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), <u>https://www.cell.com/cell-metabolism/editorial-board</u>

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?

> Median and maximum lifespan in weeks (w). Maximum lifespan was determined as the average of the longest lived 10% (n=2-3) of each cohort.

Energy Density	Protein (%)	Carb (%)	Fat (%)	Protein: Carb ratio	Median lifespan (w)	Maximum lifespan (w)
MEDIUM	5	75	20	0.07	121.86	157.43
HIGH	5	20	75	0.25	106.43	154.21
HIGH	5	75	20	0.07	1 <mark>19.43</mark>	151.79
MEDIUM	14	57	29	0.25	123.00	151.57
HIGH	42	29	29	1.45	138.86	151.14
MEDIUM	42	29	29	1.45	122.57	148.00
MEDIUM	14	29	57	0.48	<mark>113.86</mark>	147.36
HIGH	5	48	48	0.10	124.43	146.21
MEDIUM	33	48	20	0.69	1 <mark>22.5</mark> 7	145.71
MEDIUM	23	38	38	0.61	123.86	143.07
HIGH	33	48	20	0.69	<mark>98.2</mark> 9	141.00
HIGH	14	57	29	0.25	<mark>117.43</mark>	140.07
HIGH	33	20	48	1.65	107.14	136.86
LOW	33	48	20	0.69	126.57	134.14
MEDIUM	33	20	48	1.65	106.57	133.79
HIGH	14	29	57	0.48	108.00	133.71
MEDIUM	60	20	20	3.00	108.00	129.50
HIGH	60	20	20	3.00	99.57	127.57
HIGH	23	38	38	0.61	<mark>100.00</mark>	124.57
LOW	14	57	29	0.25	98.57	119.43
LOW	33	20	48	1.65	<mark>78.57</mark>	116.36
LOW	14	29	57	0.48	88.71	115.07
LOW	42	29	29	1.45	<mark>85.85</mark>	104.00
LOW	60	20	20	3.00	84.29	102.86
LOW	23	38	38	0.61	<mark>89.2</mark> 9	100.36





e-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.

<mark>"And mice are not that different from humans,</mark>" he said.

An interesting finding was that a **low-protein die**t coupled with <mark>high carbohydrates</mark> led to obesity. But <mark>these mice lived longest</mark> 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 https://www.news.com.au/national/breaking-news/prof-uses-1000-mice-to-expose-food-folly/newsstory/403238e7cccc57b86b689aaa18fa4b95





The Ratio of Macronutrients, Not Caloric Intake, Dictates Cardiometabolic Health, Aging, and Longevity in Ad Libitum-Fed Mice

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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).

Diet		1	2 ^a	3 ^b	4	5	6 ^a	7	8	9	10
<mark>%P</mark>		60	5	5	33	33	5	14	14	42	23
%C		20	75	20	47	20	48	29	57	29	38
%F		20	20	75	20	47	48	57	29	29	38
1	Р	5.03	0.42	0.42	2.77	2.77	42	1.17	1.17	3.52	1.93
LOW	С	1.67	28	67	4.02	1.67	4.12	2.43	4.77	2.43	3.18
o kj g	F	1.67	1.67	6.2	1.67	4.02	4.02	4.77	2.43	2.43	3.18
Man diama	Ρ	7.54	0.63	1 .63	4.15	4.15	1.63	1.76	1.76	5.28	2.89
Medium	С	2.51	9.41	2 51	6.02	2.51	6.22	3.64	7.15	3.64	4.77
13 KJ G	F	2.51	2.51	9.4	2.51	6.02	6.02	7.15	3.64	3.64	4.77
1.01-	Р	10.06	0.84	0.84	5.53	5.53	0.84	2.35	2.35	7.04	3.86
High 17 k l a ⁻¹	С	3.35	12.55	3.35	8.03	3.35	8.03	4.85	9.54	4.85	6.36
тикју	F	3.35	3.35	12.55	3.35	8.03	8.03	9.54	4.85	4.85	6.36

The % of protein (P), carbohydrate (C) and fat (F) (as a % of total energy). Each diet was replicated at 8 kJ g⁻¹ (low energy), 13 kJ g⁻¹ (medium energy) and 17kJ g⁻¹ (high energy). Diets varied in content of P (casein and methionine), C (sucrose, wheatstarch and dextrinized cornstarch) and F (soya bean oil). All other ingredients were kept similar. Other ingredients include cellulose, a mineral mix (Ca, P, Mg, ll com/cms/10, 1016/i cmet 2014, 02, 009/attachment/e2d00ae0.845a-4f9e-99a4-a831d55dd

pp 7-8 https://www.cell.com/cms/10.1016/j.cmet.2014.02.009/attachment/e2d00ae0-845a-4f9e-99a4-a831d55dd569/mmc1.pdf

Steve Simpson: It was the most complicated study and indeed the most ambitious study ever to look at macronutrition in any animal, particularly any mammal. What we set out to do was to look at the interactive and individual effects of protein, carbohydrate and fat in the diet of mice, and that requires a very large number of dietary treatments. Rather than a typical study which would look at a control diet of standard mouse food and compare it to a high fat diet, what we did was design 25 diets that spanned 10 different ratios of protein to fat to carbohydrate at one of three total energy densities and allowed our mice to feed ad libitum throughout their lives.

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

RESULTS

The data we present derive from 858 mice fed one of 25 diets differing systematically in protein, carbohydrate, and fat content and energy density. By their nature, these data are complex, and https://www.cell.com/action/showPdf?pii=S1550-4131%2814%2900065-5 Epic fail in University of Sydney's quality control: False mouse-diet claims promoted as "research excellence'

We're unlearning diet to help us live longer

By questioning how the body processes different foods, our researchers have discovered that a low protein, high carb diet can delay chronic disease and help us live a longer and healthier life.

Find out how we're unlearning the world's greatest challenges. sydney.edu.au/our-research



THE UNIVERSITY OF SYDNEY

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Table 3 overleaf collates information from Supplemental materials to reliably present the median lifespans from the 30diet 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

AM 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 highprotein 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 latelife 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.



PHOTO: The paleolithic or modern day Stone Age diet is one of the latest crazes. (Flickr: Megan Myers)

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"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.

https://www.abc.net.au/news/2014-03-05/low-carb-diet-may-shorten-your-life-study-finds/5299284

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-tocarbohydrate 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?

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М	ouse diets rar	nked by m	edian lo	ngevity	(weeks) of r	nice on 3	0 diets*		
			Yellow is I	ow-protein (diet				
			Blue is hig	h-protein di	et				
DIET RANKING	Median lifespan of group	Protein (%)	Carb (%)	Fat (%)	Protein: Carb ratio	Energy density	Oldest 2-3 mice (weeks of age)		
1	139	42	29	29	1.45	high	151		
3est diet's median longevity is 139 weeks, ~10% > next best. It is <i>high</i> in protein and <i>low</i> in carbohydra									
2	127	33	48	20	0.69	low	134		
3	124	5	48	48	0.10	high	146		
4	124	23	38	38	0.61	high	143		
5	123	14	57	29	0.25	medium	152		
6	123	42	29	29	1.45	medium	148		
7	123	33	48	20	0.69	medium	146		
8	122	5	75	20	0.07	medium	157		
9	119	5	75	20	0.07	high	152		
10	117	14	57	29	0.25	high	140		
11	114	14	29	57	0.48	medium	147		
12	108	14	29	57	0.48	high	134		
13	108	60	20	20	3.00	medium	130		
14	107	33	20	48	1.65	high	137		
15	107	33	20	48	1.65	medium	134		
16	106	5	20	75	0.25	high	154		
17	100	23	38	38	0.61	high	125		
18	100	60	20	20	3.00	high	128		
19	99	14	57	29	0.25	low	119		
20	98	33	48	20	0.69	medium	141		
21	89	23	38	38	0.61	low	100		
22	89	14	29	57	0.48	low	115		
23	86	42	29	29	1.45	low	104		
24	84	60	20	20	3.00	low	103		
25	79	33	20	48	1.65	low	116		
26*	23	5	75	20	0.07	low	23		
27*	23	5	48	48	0.10	medium	23		
28*	10	5	20	75	0.25	low	10		
29*	10	5	20	75	0.25	medium	10		
30*	10	5	48	48	0.10	low	10		

*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: <u>https://en.wikipedia.org/wiki/C57BL/6</u>

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": <u>https://www.jax.org/strain/000664</u>

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:

Steve Simpson: This was quite interesting. The cause of death in the high protein, low carb fed animals, so far as you can tell...the thing is, when a mouse dies, unless you are there to collect it right at the moment of death, you can't do any particularly useful physiological analysis. But the markers of health—cardio-metabolic health—showed that they were insulin resistant, they had high levels of circulating blood sugars, and they had poor cardiac function. So these mice on the high protein, low carb diet were in bad shape.

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 (highfat) 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

		Istralia			
K Brimblecombe, M Aust 2013; 198 (7): 380-3	egan M Ferguson, 184. doi:10.1	Selma C Liberato and Kerir 5694/mja12.11407	n O'Dea		
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Objective: To communities.	describe the nu	itritional quality of con	nmunity-level diets	in remote northern Australian	
Design, settin level diet in thr food services to	g and particip ee remote Abou the Australian	pants: A multisite 12- riginal communities in Food and Nutrient Da	month assessment the Northern Territ itabase. ~ 26	(July 2010 to June 2011) of community cory, linking data from food outlets and $c O O \rho e ople$	-
Main outcome energy and nut	measures: Contract of the measures of the meas	ontribution of food gro elative to requirements	oups to total food es; and food sources	xpenditure; macronutrient contribution of key nutrients.	to
Results: One-o 1.2%) was on s vegetables. Sug sweetened beve 25% optimum.	uarter (24.8% ugar-sweetene pars contribute erages. Dietary Furthermore, v	; SD, 1.4%) of total fo ed drinks. 2.2% (SD, 0 d 25.7%–34.3% of die protein contributed 1. white bread was a maj	ood expenditure wa 0.2%) was spent on etary energy, 71% 2.5%-14.1% of en or source of energy	s on non-alcoholic beverages; 15.6% (fruit and 5.4% (SD, 0.4%) on of which was <u>table sugar and sugar-</u> ergy, lower than the recommended 15% and most nutrients in all three	5D, 6-
communities.	Mean :	61% carbs	, including	~24% refined sugar!	
Conclusion: Ve profiles since th from poor-quali food purchasing intake on health	ery poor dietary e earliest studi ty nutrient-fort i in this context n outcomes.	y quality continues to l ies almost three decad cified processed foods. t is urgently needed an	be a characteristic les ago. Significant Further evidence r nd should include c	of remote Aboriginal community nutritic proportions of key nutrients are provide egarding the impact of the cost of food ost-benefit analysis of improved dietary	on ed on
ietary improven ndigenous and n actor for three o	nent for Indige Ion-Indigenous f the major cau	nous Australians is a p Australians. ¹ Poor-qu uses of premature dea living in remote areas	priority strategy for ality diet among th th — cardiovascula experience 40% of	reducing the health gap between e Indigenous population is a significant r disease, cancer and type 2 diabetes. ² the health gap of Indigenous Australian	risk The ns

Energy intake	Community A	Community B	Community C	All communities
Macronutrient distribution as a proportion of dietary energy (% [SD])				
Protein	12.5% (0.3)	14.1% (0.8)	13.4% (0.6)	12.7% (0.3)
Fat	24.5% (0.6)	31.6% (1.5)	33.5% (1.1)	25.7% (0.6)
Saturated fat	9.4% (0.3)	11.6% (0.6)	12.1% (0.3)	9.7% (0.3)
Carbohydrate	62.1% (0.8)	53.3% (1.8)	52.1% (1.1)	<mark>60.7%</mark> (0.8)
Sugars	<mark>34.</mark> 3% (0.8)	<mark>28.</mark> 9% (2.2)	<mark>25.</mark> 7% (1.8)	<mark>33.4</mark> % (0.7)

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https://www.mja.com.au/journal/2013/198/7/characteristics-community-level-diet-aboriginal-people-remote-northern-australia

Real-world evidence: Humans on low-protein, 60%-carb mouse diets are dying early via Type 2 diabetes & CVD



<u>13~Media%20Release~Aboriginal%20and%20Torres%20Strait%20Islander%20adults%20experience%20diabetes%202</u> <u>0%20years%20earlier%20than%20non-Indigenous%20adults%20(Media%20Release)~130</u> 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



are being explored. Recently, we utilized the geometric framework (Simpson and Raubenheimer, 2012) to evaluate the effects of ad libitum-fed diets varying in macronutrients and energy content on aging. Mice consuming a low-protein, highcarbohydrate, low-fat diet (LPHC, protein:carbohydrate ~1:10) lived longest and were healthier in old age, even when compared

p. 2 https://www.cell.com/cell-reports/pdf/S2211-1247(18)31674-7.pdf

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.

How does the Virta T	reatmo	nt	orady
compare to Usual Car	reatine ہم؟	iii.	
		Virta	Usual Care
HbA1c		▼ -1.3%	▲ +0.2%
Diabetes Medication Usage Rate (except m	etformin)	▼ -48%	▲ +9%
Body Weight		▼ -30 lbs	— +0 lbs
Triglycerides		▼ -48 mg/dL	≜ +28 mg/dl
HDL-c		🔺 +8 mg/dL	≜ -1 mg/dL
Inflammation (hsCRP)		▼ -39%	▲ +15%
Diabetes Ther. 2018. DOI: 10.1007/s13300-018-0373-9			
Groundbreaking Clinical Outcomes	Ş	60% OF PATH	ENTS REVERSED YPE 2 DIABETES
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https://www.virtahealth.com/research; https://link.springer.com/content/pdf/10.1007%2Fs13300-018-0373-9.pdf

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 will 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 <u>https://www.australianparadox.com/pdf/USyd-Misconduct-in-ANU-PhD.pdf</u>; and p.78 <u>http://www.australianparadox.com/pdf/Big-5-year-update-Feb-2017.pdf</u>

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 <u>https://www.foodpolitics.com/2016/03/sugar-in-australia-its-better-for-you/</u>; 46% sugars <u>https://www.gisymbol.com/product/nestle-milo/</u>; and 50% sugars <u>https://www.gisymbol.com/product/sustagen-hospital-formula-active-chocolate/</u>

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 <u>https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf</u>
- 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" <u>https://blog.virtahealth.com/improving-cardiovascular-disease-risk-factors-virta-treatment/</u>
- 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: <u>https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1323303/</u>; <u>http://linkis.com/www.samj.org.za/inde/r9grg</u>

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 <u>https://blog.virtahealth.com/dr-sarah-hallberg-type-2-diabetes-reversal/</u>
- ABC TV's Catalyst show on low-carbohydrate, high-fat diets <u>https://www.youtube.com/watch?v=8GUIBNKnT1M</u>

In sum, it's a tragic irony that Charles Perkins Centre careerists now are recklessly promoting sugary low-protein, highcarb 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?



The Charles Perkins Centre: a

new model for tackling

chronic disease

Stephen J. Simpson



Charles Perkins, 1974 National Archives of Australia,

Life Summary [details]

Birth 16 June 1936 Alice Springs, Northern Territory, Australia

Death 18 October 2000 Sydney, New South Wales, Australia

Cause of Death kidney disease

Cultural Heritage Indigenous Australian

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



THE UNIVERSITY OF SYDNEY

https://royalsoc.org.au/images/pdf/Forum2016/Simpson.29Nov2016.pdf http://ia.anu.edu.au/biography/perkins-charles-nelson-charlie-810

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?

Rory Robertson (former fattie)	Mar 12, 2014
Why were 100+ sick mice - all on low-protein d	iets - excluded from
longevity results?	
The original study reportedly involved <u>30 diets and</u>	I nearly 1000
mice: http://www.heraldsun.com.au/news/break	king-news/prof-uses-
1000-mice-to-expose-food-folly/story-fni0xqi4-12	226764591760
Yet the published paper reports the results for - an	nongst other things -
"longevity in [only 858] mice fed one of [only]	5 diets ad libitum".
Readers, I'm trying to understand why five of the 3	30 original diets - all
low-protein diets - were excluded from the final res	sults.
Specifically, "These diets were discontinued due to	o weight loss (≥
20%), rectal prolapse or failure to thrive": Table S1	1, p.
7 http://download.cell.com/cell-metabolism/mn	ncs/journals/1550-
4131/PIIS1550413114000655.mmc1.pdf	
"Failure to thrive"! Readers, imagine the disappoin	tment of those 1 <mark>00+</mark>
sick/dying mice - all on low-protein diets - when	they were told that,
sorry, we're going to euthanize you and then exclu	de 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 "find	ding" - that lower-
protein diets boost longevity in mice - is not robu	<mark>st</mark> when the analysis
is properly re-balanced - by excluding the five me	ost-unhealthy high-
protein diets - to properly adjust the study for the l	ow-profile exclusion
those five most-unhealthy low-protein diets.	
Moreover, to properly capture the underlying reality	y of the published
results, it makes sense to focus on median not m	naximum lifespans.
Checking the medians for the remaining 20 diets, t	the claimed boost to
mouse longevity from low-protein diets has disapp	eared: the top-2
diets now are high-protein, as are four of the Te	op-7 diets.
And low-protein diets now represent three of the B	Bottom-6 diets. (This
information is via Table S2 in the link above.)	
I'm an economist, so "science" is not my strong s	uit. But doesn't
ditching those five obviously unhealthy low-protein	i diets - involving
100+ sick/dying mice! - by itself invalidate the pa	per's claim that low-
protein diets boost longevity in mice (and so huma	ans)?
In my opinion, the study's longevity "findings" sho	uld be re-written to
properly reflect the underlying results from all of the	nose original 30 diets,
including the longevity of all those nearly 1000 mid	ce.
As things stand, the public is being	
misinformed: http://www.smh.com.au/lifestyle/d	iet-and-
fitness/highprotein-diet-a-factor-in-shorter-life-2	20140304-3456a.html
Regards,Rory	

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+ diedyoung 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/mmcs/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': <u>http://www.theaustralian.com.au/news/latest-news/protein-diets-nearly-as-bad-as-smoking/story-fn3dxiwe-1226845436762</u>

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": <u>http://www.heraldsun.com.au/news/breaking-news/prof-uses-1000-mice-to-expose-food-folly/story-fni0xqi4-1226764591760</u>

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

rory robertson economist and former-fattie https://twitter.com/OzParadoxdotcom

Here's me, Emma Alberici and ABC TV's *Lateline* on the University of Sydney's Australian Paradox: <u>http://www.abc.net.au/lateline/content/2015/s4442720.htm</u>

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

Here's Vice-Chancellor Spence's threat to ban me from campus: p. 64 <u>http://www.australianparadox.com/pdf/Big-5-year-update-Feb-2017.pdf</u>

During National Diabetes Week 2016, I wrote to the Department of Health about "The scandalous mistreatment of Australians with type 2 diabetes (T2D)": <u>http://www.australianparadox.com/pdf/Expanded-Letter-HealthDept-type2diabetes.pdf</u>

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

Here's the diet advised by Dr Peter Brukner, recently the Australian cricket team's doctor: <u>http://www.peterbrukner.com/wp-content/uploads/2014/08/All-you-need-to-know-about-LCHF1.pdf</u>; <u>http://www.abc.net.au/catalyst/lowcarb/</u>

A life in our times: Vale Alexander "Sandy" Robertson (1933-2015): <u>http://www.australianparadox.com/pdf/AlecRobertson-born2oct33.pdf</u>

www.strathburn.com

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