and macronutrient profile, overall and by community

<table>
<thead>
<tr>
<th>Macronutrient distribution as a proportion of dietary energy (% [SD])</th>
<th>Community A</th>
<th>Community B</th>
<th>Community C</th>
<th>All communities</th>
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<tr>
<td>Protein</td>
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<td>Fat</td>
<td>24.5 (0.6)</td>
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<td>Sugars</td>
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<td>28.5 (2.2)</td>
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Real-world evidence: Humans on low-protein, 60%-carb mouse diets are dying early via Type 2 diabetes & CVD

Aboriginal and Torres Strait Islander adults experience diabetes 20 years earlier than non-Indigenous adults

Aboriginal and Torres Strait Islander adults are more than three times as likely as non-Indigenous adults to have diabetes, and they experience it at much younger ages, according to new figures released by the Australian Bureau of Statistics today.

"Results from the largest ever biomedical collection for Aboriginal and Torres Strait Islander adults, which collected information on a wide range of chronic diseases and nutrition, reveal that diabetes is a major concern," said Dr Paul Jeffs from the ABS.

"The voluntary blood test results showed that in 2012–13, one in ten Aboriginal and Torres Strait Islander adults had diabetes. This means that, when age differences are taken into account, Aboriginal and Torres Strait Islander adults were more than three times as likely as non-Indigenous adults to have diabetes."

"What was even more striking was how much earlier in life Aboriginal and Torres Strait Islander adults experience diabetes. In fact, the equivalent rates of diabetes in the Aboriginal and Torres Strait Islander population were often not reached until 20 years later in the non-Indigenous population." said Dr Jeffs.

The survey revealed that diabetes was twice as common among Aboriginal and Torres Strait Islander adults living in remote areas. Around one in five in remote areas had diabetes compared with around one in ten in non-remote areas.

Also of interest was the fact that many Aboriginal and Torres Strait Islander adults with diabetes also had signs of other chronic conditions.

"More than half of all Aboriginal and Torres Strait Islander adults with diabetes also had signs of kidney disease. This compared with a third of non-Indigenous adults with diabetes", said Dr Jeffs.

"Given these findings, it is not surprising that the death rate for diabetes among Aboriginal and Torres Strait Islander people is seven times higher than for non-Indigenous people."

Other results released today suggest that many Aboriginal and Torres Strait Islander adults may not be aware they have high cholesterol, with one in four having high cholesterol levels, yet only one in ten being aware they had it.

Further information is available in Australian Aboriginal and Torres Strait Islander Health

http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/4727.0.55.003~2012-13~Media%20Release~Aboriginal%20and%20Torres%20Strait%20Islander%20adults%20experience%20diabetes%200%20years%20earlier%20than%20non-Indigenous%20adults%20(Media%20Release)~130
Charles Perkins Centre’s mouse-diet “science” expanded into Dementia in 2018, with 2014 longevity results still misrepresented and fact that human and C57BL/6 mouse metabolisms are profoundly different still ignored


Making utter nonsense of the Charles Perkins Centre’s bogus high-carbohydrate mouse-diet advice for human longevity, competent scientists, doctors and dietitians in the US are using a well-known low-carb, high-fat diet to reverse (cure) type 2 diabetes in ~60% of human patients, while overseeing dramatic reductions in weight and use of costly ineffective drugs.

Endpiece: Academic competence, scientific integrity, mouse diets, human misery, dementia and early death

The authors’ misrepresentation of their 30-diet experiment’s mouse-longevity findings might have been inadvertent. If so, they would choose to immediately retract their blatantly false claim that median-mouse lifespan was greatest on low-protein, high-carbohydrate diets. Again, that claim is clearly falsified by the study’s actual results, reproduced in Table 3 on p.6.

If the paper’s mouse-diet misrepresentation was deliberate – perhaps to promote its influential authors’ preferred “protein leverage” hypothesis - we can expect the authors to fight against the truth by choosing not to retract the paper. I note that stephen.simpson@sydney.edu.au is one of two corresponding authors. Further, I note that Professor Stephen Simpson’s recent record of insisting on competence, scientific integrity and the retraction of blatantly false claims is poor. Alas, he is a key supporter of the University of Sydney’s infamous Australian Paradox sugar-and-obesity fraud that seeks to falsely exonerate modern doses of added sugar as a key driver of obesity and type 2 diabetes.

As the Academic Director of the University of Sydney’s palatial Charles Perkins Centre, Professor Simpson has been actively assisting his friend Professor Jennie Brand-Miller to dishonestly pretend that flat, dead-ending and obviously made-up/fake/unreliable/invalid data are valid and reliable, even “robust and meaningful”, as she falsely insists that valid sugar indicators that clearly trend up not down - over the relevant 1980 to 2010 timeframe – in fact show “a consistent and substantial decline”: p.6 https://www.australianparadox.com/pdf/USyd-Misconduct-in-ANU-PhD.pdf; and p.78 http://www.australianparadox.com/pdf/Big-5-year-update-Feb-2017.pdf

Tasked with reducing the big societal problems of obesity, type 2 diabetes, CVD and related maladies, Steve Simpson’s fledgling Charles Perkins Centre has quickly become a shopfront for unreliable studies promoting sugary diets high in carbohydrate as healthy choices. It even promotes its own range of sugary Low-GI healthfoods, as assessed by Brand-Miller’s faulty Glycemic Index methodology: 99.4% sugar https://www.foodpolitics.com/2016/03/sugar-in-australia-its-better-for-you/; 46% sugars https://www.gisymbol.com/product/nestle-milo/; and 50% sugars https://www.gisymbol.com/product/sustagen-hospital-formula-active-chocolate/

Beyond being home to the Australian Paradox fraud and the University of Sydney’s shonky but influential Glycemic Index approach to nutrition, the Charles Perkins Centre’s controversial mouse-diet “science” is a growing threat to public health. Its reckless promotion of low-protein, high-carbohydrate mouse diets to maximise human longevity – and limit dementia - is a problem for vulnerable consumers, especially type 2 diabetics. It’s a tragic irony that Indigenous Australians are dying early in droves on exactly the sort of sugary low-protein, high-carb mouse diets advised by Charles Perkins (pp.8-10).

Despite its clear lack of scientific competence and integrity on diet-and-health matters, the Charles Perkins Centre is becoming influential in national discussions of dementia. That’s a problem because its mouse-diet science clearly has little useful to add; indeed, so far it has been worse than useless. While we don’t know all that much about the growing modern malady of dementia (including Alzheimer’s disease), there are several key facts that will need to be embraced:

- **Dementia is often referred to as “type 3 diabetes”, because it’s notably correlated with type 2 diabetes**
- For ~100 years, it’s been convincingly documented that excessive consumption of sugar and other carbohydrate is the main cause of (type 2) diabetes: p.422 https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf
- Removing excess intake of carbohydrate fixes/cures type 2 diabetes in ~60% of cases (prev. page & p.433 above)
- Low-carb diets work to minimise the risk of heart disease and “What’s good for the heart is good for the brain” https://blog.virtahealth.com/improving-cardiovascular-disease-risk-factors-virta-treatment/
- Obesity, type 2 diabetes, cardiovascular disease (CVD), obesity-related cancers, dementia... All connected?
- Dementia in humans appears to be another malady boosted by insulin resistance, a.k.a. Metabolic Syndrome

“Metabolic Syndrome” - now affecting more than one-third of all adults across the western world - is perhaps the best guide to eventual early death via type 2 diabetes or CVD. Yet nutrition “scientists” and public-health officials largely ignore it as an issue, running a mile from evidence that simple carbohydrate restriction fixes Metabolic Syndrome better than anything else: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1323303/; http://linkis.com/www.samj.org.za/inde/r9grq

Surely a diet that fixes/cures type 2 diabetes (straightforward carbohydrate restriction) is **likely to be more helpful in limiting dementia** (aka type 3 diabetes) than a sugary low-protein, high-carbohydrate mouse diet, given that excessive consumption of sugar and other carbohydrate causes type 2 diabetes. Here’s some further background on these issues:

- Gary Taubes discussing these issues with health professionals https://www.youtube.com/watch?v=xRp0sJuqkBk
- Dr Sarah Hallberg speaking about how Virta Health is reversing Type 2 diabetes https://blog.virtahealth.com/dr-sarah-hallberg-type-2-diabetes-reversal/
- ABC TV’s Catalyst show on low-carbohydrate, high-fat diets https://www.youtube.com/watch?v=8GUIBNKnT1M

In sum, it’s a tragic irony that Charles Perkins Centre careerists now are recklessly promoting sugary low-protein, high-carb mouse diets – much like those bringing early death to Indigenous and other vulnerable Australians (pp.2-3 and pp.8-9) – as the dietary approach that’s likely to minimise dementia in humans. My goodness, what would Charlie think? (over)
What would Charlie think of what’s being done under his name, if he hadn’t died young, via kidney disease?

[Image of Dr Charles Perkins]

The Charles Perkins Centre: a new model for tackling chronic disease

Stephen J. Simpson

[life summary]

Birth
18 June 1936
Alice Springs, Northern Territory, Australia

Death
18 October 2000
Sydney, New South Wales, Australia

Cause of Death
knight disease

Education
Le Fevre High School (Adelaide)
University of Sydney

Occupation
Indigenous rights activist/supporter
public servant
public service head
soccer player

Awards
Officer of the Order of Australia

Key Events
Freedom Ride, 1965

Key Organisations
Foundation for Aboriginal Affairs
Student Action for Aborigines
National Aborigines Consultative Committee
Aboriginal and Torres Strait Island Commission

APPENDIX: March 2014 interaction – in Cell Metabolism journal - with authors discussing paper’s problems

RR published queries re 100+ dead mice, 5 missing killer diets and median ranking. Journal Cell retracted. Why?

Why were 100+ sick mice - all on low-protein diets - excluded from longevity results?

The original study reportedly involved 30 diets and nearly 1000 mice. [Hyperlink to original study]

Yet the published paper reports the results for - amongst other things - "...longevity in only 868 mice fed one of [only] 25 diets ad libitum".

Readers, I'm trying to understand why five of the 30 original diets - all low-protein diets - were excluded from the final results.

Specifically, "These diets were discontinued due to weight loss (> 20%), rectal prolapse or failure to thrive": Table S1, p. 7 [Hyperlink to supplementary material]

"Failure to thrive?" Readers, imagine the disappointment of those 100+ sick/dying mice - all on low-protein diets - when they were told that, sorry, we're going to euthanize you and then exclude you from this longevity study.

It's a longevity study: sick and dying mice are the main thing we are looking for! Yet they were excluded. Why?

My observation is that the study's high-profile “finding” - that low-protein diets boost longevity in mice - is not robust when the analysis is properly re-balanced - by excluding the five most-unhealthy high-protein diets - to properly adjust the study for the low-profile exclusion those five most-unhealthy low-protein diets.

Moreover, to properly capture the underlying reality of the published results, it makes sense to focus on median - not maximum - lifespans. Checking the medians for the remaining 20 diets, the claimed boost to mouse longevity from low-protein diets has disappeared: the top-2 diets now are high-protein, as are four of the Top-7 diets.

And low-protein diets now represent three of the Bottom-6 diets. (This information is via Table S2 in the link above.)

I'm an economist, so "science" is not my strong suit. But doesn't ditching those five obviously unhealthy low-protein diets - involving 100+ sick/dying mice! - by itself invalidate the paper's claim that low-protein diets boost longevity in mice (and so humans)?

In my opinion, the study's longevity "findings" should be re-written to properly reflect the underlying results from all of those original 30 diets, including the longevity of all those nearly 1000 mice.

As things stand, the public is being misinformed. [Hyperlink to article on public information]

Regards, Rory

[Hyperlink to additional article or discussion]

http://www.cell.com/cell-metabolism/comments/S1550-4131(14)00065-5
Why median-mouse longevity buried in Supplemental Table S2, with ranking scrambled? Are humans like mice?

Submitted March 2014 but not published

Authors,

Thanks for your response, although it added to my concerns rather than reduced them.

I get the bit that those five low-protein diets were discontinued because 100+ young mice were dying, and so had to be euthanized according to the terms of the ethics protocol.

What I don't get is why those sick/dying/dead mice are not counted in your longevity results. Excluding those 100+ died-young low-protein mice from your longevity results and then concluding that low-protein diets boost the longevity of mice seems a rather idiosyncratic "finding".

Indeed, your latest claim that "including the five discontinued diets would make the conclusions even stronger" is nonsense. Clearly, including those 100+ died-young low-protein mice in the longevity results would further discredit your "finding" that low-protein diets boost longevity.

Authors, I note that you chose not to respond to my observation that your ranking of longevity results in terms of outliers - Maximum lifespan - rather than a standard measure of central tendency - Median lifespan - seems designed to ensure than a low-protein diet sits atop the published longevity ranking in Table S2: (p. 8) http://download.cell.com/cell-metabolism/mmc/journals/1550-4131/PIIS1550413114000655.mmc1.pdf

Regardless, if we were a group of mice seeking to maximise our longevity - and we could choose only one diet - I assume that you like me would choose the diet that maximised the median longevity of the group.

In those terms, it turns out that the single-BEST diet was a HIGH-protein (42%), LOW-carb (29%), high-energy diet, NOT a low-protein diet.

Again, your actual results seem somewhat inconsistent with your headline "finding" that low-protein, high-carbohydrate diets maximise longevity, inconsistent with the story high protein diets are 'nearly as bad as smoking': http://www.thetheaustralian.com.au/news/latest-news/protein-diets-nearly-as-bad-as-smoking/story-fn3dxiwe-1226845436762

Another issue here – beyond the veracity of your published results – is the Charles Perkins Centre’s cavalier (reckless?) extrapolation of its mouse “findings” to humans:

"A good balance for a mouse is about 20 per cent protein, about 60 per cent carbohydrates and about 20 per cent fat. 'And mice are not that different from humans,' he [the academic head of the Charles Perkins Centre] said": http://www.heraldsun.com.au/news/breaking-news/prof-uses-1000-mice-to-expose-food-folly/story-fni0xqi4-1226764591760

It is ironic – or worse - that the Charles Perkins Centre is promoting processed carbohydrates as healthy - the mice diets deemed most healthy were dominated by processed grains and sugar - and downplaying the importance of protein, when back in the real world the disadvantaged Australians Charlie Perkins cared most about are dying prematurely on diets that are dominated by unhealthy sugar and processed grains, and are dangerously low in protein? Box 2 https://www.mja.com.au/journal/2013/198/7/characteristics-community-level-diet-aboriginal-people-remote-northern-australia

Regards,
Rory

http://www.cell.com/cell-metabolism/abstract/S1550-4131(14)00065-5#Comments
Here’s me, Emma Alberici and ABC TV’s *Lateline* on the University of Sydney’s Australian Paradox: [http://www.abc.net.au/lateline/content/2015/s4442720.htm](http://www.abc.net.au/lateline/content/2015/s4442720.htm)

Here’s the latest on that epic *Australian Paradox* sugar-and-obesity fraud: [http://www.australianparadox.com/pdf/ABC-investigation-AustralianParadox.pdf](http://www.australianparadox.com/pdf/ABC-investigation-AustralianParadox.pdf)


Want to stop trends in your family and friends towards obesity, type 2 diabetes, heart disease and various cancers? Stop eating and drinking sugar: [http://www.youtube.com/watch?v=xDaYa0AB8TQ&feature=youtu.be](http://www.youtube.com/watch?v=xDaYa0AB8TQ&feature=youtu.be)


Comments, criticisms, questions, compliments, whatever welcome at strathburnstation@gmail.com

**www.strathburn.com**

Strathburn Cattle Station is a proud partner of YALARI, Australia’s leading provider of quality boarding-school educations for Aboriginal and Torres Strait Islander teenagers. Check it out at [http://www.strathburn.com/yalari.php](http://www.strathburn.com/yalari.php)