**Rory Robertson (+61 414 703 471)**
**July 2019**
***Supplementary Submission***
**Crucial new evidence for University of Sydney's Investigation into USyd/Harvard's median-lifespan fraud**

Dear Dr Rebecca Halligan (Director of Research Integrity & Ethics), Vice-Chancellor Michael Spence, Deputy Vice-Chancellor (Research) Duncan Ivison, General Council Richard Fisher and interested observers, including journalists,

I hope you are well. I am sorry you chose not to meet with me on campus in May, June or July. Today, I am writing for two reasons, both regarding your current research-misconduct Investigation into blatantly false mouse diet-and-lifespan "findings" by Charles Perkins Centre science careerists (p. 17). Here is my *Submission* to assist that Investigation: https://www.australianparadox.com/pdf/USyd-Misconduct-June19.pdf

First, I am writing to ensure that my *Submission* is provided to your research-integrity Investigator/s. Rebecca, please ensure that happens, if you have not done so already. The community is unlikely to regard the University's response to my concerns about research fraud on campus as credible, if I am again not interviewed and the Investigation does not properly address my *Submission*'s link between the 2014 paper's misrepresentations and the *Australian Paradox* fraud.

Second, I am writing to provide the current Investigation with **new evidence**, on apparent motivations for the median-lifespan misrepresentations in the 2014 paper and the authors' 2019 denials. There appear to be two such motivations:

**(a)** Two influential co-authors predicted in their **2012** book that their big 30-diet mouse-diet experiment would confirm that lifespan is maximised on low-protein, high-carbohydrate (**LPHC**) diets, beginning the process of "unifying" their decades of work on insects and "protein leverage" with the science relating to mammals, including mice and humans. That is, two of the 18 authors were "highly motivated" to "find" what they *needed* to find, regardless of the experiment's *actual* results.

**(b)** Fresh NHMRC funding worth $13m over 2019-2023 (p. 7), funding at risk if research-integrity problems are conceded.

Rebecca, this mouse-lifespan fraud matters because its LPHC falsehoods quickly became harmful diet and diabetes advice; **in humans, LPHC diets cause type 2 diabetes and early death, especially in indigenous communities** (pp. 8, 24-34). You explained in your 9 May letter (p. 17) that it was the NHMRC alerting you to my concerns about scientific fraud at Charles Perkins that forced the University to begin an inquiry into this matter. Please forward my *Supplementary Submission* and its new evidence to your NHMRC contacts and to your research-misconduct Investigator/s.

**2. Is blatant misrepresentation of median-lifespan results explained by careerists "finding" what they *needed*?**

As discussed in my *Submission*, the faulty 2014 paper reporting on the University of Sydney's 1,000-mouse experiment - overseen by "Principal investigator" Professor Stephen Simpson, also the Academic Director of the Charles Perkins Centre - blatantly misrepresents the 30 diets' median-lifespan results, including by presenting and discussing the results from **only 25 diets and 858 mice**. **Table 3 on p. 11 presents the *actual* results**, facts retrieved from "Supplemental Information", including Table S2 and details about ~150 dead young mice and the **five killer LPHC diets** quietly hidden.

The misrepresentation of median-lifespan results is so blatant that one can only wonder – **why** would eminent science careerists risk being found doing what I have documented has been done? Why would Simpson *et al* be so dopey, desperate and/or dishonest as to blatantly misrepresent the *actual* results of their high-profile experiment? I don't know. But I have searched around and tried to understand. That 2012 book by the two dominant authors of the 2014 paper - a book describing their big plans for the future – appears to be a key factor. After their career-defining experiment's *actual* lifespan results turned out nothing like what the two ambitious careerists predicted in their 2012 book, they seem to have faced a **profound dilemma**: tell the truth and kill the dream of more-exciting careers, or shamelessly fake it to make it!

Please let me explain. Professors Stephen Simpson and David Raubenheimer presented themselves in their ambitious 2012 book - ***The Nature of Nutrition: A unifying framework from animal adaptation to human obesity*** (Princeton University Press) - as keen for their decades of work on "protein leverage" and lifespan in insects to be viewed as highly relevant to human health and lifespan. The book (key extracts are reproduced in coming pages) shows them planning to extend their findings on insects to mammals, starting with mice, then humans. They outlined the purpose and priors of the three-year, 30-diet, 1,000-mouse experiment "still underway", detailing *exactly* what they expected and *needed* to find.

For longevity in insects, Simpson and Raubenheimer observed: "**the ratio of protein to carbohydrate [P:C] is crucial**". **But "What about in mammals?" Well, "There have been numerous reports...that protein restriction...extends life span in rodents", so "...it is at least plausible that the response of mammals – including humans – is similar to that of insects".** Critically, key diet influences on mammals' lifespan **remained to be seen**. Accordingly, "...we have embarked on just such a study in mice with David Le Couteur ...University of Sydney". We're really keen to publish our results, but "At the time of writing [c. 2012], the 30-diet experiment is still underway..." (p. 62, reproduced on p. 4 below).

For Simpson and Raubenheimer's career-expanding ambitions, the 30-diet mouse experiment's basic hypothesis was as follows**: In mice as in insects, "protein restriction ... extends life span" while** "**increasing the ratio of protein to non-protein energy ... decreases life span**...". As far back as 2009, that's what they planned and *needed* to find. Alas, Table 3 on p.11 shows the experiment's *actual* results, falsifying that basic hypothesis. (**Discussion resumes p.11)**

# The Nature of Nutrition

## A Unifying Framework from Animal Adaptation to Human Obesity

Stephen J. Simpson AND David Raubenheimer

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eight for locusts. Omission of only one of these eight amino acids from an otherwise complete supplementary mix rendered a diet "low protein" so far as the animal was concerned. Signaling elevated protein status, whether to induce protein satiety in locusts or to trigger pathways involved in shortening life span in flies, therefore requires a specific mixture of amino acids.

Taken together, the results from insects provide overwhelming evidence that caloric restriction is not responsible for life span extension. Instead, the ratio of protein to carbohydrate in the diet is crucial, with the protein component of the response mediated by a mixture of key amino acids, which includes, but is not exclusively, methionine. An important message from the insect results is that experiments in which single amino acids are manipulated in the diet without taking account of interactions with other amino acids (or with other macronutrients, notably carbohydrate) are at risk of being misinterpreted—a message that applies to studies on other animals too.

What about mammals? Although it is widely held that caloric restriction, not specific nutrient effects, is responsible for life span extension in mammals (Weindruch and Walford 1988; Masoro 2005; Everitt et al. 2010), no experiment to date has contained sufficient dietary treatments to disentangle calories from specific nutrients (Simpson and Raubenheimer 2007). There have been numerous reports, stemming back to early work by Ross (1961), that protein restriction, and restriction of methionine in particular, extends life span in rodents (Orentreich et al. 1993; Zimmerman et al. 2003; Miller et al. 2005; Ayala et al. 2007; Sun et al. 2009), so it is at least plausible that the response of mammals—including humans—is similar to that of insects.

Spurred on by the need for a geometric analysis of aging in mammals, we have embarked upon just such a study in mice with David Le Couteur at the ANZAC Research Institute in the University of Sydney. A full design for rodents has required expanding from two to three macronutrient dimensions with the inclusion of dietary lipid in addition to protein and carbohydrate. At the time of writing, the 30-diet experiment is still underway, but the data are already proving to be instructive.

## 4.1 How Does Macronutrient Balance Affect Life Span?

We have seen that eating excess protein relative to nonprotein energy shortens life span, at least in insects and perhaps also in mammals. The mechanisms causing this effect are not yet understood, but there are some tantalizing candidates. These include altered production of radical oxygen species ("free radicals") with associated damage to DNA and cellular pro-

teins (Sanz et al. 2004; Ayala et al. 2007); toxic effects of nitrogenous breakdown products arising when protein is used instead of carbohydrate or fat as an energy source; changes in immune function and alteration in the capacity to deal with other dietary toxins (as we discuss in chapter 5); and perhaps even changes in the entrainment of circadian rhythms (Hirao et al. 2009). However, it is becoming increasingly apparent that the central coordinators of the effect of macronutrient balance on life span are the nutrient-signaling pathways that we introduced in chapter 3. These pathways are shared by a diversity of organisms from yeasts to humans and include the insulin/insulin-like growth factor (IGF), TOR, and AMPK pathways (Kapahi et al. 2010; Kenyon 2010; Katewa and Kapahi 2011; Mair et al. 2011). It is not only aging that is affected by these pathways; they are emerging at the heart of multiple life-threatening disease processes, including eating disorders such as anorexia and cachexia (a wasting condition common in cancer patients), obesity, cancer, type 2 diabetes, cardiovascular disease, and other metabolic disorders (fig. 4.1). What is needed next are biochemical and molecular genetic studies in which gene expression patterns and metabolic responses are mapped as surfaces onto nutrient intake arrays, as has been done for major life history variables such a life span and fecundity (plate 3). Such studies will help unite nutrition, aging, and their affiliated diseases within a single explanatory framework, spanning genes to behavior.

Increasing the ratio of protein to nonprotein energy in the diet decreases life span, but if this ratio falls too far there is an increased risk of an early death associated with obesity. We will address this issue in detail in chapter 10, but it warrants some discussion here. The reason why the risk of obesity increases as the dietary ratio of protein to nonprotein energy falls below the intake target ratio is that many animals, especially herbivores and omnivores (including humans, as we shall see in chapter 10), regulate their intake of protein more strongly than that of carbohydrate and fat. Consequently, when confined to diets that are high in the proportion of fat and/or carbohydrate relative to protein, animals overeat to gain the target protein intake. Unless these excess calories from fat and carbohydrate are voided by increased activity levels or the up-regulation of thermogenic (heat-generating) mechanisms (see chapter 3), the animal becomes obese and prone to various metabolic disorders. As we discuss in chapter 6, the propensity to store excess calories as body fat, rather than burn them off, varies among species, populations, individuals, and sexes, and can be shown to shift across generations in response to a change in the nutritional environment (e.g., Warbrick-Smith et al. 2006). An example of how individuals of the same species differ can be seen in the comparison of male and female field crickets shown in plate 3C and D; other examples are provided in chapter 6.

usually formulated is not correct, nor is the variant hypothesis that there are direct costs of reproduction that shorten life span (see also Flatt 2011; Tatar 2011).

## 4.3 Conclusions

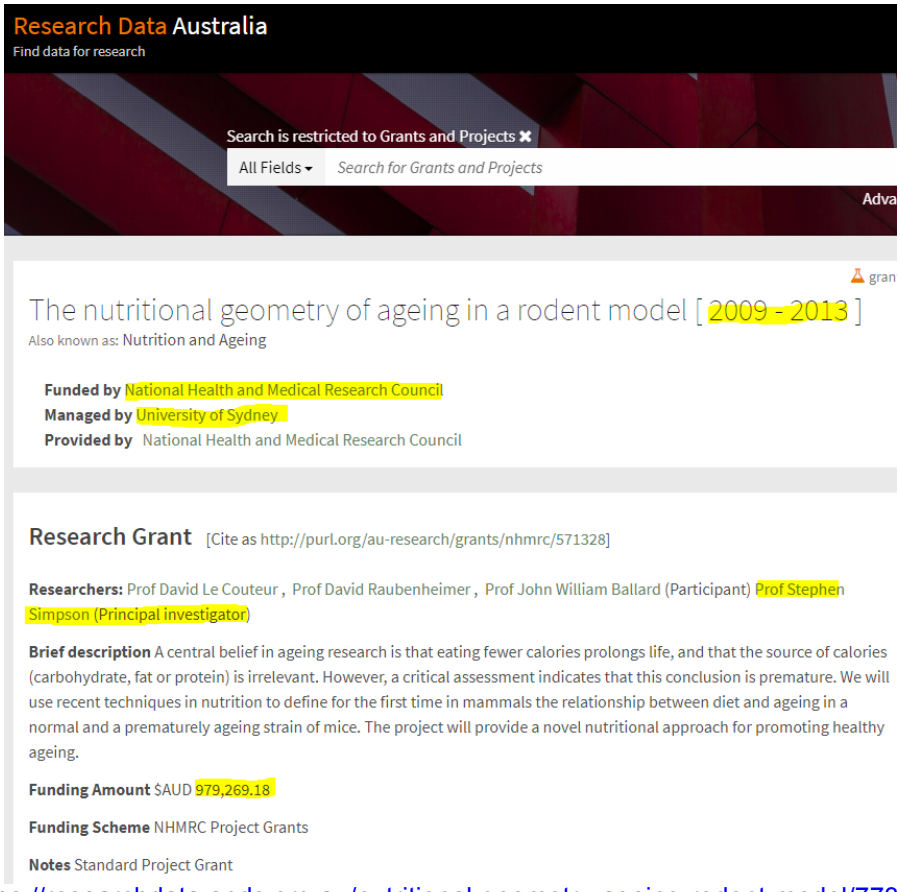
Dietary restriction without malnutrition is considered to be a universal mechanism for prolonging life span. It is generally believed that the benefits of dietary restriction arise from eating fewer calories. However, GF experiments on insects in which the effects of macronutrients have been separated indicate that, rather than calories, a key determinant of the relationship between diet and longevity is the balance of protein to nonprotein (fat and/or carbohydrate) energy in the diet. Whether the same is true for mammals remains to be seen, but existing data indicate that it may well be.

As we shall see in following chapters, the ratio between protein and nonprotein energy intake affects not only life span but also total energy intake, metabolism, immunity, and the likelihood of developing obesity and associated metabolic disorders. Among various possible mechanisms linking macronutrient balance to life span, the interaction between the TOR and AMPK signaling pathways is emerging as a central coordinator. The nutrient signals that activate these pathways remain to be elucidated, but it is likely that a mixture of amino acids must be elevated in the circulation to produce protein satiety and to activate parallel metabolic pathways that are implicated in aging.

Finally, the presumption in much of life history theory that life span and reproduction trade off against each other for limiting resources (usually considered to be energy) is shown to be too simplistic. These two life-history variables certainly have differing nutritional optima, but they can be dissociated and do not inevitably trade off. Reproductive senescence and aging may proceed at different rates in males and females, as predicted by sexual selection theory.

In the next chapter we show that it is not only aging and reproduction that have differently shaped response surfaces when mapped onto nutrient intake arrays, but so too do the physiological systems that respond to toxins and disease.

## NHMRC's concern regarding misrepresentation in 2014 paper puts authors' new $13m research funding at risk

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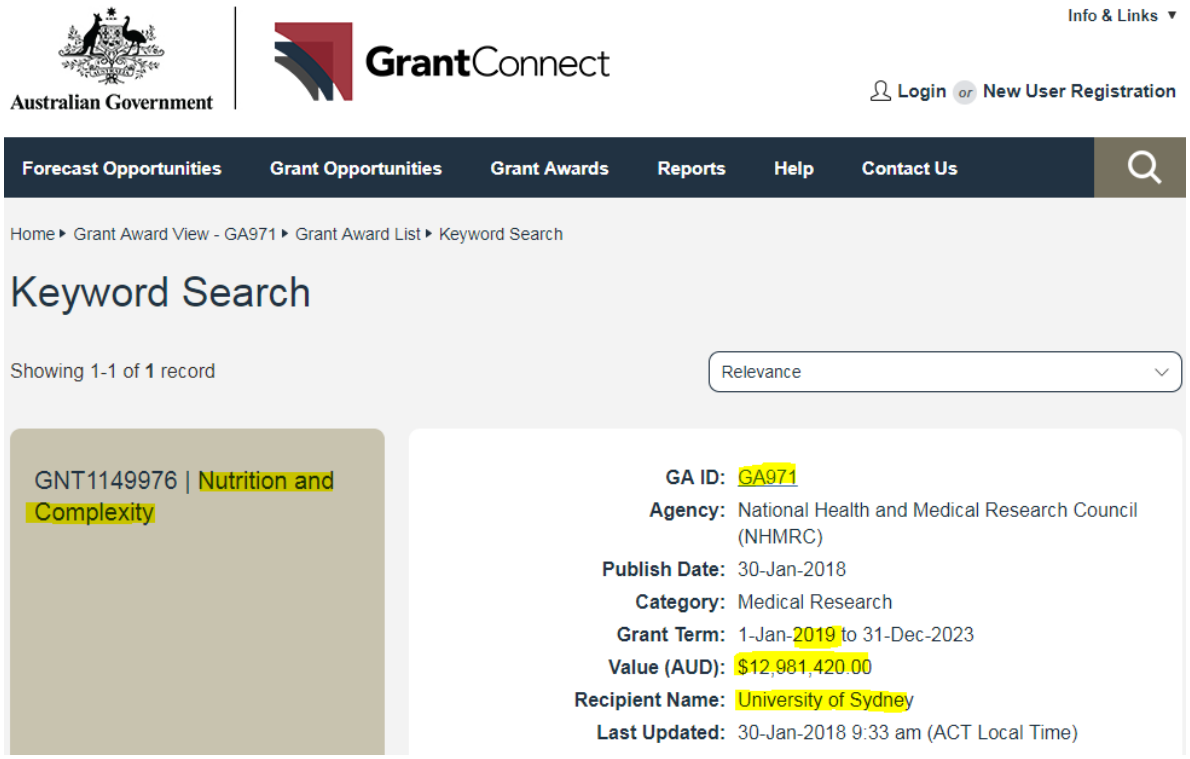



**Purpose:**
Nutrition shapes the relationship between genes and health, and failure to attain dietary balance has profound biological consequences leading to disease. This Application proposes an integrated program that harnesses advances in nutritional theory, systems metabolism, and data modelling that evaluates the effects of macro- and micro-nutrients on mice, cells and humans. This will provide the scientific foundations necessary for the development of evidence-based precision nutrition.
https://www.grants.gov.au/?event=public.GA.show&GAUUID=A88D3135-0238-7750-40C0D7DCFCCCF9B9
https://pdfs.semanticscholar.org/8d58/7c7cb42378e6e263223edd4abc8e5bc9d801.pdf

**Disaster: 10-15%+ of over-55s suffer type 2 diabetes, caused by decades on (sugary) high-carbohydrate diets**



*Table 5: Prevalence of selected serious illness conditions, by gender and age group in 2017 (%)*

| | Males | | | Females | | |
|---|---|---|---|---|---|---|
| | 15–34 | 35–54 | > 55 | 15–34 | 35–54 | > 55 |
| Arthritis or osteoporosis | 1.1 | 9.2 | 27.6 | 1.6 | 11.2 | 45.9 |
| Asthma | 10 | 8 | 9 | 11.5 | 11.7 | 12.9 |
| Any type of cancer | 0.2 | 2 | 9.1 | 0.4 | 2.5 | 5.6 |
| Chronic bronchitis or emphysema | 0.4 | 0.7 | 4.4 | 0.2 | 1.5 | 4.6 |
| Type 1 diabetes | 0.5 | 0.8 | 2 | 0.4 | 0.9 | 1.2 |
| Type 2 diabetes | 0.5 | 3.3 | 15.2 | 0.5 | 3.1 | 10.3 |

https://melbourneinstitute.unimelb.edu.au/__data/assets/pdf_file/0005/3126038/LivingInAus-2019.pdf

**Alas, it was known a century ago that excess consumption of sugar and carbohydrate causes type 2 diabetes**

The following are the conditions which influence the appearance of sugar in the urine:

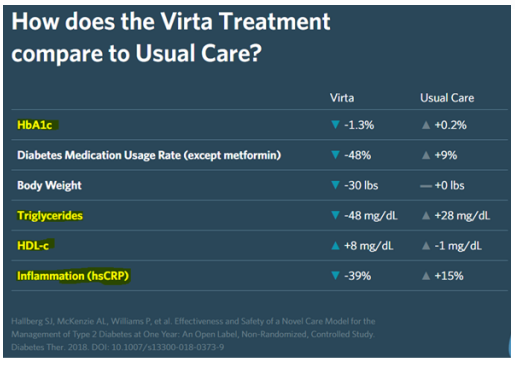
(a) EXCESS OF CARBOHYDRATE INTAKE.—In a normal state the sugar in the blood is about 0.1 per cent. In diabetes the percentage is usually from 0.2 to 0.4 per cent. The hyperglycæmia is immediately manifested by the appearance of sugar in the urine. The healthy person has a definite limit of carbohydrate assimilation; the total storage capacity for glycogen is estimated at about 300 gms. Following the ingestion of enormous amounts of carbohydrates the liver and the muscles may not be equal to the task of storing it; the blood content of sugar passes beyond the normal limit and the renal cells immediately begin to get rid of the surplus. Like the balance at the Mint, which is sensitive to the correct weight of the gold coins passing over it, they only react at a certain point of saturation. Fortunately excessive quantities of pure sugar itself are not taken. The carbohydrates are chiefly in the form of starch, the digestion and absorption of which take place slowly, so that this so-called alimentary glycosuria very rarely occurs, though enormous quantities may be taken. The assimilation limit of a normal fasting individual for sugar itself is about 250 gms. of grape sugar, and considerably less of cane and milk sugar. Clinically one meets with many cases in which glycosuria is present as a result of excessive ingestion of carbohydrates, particularly in stout persons and heavy feeders—so-called lipogenic diabetes—a form very readily controlled.

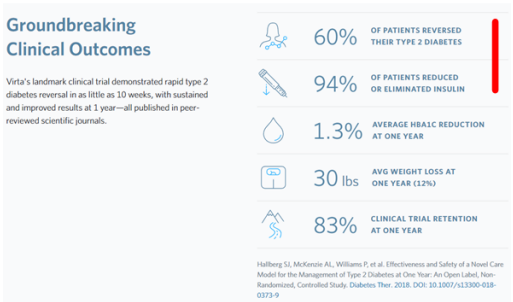
https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf

**Today, competent US scientists, doctors and dietitians use LCHF diet (via 1923 med. text) to fix type 2 diabetes in ~60% patients (v. <1% usual care), overseeing large reductions in weight and use of costly ineffective drugs**

Diabetes Therapy
April 2018, Volume 9, Issue 2, pp 583–612 | Cite as

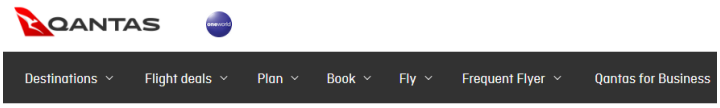
Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at 1 Year: An Open-Label, Non-Randomized, Controlled Study




https://www.virtahealth.com/research ; https://link.springer.com/content/pdf/10.1007%2Fs13300-018-0373-9.pdf

**Main author of high-carb mouse-diet fraud is Qantas's main scientific advisor on diet/menu and "well-being"**




THE EXPERIENCE

## Qantas and Charles Perkins Centre announce partnership


*Qantas passengers are set to benefit from a world first collaboration between the airline and one of Australia's leading academic institutions to reshape the travel experience.*

*The University of Sydney's* ***Charles Perkins Centre*** *will work with Qantas to help develop the airline's new approach to long haul travel ahead of the first Boeing 787 Dreamliner flights this year. The centre brings together researchers across a variety of fields from nutrition to physical activity, sleep and complex systems modelling. Research projects include strategies to counteract jetlag, onboard exercise and movement, menu design and service timing, pre and post-flight preparation, transit lounge wellness concepts and cabin environment including lighting and temperature.*

***Qantas Group CEO Alan Joyce*** *said the partnership has the potential to transform the journey for passengers, particularly on the long haul routes that the Dreamliner is scheduled to operate. "While the Dreamliner aircraft itself is already a step change for passengers with its larger windows, increased cabin humidity and lower cabin altitude, the findings that will come from Charles Perkins Centre researchers will allow Qantas to design and develop a range of new innovations and strategies to complement the Dreamliner experience". …*

*"****The centre's research has already influenced what meals and beverages we'll be serving onboard*** *... **Neil Perry** is working with the centre on new menus for the 787 flights so we are excited that one of Australia's best culinary minds is **teaming up with the best scientific minds to design the best possible menu to look after both health and hunger**."*

*Qantas and the Charles Perkins Centre are looking at opportunities to involve some Qantas frequent flyers in trials that involve wearable technology in the measurement of existing biorhythms during travel, enabling future products to be developed and designed with the insight of robust data.* ***Professor Steve Simpson, Academic Director of the Charles Perkins Centre****, said the partnership is hugely exciting as it's the first time there has been an integrated multidisciplinary* ***collaboration between an airline and a university around in-flight health and well-being*** *beyond medical emergency. "There is the potential for extraordinary health, science and engineering discoveries and innovations to come out of this research partnership, which will also provide the evidence-base needed for Qantas to implement strategies to further improve how people feel after a long haul flight," he said.*

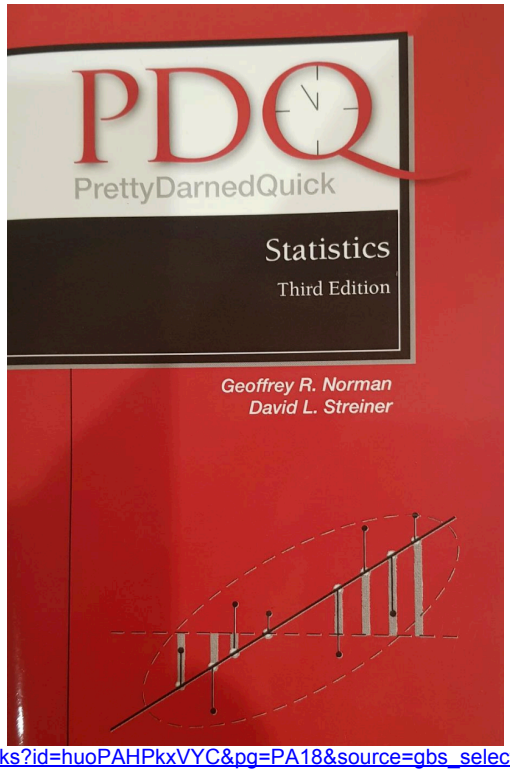
***The University of Sydney's Vice-Chancellor and Principal, Dr Michael Spence****, said the collaboration between the Australian airline and university reflected the vision of both institutions. "The Dreamliner is a transformative project for Qantas, as the Charles Perkins Centre was for the University of Sydney when we brought together multidisciplinary teams of scholars to find solutions to some of the world's most pressing health problems.*
*"Adapting and innovating is in both our DNA. The real-world outcomes from this new partnership have the potential to significantly alter the future experience of long haul flying."*

https://dreamliner.qantas.com/accessibility/article/qantas-and-charles-perkins-centre-announce-partnership/

## *Statistics* textbook says 2014 paper should not have hidden Table S2 before launching statistical shenanigans

chapter. The important point, which we raised in Chapter 1, is that the onus is on the author to convey to the reader an accurate impression of what the data look like, using graphs or standard measures, before beginning the statistical shenanigans. Any paper that doesn't do this should be viewed from the outset with considerable suspicion.

[1]Huff D. *How to lie with statistics.* New York: WW Norton; 1954.




p. 12 in https://books.google.com.au/books?id=huoPAHPkxVYC&pg=PA18&source=gbs_selected_pages&cad=2#v=onepage&q&f=false

## Table S2 falsifies authors' claim that greatest *median* lifespan over 30 diets is via low-protein, high-carb (LPHC)

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

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 | 119.43 | 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 | 113.86 | 147.36 |
| HIGH | 5 | 48 | 48 | 0.10 | 124.43 | 146.21 |
| MEDIUM | 33 | 48 | 20 | 0.69 | 122.57 | 145.71 |
| MEDIUM | 23 | 38 | 38 | 0.61 | 123.86 | 143.07 |
| HIGH | 33 | 48 | 20 | 0.69 | 98.29 | 141.00 |
| HIGH | 14 | 57 | 29 | 0.25 | 117.43 | 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 | 100.00 | 124.57 |
| LOW | 14 | 57 | 29 | 0.25 | 98.57 | 119.43 |
| LOW | 33 | 20 | 48 | 1.65 | 78.57 | 116.36 |
| LOW | 14 | 29 | 57 | 0.48 | 88.71 | 115.07 |
| LOW | 42 | 29 | 29 | 1.45 | 85.85 | 104.00 |
| LOW | 60 | 20 | 20 | 3.00 | 84.29 | 102.86 |
| LOW | 23 | 38 | 38 | 0.61 | 89.29 | 100.36 |

https://ars.els-cdn.com/content/image/1-s2.0-S1550413114000655-mmc1.pdf

**Table 3 (modified)**

| 30 mouse diets spanning ~1000 mice, ranked by median lifespan (weeks) per cohort * # | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | HPLC | P:C > 0.5 (high-protein diet) | | |
| | | | | LPHC | P:C < 0.5 (low-protein diet) | | |
| Diet ranking | Protein: Carb (P:C) ratio | Median lifespan of cohort (weeks) | Protein (%) | Carb (%) | Fat (%) | Energy density | 2-3 oldest mice (weeks) |
| 1 winner | 1.45 | 139 | 42 | 29 | 29 | high | 151 |
| 2 | 0.69 | 127 | 33 | 48 | 20 | low | 134 |
| 3 # | 0.10 | 124 | 5 | 48 | 48 | high | 146 |
| 4 | 0.61 | 124 | 23 | 38 | 38 | medium | 143 |
| 5 | 0.25 | 123 | 14 | 57 | 29 | medium | 152 |
| 6 | 1.45 | 123 | 42 | 29 | 29 | medium | 148 |
| 7 | 0.69 | 123 | 33 | 48 | 20 | medium | 146 |
| 8 # | 0.07 | 122 | 5 | 75 | 20 | medium | 157 |
| 9 # | 0.07 | 119 | 5 | 75 | 20 | high | 152 |
| 10 | 0.25 | 117 | 14 | 57 | 29 | high | 140 |
| 11 | 0.48 | 114 | 14 | 29 | 57 | medium | 147 |
| 12 | 0.48 | 108 | 14 | 29 | 57 | high | 134 |
| 13 | 3.00 | 108 | 60 | 20 | 20 | medium | 130 |
| 14 | 1.65 | 107 | 33 | 20 | 48 | high | 137 |
| 15 | 1.65 | 107 | 33 | 20 | 48 | medium | 134 |
| 16 | 0.25 | 106 | 5 | 20 | 75 | high | 154 |
| 17 | 0.61 | 100 | 23 | 38 | 38 | high | 125 |
| 18 | 3.00 | 100 | 60 | 20 | 20 | high | 128 |
| 19 | 0.25 | 99 | 14 | 57 | 29 | low | 119 |
| 20 | 0.69 | 98 | 33 | 48 | 20 | high | 141 |
| 21 | 0.61 | 89 | 23 | 38 | 38 | low | 100 |
| 22 | 0.48 | 89 | 14 | 29 | 57 | low | 115 |
| 23 | 1.45 | 86 | 42 | 29 | 29 | low | 104 |
| 24 | 3.00 | 84 | 60 | 20 | 20 | low | 103 |
| 25 | 1.65 | 79 | 33 | 20 | 48 | low | 116 |
| 26 * # | 0.07 | 23 | 5 | 75 | 20 | low | 23 |
| 27 * # | 0.10 | 23 | 5 | 48 | 48 | medium | 23 |
| 28 * | 0.25 | 10 | 5 | 20 | 75 | low | 10 |
| 29 * | 0.25 | 10 | 5 | 20 | 75 | medium | 10 |
| 30 * # | 0.10 | 10 | 5 | 48 | 48 | low | 10 |
| | | START (week 1) | | | | | |
| * ~30 mice dead after diet discontinued; cohort died or "failed to thrive" ("would soon have died from malnutrition") | | | | | | | |
| # Diet claimed by authors in 2018 mouse-dementia paper to maximise lifespan (P:C ratio of ~0.1) | | | | | | | |

Source: pp. 7-8 https://ars.els-cdn.com/content/image/1-s2.0-S1550413114000655-mmc1.pdf

Even before Simpson and Raubenheimer's 2012 book, a 2009 paper showed them planning their 30-diet experiment and the LPHC results needed to promote their "unifylng framework", expanding the relevance of their decades of work with insects to mice, then to humans (p. 877 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2815731/pdf/aging-01-875.pdf ). Again, the main co-authors of the 2014 paper were "highly motivated" to find what they *needed* to find, and they "found" it. They now insist: "Median lifespan was greatest for animals whose intakes were low in protein and high in carbohydrate".

**3. Looking at Table 3, Professor Simpson dishonestly claims: "Rory's concerns are in every respect unfounded"**

I think objective observers can see from my Table 3 that median lifespan was ***not*** greatest on LPHC diets. Clearly, five of the top-seven diets for median longevity are **HPLC** (P:C > 0.5) diets. Simpson *et el*'s "finding" that LPHC (P:C < 0.5) diets outperformed relies on "statistical shenanigans" that delete LPHC diets 26-30 and "disappear" key HPLC diets 1, 2 and 4.

For science careerists with their "Protein restriction [LPHC] extends lifespan" claim nailed to their mast, Simpson *et al*'s three years watching their career-defining experiment falsify their LPHC hypothesis must have been a living hell. We can mimic an **"action replay"** by working our way up from the bottom of Table 3. Simpson's nightmare began straight away, when cages of LPHC mice "failed to thrive" and started dying: five 5%-protein diets (26-30 above) had to be discontinued.

By 10-23 weeks, roughly 150 LPHC mice were dead. When pressed by me, Simpson conceded that mice on those five killer LPHC (all P:C < 0.3) diets were euthanised because they "**would soon have died from malnutrition**" (*Submission*, p. 24). What he will not concede is that dead and dying mice are **gold** in any longevity study, so his dead LPHC mice remain hidden from readers of the 2014 paper's main text. Imagine the trauma suffered by the parents of those ~150 young LPHC mice. Ever the professional, Simpson kept a straight face while notifying parents that not only are your sons and daughters dead but they died in vain, as I'm ditching them from the lifespan results in my journal article. Through their tears, the mummy and daddy mice heard Simpson mumble that 15% of ~1,000 mice dying young is completely irrelevant when a career-defining "unifying framework" is at stake. Not relevant? ~150 young mice are dead! It's a lifespan analysis?

That first round of disastrous news for Simpson came fast. The next round came slowly, agonisingly slowly: hundreds of mice on high-protein (P:C > 0.5) diets simply wouldn't die, as Simpson had predicted years earlier. Hundreds of HPLC

mice just kept going and going *and going*, outlasting even the famous Energizer Bunny. Notably, the five best HPLC diets (Diets 1, 2, 4, 6 and 7) devastated Simpson's forecast that LPHC diets would outperform. One can imagine smoke rising from "Principal investigator" Simpson's ears if, at 122.5 weeks, his smart, young (pliable?) research assistant and co-author (his PhD student at the time) had asked why more mice were surviving on those five HPLC diets than on 23 of the 25 other diets. Again, by 122.5 weeks, five of seven median mice still alive were living on HPLC diets. You can bet he didn't confess: "What a debacle: My LPHC-extends-lifespan credibility just died with those hundreds of LPHC mice".

As the results rolled in, it turned out that Simpson's LPHC mice were nothing like his LPHC insects. Imagine being a fly on the wall listening to any conversations between the four heaviest-hitter co-authors - Simpson and the three Davids: Raubenheimer, Le Couteur and Sinclair (Harvard) - as the LPHC hypothesis was falsified. (Unlike the other Davids, Harvard's famous David is not emotionally invested in Simpson's "Protein restriction [LPHC] extends lifespan" story.)

It must have been excruciating for Simpson, Raubenheimer and Le Couteur as they waited and waited *and waited* for the *longest-lived* median mouse to die: that middle mouse on Diet 1 (42% protein, 29% carbs; P:C 1.45) lived ~12 weeks or nearly three months *longer* than the big 127 weeks survived by the median mouse on Diet 2. At week 138, Simpson may have been struggling, perhaps screaming "Just die already" at the mice on Diet 1, startling many HPLC old-timers sitting about innocently playing cards and watching TV. David Sinclair from Harvard might then have playfully advised Simpson to "restrict their protein", joking that protein restriction appeared to be an effective mouse killer (not lifespan extender).

These four men all are distinguished science careerists. When that HPLC median mouse on Diet 1 (P:C 1.45) died aged a gob-smacking 139 weeks, they all knew the score: HPLC had dominated the long-planned 30-diet mammal experiment. Median lifespan was greatest on a diet high (42%) in protein and low (29%) in carbohydrate, while five of the top-seven diets for median lifespan were HPLC. All this made nonsense of Simpson and Raubenheimer's "protein restriction [LPHC] extends lifespan" hypothesis. Alas, their career-boosting unifying-framework dreams had been devastated by hundreds of notably short-lived LPHC mice. **Unless... Unless what?** Unless their 30-diet experiment's results could be "fine-tuned" to show that *LPHC diets had indeed maximised median lifespan*. Don't be silly, said one. How? said Fred. After the authors agreed to delete those ~150 dead mice on five killer LPHC diets from their formal write up, did one thing lead to another?

Did the heaviest-hitters (Simpson and the three Davids) suggest or agree to hide from readers of the paper's main text the profound fact that the HPLC median mouse on Diet 1 lived 10% - a decade in human years! - longer than the next oldest of 30 medians? I have no idea. But that's what Rebecca's Investigators need find out. **Co-author David Sinclair (UNSW and Harvard) may be able to help, as he's *really* smart**. A giant in the science of prolonging lifespan, he was on *TIME*'s list of "100 most influential people in the world": https://genetics.med.harvard.edu/sinclair/people/sinclair.php

Critically, despite the *actual* diet-and-lifespan results of the experiment (in Table 3) ultimately looking nothing like what Simpson and Raubenheimer had planned and *needed*, the 2014 paper was published with **LPHC declared the winner**:

- "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"; and "...there was a clear correlation between the [P:C] ratio and lifespan" (p. 421).
- Further, "Mice consuming a low-protein, high-carbohydrate, low-fat diet (LPHC, protein:carbohydrate ~1:10) lived longest...", according to the authors' subsequent 2018 mouse-dementia paper (p. 2/17 https://www.cell.com/cell-reports/pdf/S2211-1247(18)31674-7.pdf ).

Marketing their invented "findings" to the public, influential Charles Perkins Centre science careerists duped ABC radio's national audience, including by claiming: **"...what we did was design 25 [not 30] diets**"; "If you're interested in a longer lifespan…then a diet that is low in protein, high in carbohydrate…is preferable"; and "The healthiest diets were the ones that had the lowest protein, 5 to 10 to 15% protein" (pp. 20-22 below). No mention of ~150 dead young 5%-protein mice!

Again, Table 3 shows the authors' LPHC claims are false, blatantly misrepresenting the *actual* results. In fact:

• Median lifespan was greatest by far for mice on a diet *high* in protein (42%) and low in carbohydrate (29%; P:C 1.45);
• That HPLC diet's median mouse lived for 139 weeks, 10% longer (a decade in human years!) than on the next-best diet;
• **Three of the top-four diets and five of the top-seven diets** for median lifespan are *high* not low in protein (P:C> 0.5);
• So too, 10 of 18 diets (56%) on which the median mouse **lived for at least 100 weeks** are high in protein (HPLC); while
• Seven of 12 diets (58%) on which mice struggled to thrive - with the median mouse **dead before 100 weeks** - are LPHC

Importantly, **three of the six P:C ~1:10 (~0.1) diets** that Charles Perkins' 2018 mouse-dementia paper (p. 31; largely the same authors as the 2014 paper) says maximises longevity are **three of the five killer 5%-protein diets** in Table 3 on which all the mice were dead by 23 weeks. In the 2014 paper, three killer P:C ~0.1 diets maximised early death in mice, a key fact hidden from readers; in the 2018 paper, such diets are said to maximise longevity! What is going on?

How did the false claims get published? That's what Investigators need to know. Was it merely hopeless incompetence? Did 18 co-authors not know that dead animals are the "bread and butter" of any longevity analysis? Who decided it's okay to publish a formal discussion hiding dead mice and other key lifespan results from readers? Who scrambled the median-lifespan data in Table S2 and parked it in "Supplemental information", hiding from readers the fact that one HPLC diet has

a median lifespan 10% longer than any of the 29 other diets? Were the authors merely incompetent? I could perhaps believe that, if Simpson in January had not responded to the clear, valid and substantial concerns in my *Expression of Concern* with his dishonest blanket denial: "...Rory's concerns are in every respect unfounded" (p. 21, my *Submission*). That was a profoundly revealing response. If I didn't know for sure beforehand, I knew then: the Principal Investigator is determined to pretend that all is fine despite knowing that his claim - median lifespan is greatest on LPHC diets - is false.

**4. Summary, and significance of Charles Perkins Centre's research frauds: early death in Indigenous Australia**

In my *Submission*, I documented that Professor Stephen Simpson as Academic Director of the Charles Perkins Centre has been overseeing not one but two serious scientific frauds: Professor Jennie Brand-Miller's infamous *Australian Paradox* sugar-and-obesity fraud, and his own sugary LPHC median-lifespan fraud. There are now four reasons why I am confident that this episode is properly described as a serious scientific fraud:

(i) The authors' conclusions are falsified by their *actual* results, presented in their Table S2 and my Table 3.
(ii) Professor Simpson can see (i) but dishonestly insists that "Rory's concerns are in every respect unfounded".
(iii) Simpson is the main University of Sydney manager unethically protecting Professor Jennie Brand-Miller's infamous *Australian Paradox* sugar-and-obesity fraud, including by actively assisting – as head of "Faculty" - her dishonest 2017 expansion into the *American Journal of Clinical Nutrition* (pp. 5-6 in my *Submission*).
(iv) The three items above give me complete confidence that we are dealing with serious scientific fraud. The 2012 book by Professors Simpson and Raubenheimer (pp. 2-6 earlier) provides the needed "**why?**" - as in, why would eminent science careerists risk blatantly misrepresenting the *actual* median-lifespan results?

Again, I only became 100% confident that Simpson's blatant median-lifespan misrepresentations are part of a major scientific fraud in January of this year, when Simpson responded to my heavy-hitting *Expression of Concern* by dishonestly claiming to his *Cell Metabolism* journal's Editor-in-Chief Nikla Emambokus (nemambokus@cell.com ), its Editorial Board and a local journalist that "...Rory's concerns are in every respect unfounded" (p. 21 of my *Submission*).

The paper should be retracted, then rewritten under competent and honest supervision so that the 30-diet experiment's *actual* lifespan results are properly presented and discussed. Taxpayers don't like funding blatant dishonesty. It is particularly troubling that in June, soon after I had provided my *Submission* to the Academic Board and Chief Counsel, an anonymous "University spokesperson" began responding to media inquiries with the authors' false, dishonest defence:

*We [the University of Sydney] note the authors of the [2014] paper continue to reject Mr Robertson's claims as profoundly misconceived and unfounded. The findings of the study are supported by various works from independent research groups. The study is highly cited in the field of the biology of ageing. No questions have been raised by members of the board or other members of the scientific community*. http://honisoit.com/2019/06/peak-medical-research-body-asks-usyd-to-investigate-concerns-2/

Principal Investigator Simpson's claim that "The findings of the study are supported by various works from independent research groups" is a deliberate furphy. Other work elsewhere is not relevant. The harsh reality for the 18 authors – including Harvard's superstar longevity researcher David Sinclair – remains that the median-lifespan "findings" claimed in their 2014 paper are clearly falsified by the *actual results* of the 30-diet experiment published in Table S2 and my Table 3.

Which "independent research groups" support "disappearing" ~17% (5/30) of an experiment's results from its lifespan analysis? Who supports the authors hiding from readers the fact that - rather than prolonging median lifespan - five LPHC diets worked to kill 15% of the ~1,000 mice rather quickly? Let's hear from those who support Simpson and Sinclair *et al* hiding the extraordinary fact that the median mouse on one HPLC diet lived 10% (a decade in humans years) longer than any of the 29 other median mice. Who exactly supports the (false) claim that median lifespan is greatest on LPHC diets?

In my opinion, this episode is a **classic case-study in scientific fraud**, motivated as usual by prestige and money:

- Influential science careerists decided that they know how the world works ("protein restriction extends lifespan").
- To prove their preferred story, they planned a big career-defining experiment (30 diets, 1,000 mice, three years).
- They wrote a book highlighting the experiment and findings needed, *needed* to boost careers and new funding.
- Alas, the experiment surprised authors by rejecting their "protein restriction [LPHC] extends lifespan" hypothesis.
- Careerists then rejected the *actual* results that falsified their hypothesis, choosing to invent the *needed* "findings".
- "Peer review" was laid-back and lax for influential careerists, allowing *actual* results to be hidden from readers.
- Heaps of dead LPHC mice and long-lived HPLC median mice "disappeared" as invented "findings" published.
- Harvard's longevity superstar turned up on list of 18 authors – did he notice *actual* results contradict "findings"?
- Whistleblower alerts authors' journal to blatant misrepresentations, with authors dishonestly denying all problems.
- False mouse-diet "findings" used to promote LPHC diets that in humans cause type 2 diabetes and early death.
- NHMRC took whistleblower's concerns seriously, forcing longevity-fraud investigation at University of Sydney.
- NHMRC funded initial mouse study with $1m grant, but mouse-diet group's new $13m of funding now is at risk.
- University of Sydney and Group of Eight promise "excellence", so sci-fraud puts at risk billions of dollars per year.
- The paper should be retracted then rewritten so that *actual* lifespan results are properly presented and discussed.
- Beyond taxpayers funding shonky science, Charles Perkins is Qantas's scientific advisor on diet and health (p.9).

To claim my concerns are "unfounded" is dishonest pretending. While *Cell Metabolism* journal's Editor-in-Chief Nikla Emambokus has said nothing so far, she too presumably can now see the big problems earlier hidden by Professor Simpson *et al*, assisted by the paper's colourful three-dimensional charts that most observers don't really understand.

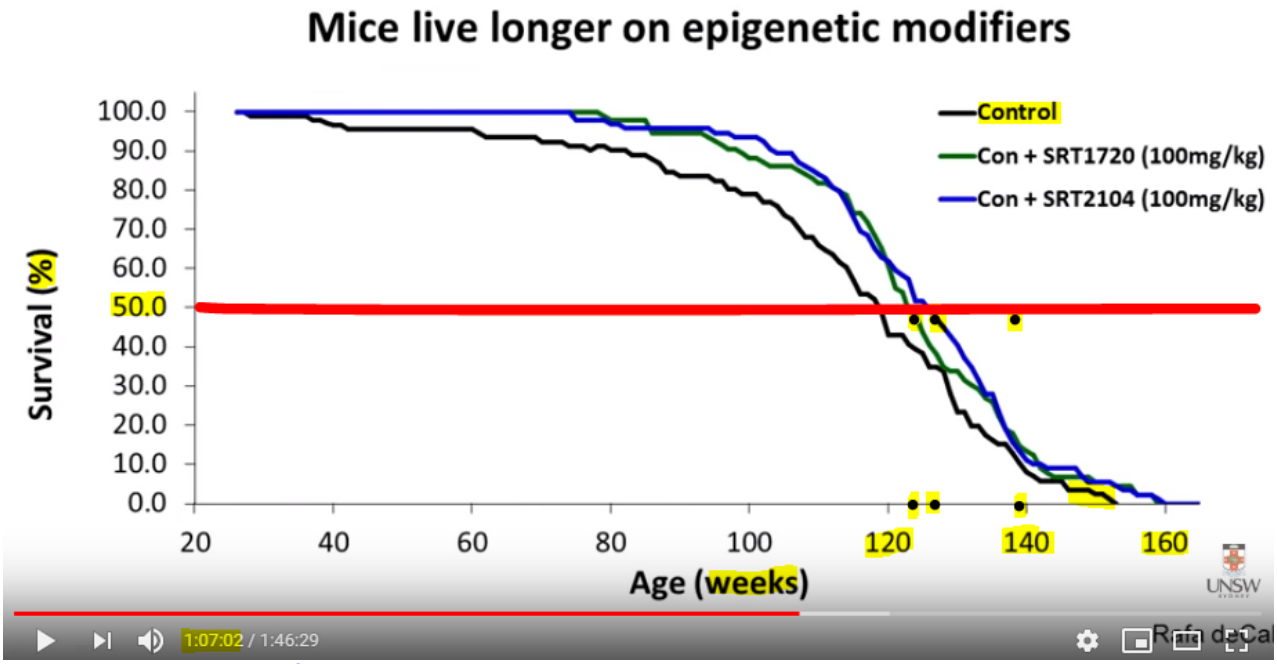
What is the significance of what I have documented? Well, for starters, we now know that the great scientific race to boost human longevity – reported in the ***Sydney Morning Herald*** **in 2017 by Liam Mannix: "Fountain of youth: Australian scientists in race to find a cure for ageing"**: https://www.smh.com.au/technology/three-australian-teams-race-each-other-and-time-itself-to-crack-a-cure-for-aging-20171027-gz9j2g.html – is an expensive sham. "Dr Simpson said [his] team and their Australian competitors were among the world's best anti-ageing researchers. 'If you include us and University of NSW and Monash teams, I think we have probably the strongest group in ageing biology anywhere'."

Two of the three groups in that "race" - from the University of Sydney and UNSW/Harvard - are involved in this mouse median-lifespan fraud. At a grand scientific lecture at UNSW in 2014, Simpson – alongside Professor Sinclair - declared that his career-defining experiment had discovered that mammals are just like his insects: "And that paper caused quite a stir... Now, what we found [via "900 mice" on "30 experimental diets"]...was that longevity in the mice was also, **just like the fly**, greatest on low-protein, high-carbohydrate diets": minute 28:20 https://www.youtube.com/watch?v=x0-Jt7az-54

Again, Simpson's "protein restriction [LPHC] extends lifespan" story for mice is falsified by Table 3. Moreover, extending his LPHC advice to humans promotes tragedy: his sugary LPHC mouse diet for humans is a perfect recipe for type 2 diabetes, misery and early death, especially in Indigenous communities (pp. 8, 24-34). With his Charles Perkins Centre's two shameless diet-advice frauds harming public health, Simpson's team should not receive another cent from taxpayers.

I'm agnostic on what this longevity scandal means for UNSW and Harvard superstar David A. Sinclair and his research teams. Investigators need to ask Sinclair why exactly his name is on Simpson *et al*'s 2014 paper. Did he even consider the *actual* lifespan results before the paper was published? Unlike the other heavy-hitters among the 18 co-authors, Sinclair has had little formal involvement with Simpson's research over recent decades. What did he do to get his name on the paper besides bringing his prestige to it? Did his name help encourage lax "peer review", allowing publication without proper scrutiny, without anyone noticing that the *actual* median-lifespan results (that falsify the claimed "findings") had been hidden from readers? Again, Sinclair was "one of the 100 most influential people in the world" and in 2018 was awarded an Order of Australia (AO): https://medicalsciences.med.unsw.edu.au/people/professor-david-sinclair%20

Notably, Sinclair and Simpson are competitors. Speaking after Simpson at that UNSW lecture in 2014, Sinclair showed a chart of his drug research using mice, nothing to do with his and Simpson's supposedly path-breaking 2014 mouse-diet paper. That chart, below, shows mice fed not Sinclair's cancer-suppressing "Resveratrol" but an even "more potent" drug. Please notice two things. First, Sinclair's "**controls**" on usual chow lived into the 150s (weeks), similar to the old outliers on special diets in Simpson and Sinclair's "path-breaking" 30-diet experiment. **Again, the oldest *controls* in Sinclair's drug tests and oldest *outliers* on Simpson's special diets both lived to around the same age, the 150s** (see Table 3). In what sense did Simpson's diets extend longevity? **Second, the longest-lived median in Simpson and Sinclair's 2014 paper (139 weeks on a HPLC diet) lived longer than the median mice on Sinclair's drug-boosted diets** (that is, the blue and green lines below show the median - 50th percentile - stuck in the 120s). So, Simpson and Sinclair's 2014 paper hid from readers the profound fact that the median mouse on a HPLC diet lived to 139 weeks, which is both ~10% longer than for any of the 29 other diets in that experiment ***and*** ~10% longer than Sinclair's drug-boosted medians. What chance that - despite heaps of funding and impressive careers being made - **nothing much useful is happening here**?




https://www.youtube.com/watch?v=x0-Jt7az-54 ; https://newsroom.unsw.edu.au/news/health/making-age-reversal-real

**5. Charles Perkins research fraud suppressing cure for type 2 diabetes; University of Sydney highly conflicted**

Importantly, one thing Simpson got right when planning his 30-diet mouse experiment in 2009 was his assesment that the main way to boost longevity in primates - including humans - is via "a reduction in the incidence of diabetes, cancer and cardiovascular disease [CVD]": p. 877 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2815731/pdf/aging-01-875.pdf

That's where I come in. With over one million Australians today suffering type 2 diabetes, the number growing rapidly, the big problems with competence and integrity at the Charles Perkins Centre matter a great deal. Tragically, the dietary cause of type 2 diabetes – excess consumption of sugar and other carbohydrate – was documented at the highest levels of medical science as early as 1923, a century ago. Back then, GPs across the western world could and would quickly treat the malady into remission. Today, despite that treatment's ability to fix ~60% of patients within 12 months - versus a tragic ~1% on usual care - that effective cure is suppressed by influential incompetence and worse (pp. 23-31).

The **scandalous mistreatment** of millions of people with type 2 diabetes – here and abroad – is why I remain determined to fix faulty and harmful "science" at the University of Sydney. The main effect of the Charles Perkins Centre's infamous *Australian Paradox* sugar-and-obesity fraud and its sugary LPHC mouse-diet deception is to suppress the effective dietary cure for type 2 diabetes, stopping millions of everyday people being rescued from misery and early death (p. 8).

**Ironically, the birth of the Charles Perkins Centre in 2012 was supposed to reduce - not boost - "the burden of diabetes, obesity and cardiovascular disease, and their related conditions."** https://sydney.edu.au/charles-perkins-centre/ Unfortunately, the two sugary high-carb deceptions that two high-profile careerists – Professors Stephen Simpson and Jennie Brand-Miller – refuse to correct are pushing Australia in *exactly* the wrong direction. As I wrote to ACCC Chair Rod Sims, the Charles Perkins Centre's false scientific claims in the public debate promote elevated modern doses of sugar and carbohydrates as harmless, even healthful, when in fact sugary high-carb diets cause type 2 diabetes, misery, CVD and early death, particularly in Indigenous communities: https://www.australianparadox.com/pdf/Letter-to-ACCC.pdf

Professor Simpson and University of Sydney Vice-Chancellor Michael Spence - whose advertisements suggest that (false) mouse-diet results extend automatically to humans (p. 22) – so far have failed to address the fact that **mice and humans have profoundly different metabolic responses to sugary high-carbohydrate diets** (pp. 23-24). So the Charles Perkins Centre continues to promote as lifespan-extending a sugary LPHC mouse diet that is distressingly similar to the sugary low-protein, high-carbohydrate diets that are driving type 2 diabetes, misery and early death by the thousands across Australia, particularly in Indigenous communities (pp. 8, 25-34).

These problems need fixing. And it's excellent that the University of Sydney's 2019 research-fraud Investigation is now underway (p. 17). Unfortunately, following the debacle of its 2014 *Australian Paradox* research-fraud Inquiry (*Submission*, pp. 5-6), I strongly suspect that another unethical whitewash is in the pipeline. If I am right, the authors' false and harmful LPHC mouse-diet claims will remain on the scientific record and will continue to poison public debate on diet and health.

On the need to fix the Charles Perkins Centre's LPHC mouse-lifespan deception, the University of Sydney's leadership team is highly conflicted. Doing the right thing – formally retracting the false information - is not its only option:

(i) The University of Sydney has not been penalised at all for its ongoing support of Professor Jennie Brand-Miller's infamous *Australian Paradox* fraud (again, pp 5-6 in my *Submission*). The University could simply retract the LPHC mouse-lifespan falsehoods, as it should, but Vice-Chancellor Michael Spence and his senior management may feel that the best approach is to unethically oversee another research-integrity whitewash.

(ii) The University of Sydney's management was itself duped by the 2014 paper, paying for full-page newspaper advertisements stating that "...our researchers have discovered that a low protein, high carb diet can help us [humans] live a longer and healthier life" (p. 22). Not only is that LPHC median-lifespan "finding" false and misleading (see Table 3), **there is no mention of mice in the ad!** The University could simply retract the faulty mouse-diet paper, as it should, but is Vice-Chancellor Michael Spence prepared to admit that he and his leadership team were duped by the work of ambitious Academic Director of the Charles Perkins Centre?

(iii) Professor Stephen Simpson – the "Principal investigator" on the 30-diet experiment and Academic Director of the Charles Perkins Centre - recklessly extrapolates from mice to humans: "...**mice are not that different from humans**" (p. 27). He disregards the fact that the sugary LPHC diets his work promotes as lifespan-extending in mice are the main cause of type 2 diabetes in humans, driving great misery and early death across Australia. The University could take out a series of corrective advertisements to apologise for pretending that mice and humans do not have profoundly different metabolic responses to high-carbohydrate diets (pp. 22-25), as it should, but is Vice-Chancellor Spence prepared to admit that the Charles Perkins Centre since its launch in 2012 has been a menace to public health, harming particularly the well-being and longevity of the peoples Charlie loved and worked a lifetime to help? (How's that for tragic irony - a travesty?)

(iv) The 2014 mouse-diet paper was funded using a $1m NHMRC grant for the period 2009-2013. Most recently, Simpson's group has secured a further $13m of NHMRC funding to do more of the same, studying "Nutrition Complexity" in "mice, cells and humans" (p. 7). The fresh funding began flowing on 1 January, just before I

sent my *Expression of Concern*. The University could simply retract the faulty 2014 paper, as it should, but is Vice-Chancellor Michael Spence prepared to risk losing the $13m worth of new NHMRC grants, funding that may be withdrawn if it is conceded that the initial grant of $1m funded serious research misconduct?

(v) **The University of Sydney is gifted ~$700m each year by taxpayers while Group of Eight (Go8) universities receive "two-thirds of all research funding to Australian Universities".** Those outsized amounts exist because Go8 universities have promised taxpayers, politicians and hundreds of thousands of fee-paying students that the Go8 is uniquely devoted to "excellence". The sad truth – confirmed year after year by Go8 tolerance of the Charles Perkins Centre's infamous *Australian Paradox* fraud - is that Go8 promises of "excellence" are a sham. **The sad truth is that there is no competent and honest quality control when it matters.** University of Sydney management is highly conflicted on "excellence" in research, knowing that *reintroducing* proper quality control now would reveal it's been **defrauding taxpayers and students on a massive scale for years**, by simply ignoring the need for competence and honesty, let alone "excellence". (Much of the discussion above is drawn from my December 2018 letter to ACCC Chair Rod Sims and my *Submission to ACCC's Scamwatch*: https://www.australianparadox.com/pdf/Letter-to-ACCC.pdf

**6. Endpiece**

With its high-profile *Australian Paradox* sugar-and-obesity fraud, its misguided promotion of sugary LPHC mouse diets as excellent for human longevity, and its sales of millions of Low-GI diet books claiming that "There is absolute consensus that sugar in food [and drink] does not cause [type 2] diabetes", the Charles Perkins Centre is driving incalculable harm. In particular, it is a public-health scandal that Professors Simpson and Jennie Brand-Miller are using their clearly faulty "scientific" advice to (mis)inform diabetes educators, falsely suggesting that sugary "low GI" and/or low-protein, high-carbohydrate diets are likely to boost the health, well-being and/or lifespans of people with type 2 diabetes:

**JBM**: https://www.australianparadox.com/pdf/letterbdusydfraudaustdiabetesconf.pdf
**SJS**: https://daa.asn.au/wp-content/uploads/2016/11/Minutes-Diabetes-Interest-Group-17th-June-2015.pdf

Refusing to correct the blatant false claims behind its dietary advice, while promoting high-carbohydrate diets to people suffering type 2 diabetes, the Charles Perkins Centre is a menace to public health. Again, modern doses of carbohydrate including sugar are the main cause of type 2 diabetes suffered by over one million Australians (p. 8). By suppressing the known dietary cure for type 2 diabetes, the Charles Perkins Centre is promoting profound harm to public health.

As discussed, I think the false LPHC median-lifespan "findings" of the 2014 mouse-diet paper should be formally retracted, and the paper rewritten under competent and honest supervision. The *Australian Paradox* paper (2011) also should be retracted. The need for retraction should be uncontroversial. After all, formal retraction is the standard scientific approach to faulty papers with false conclusions that misinform the community and work to harm public health. The two extraordinarily faulty papers above are highly qualified. Roughly one thousand faulty scientific papers are retracted each year: https://retractionwatch.com/2018/12/28/the-year-in-retractions-2018-what-18000-retractions-and-counting-told-us/

Taxpayers not wanting to fund faulty dishonest "science" for most is a no-brainer, but a range of science and university careerists reportedly have concerns about Group of Eight (Go8) data showing that basic research income for universities declined to $1.6b in 2017 from its earlier peak of $1.8b in 2014: *Funding for basic research disappears*, 24 June, 2019 https://www.afr.com/news/policy/health/funding-for-basic-research-disappears-in-a-wave-of-populism-20190620-p51zhj

In my opinion, that - and further - defunding is entirely appropriate. We know from (i) to (v) above that high-profile Go8 promises of research "excellence" are a sham. When the University the Sydney defends clearly faulty papers as flawless, all Go8 research is devalued. Until the Go8 *reintroduces* effective quality control when it matters – so policymakers and the rest of us can again trust its research "findings" - the best level of taxpayer funding for Go8 research is zero.

As a menace to public health, the University of Sydney's Charles Perkins Centre should be defunded immediately. Most obviously, the NHMRC should withdraw the $13m of 2019-2023 funding from Professor Simpson's mouse-science group.

Some victims of the Charles Perkins' research misconduct are surprising. Notably, the entity supporting the *Australian Paradox* sugar-and-obesity fraud and the low-protein, high-carb lifespan fraud also is Qantas's main scientific advisor on nutrition. **Awkwardly, Qantas CEO Alan Joyce may have been misled and many Australians who fly are affected: "The centre's research has already influenced what meals and beverages we'll be serving onboard...**" (p. 9).

Finally, Rebecca, Michael, Duncan and Richard, I note that **ethical failures** of leaders of organisations across the world have seen them removed from their posts. I hope that each of you as University of Sydney and Go8 leaders will find a way to do what is right, to correct the ethical failures documented on your watch. It is no longer sufficient to pretend there is no problem. Taxpayers, journalists and politicians are starting to see the problem, and may no longer tolerate inaction.

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[Appendix: Information relating to Charles Perkins Centre's research misconduct and harm to public health]

THE UNIVERSITY OF SYDNEY

**Rebecca Halligan**
Director, Research Integrity & Ethics Administration

9 May 2019

Mr Rory Robertson

By email: strathburnstation@gmail.com

**PRIVATE & CONFIDENTIAL**

Dear Mr Robertson

**Confidential: Concerns with 2014 Cell Metabolism paper**

I am writing to acknowledge the concerns you have raised regarding the publication '*The Ratio of Macronutrients, Not Caloric Intake, Dictates Cardiometabolic Health, Aging, and Longevity in Ad Libitum-Fed Mice*,' Cell Metabolism (2014), 19, 418-430 (the "**2014 Cell Metabolism paper**") by researchers at the University of Sydney. Your concerns were brought to the attention of the National Health and Medical Research Council (NHMRC), who subsequently asked the University to consider the issues raised.

I understand that you have raised concerns regarding the representation of results in the 2014 Cell Metabolism paper and the communication of the paper's findings to the general public. As these matters fall within the scope of the University's *Research Code of Conduct 2013* and the *Australian Code for the Responsible Conduct of Research 2007* (copies of which are attached), these concerns will be assessed in accordance with these policies.

I will provide a further update when it is available. In the meantime, please treat this email as confidential.

Yours sincerely,

**Dr Rebecca Halligan**
Director, Research Integrity & Ethics Administration

Attachments: University Research Code of Conduct 2013
Australian Code for the Responsible Conduct of Research 2007

**Here is the 2014 paper and highly relevant information, including some hidden in "Supplemental information"**

Cell Metabolism
Article

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

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https://www.cell.com/action/showPdf?pii=S1550-4131%2814%2900065-5

This faulty paper is one of the highest-profile papers ever written in Australia. The University of Sydney promoted it in full-page advertisements in weekend newspapers (p. 22). The authors' false mouse-diet claims quickly became harmful diet advice for humans (pp. 20-22), and used to justify public funding of misguided mouse-diet research into dementia (p. 31).

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 ~150 dead mice) were buried in the Supplemental material (below).

| Diet | | 1 | 2[a] | 3[b] | 4 | 5 | 6[a] | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| %P | | 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 |
| Low 8 kJ g$^{-1}$ | P | 5.03 | 0.42 | 0.42 | 2.77 | 2.77 | 0.42 | 1.17 | 1.17 | 3.52 | 1.93 |
| | C | 1.67 | 0.28 | 1.67 | 4.02 | 1.67 | 4.02 | 2.43 | 4.77 | 2.43 | 3.18 |
| | F | 1.67 | 1.67 | 6.25 | 1.67 | 4.02 | 4.02 | 4.77 | 2.43 | 2.43 | 3.18 |
| Medium 13 kJ g$^{-1}$ | P | 7.54 | 0.63 | 0.63 | 4.15 | 4.15 | 0.63 | 1.76 | 1.76 | 5.28 | 2.89 |
| | C | 2.51 | 9.41 | 2.51 | 6.02 | 2.51 | 6.02 | 3.64 | 7.15 | 3.64 | 4.77 |
| | F | 2.51 | 2.51 | 9.41 | 2.51 | 6.02 | 6.02 | 7.15 | 3.64 | 3.64 | 4.77 |
| High 17 kJ g$^{-1}$ | P | 10.06 | 0.84 | 0.84 | 5.53 | 5.53 | 0.84 | 2.35 | 2.35 | 7.04 | 3.86 |
| | C | 3.35 | 12.55 | 3.35 | 8.03 | 3.35 | 8.03 | 4.85 | 9.54 | 4.85 | 6.36 |
| | 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,

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

**Here is the first media report discussing the University of Sydney's current research-integrity Investigation:**

*Honi Soit*

NEWS CULTURE FEATURES INVESTIGATION ANALYSIS PERSPECTIVE OPINION

News //

# Peak medical research body asks USyd to investigate concerns

Rory Robertson is back, and he's raised concerns over another study from the Charles Perkins Centre

by Alan Zheng

June 15, 2019


Left: A full-page advertisement displaying the findings of the 2014 mouse diet study in the Sydney Morning Herald last December.

Former Reserve Bank Economist and well-known anti-sugar campaigner Rory Robertson — who first exposed fundamental issues with the 2011 *Australian Paradox* study — is back, this time avowing concerns towards a 2014 Charles Perkins Centre (CPC) study and Australia's peak medical research body has asked the University of

http://honisoit.com/2019/06/peak-medical-research-body-asks-usyd-to-investigate-concerns-2/

## Charles Perkins Centre careerists dupe ABC then insist "...Rory's concerns are in every respect unfounded"

In response to my hard-hitting *Expression of Concern* about the blatant misrepresentation of median-lifespan results in his 2014 paper, Professor Stephen Simpson insisted "...Rory's concerns are in every respect unfounded". Yet the long-planned mouse experiment involved 30 diets, not 25, the latter figure falsely promoted by influential science careerists David Le Couteur and Steve Simpson, in the process of misinforming Sarah Dingle, Norman Swan and their nationwide ABC audiences. Table 3 and the media reports below confirm the authors' deceptions: in fact, five low-protein (LPHC) diets worked to maximise early death in mice, not lifespan, while five of the seven top diets for median lifespan are HPLC.

***Sarah Dingle****: From the Atkins diet to the Paleo craze, we've been encouraged to ditch carbohydrates in favour of protein - often protein derived from meat. But now a three year study … has found if you plan on living to a ripe old age, that could be dangerous advice. …Professor Le Couteur and his team* ***put their mice on 25 [not 30] different diets****…*

***David Le Couteur****:* ***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****, the highest amount of carbohydrate, so 60, 70, 75 per cent carbohydrate…*
https://www.abc.net.au/radio/programs/am/time-to-put-down-the-shake-study-warns-high/5299324

***Norman Swan****: Hello and welcome to the Health Report with me, Norman Swan. … One of the study's leaders was Professor Steve Simpson, who's director of the Charles Perkins Centre at the University of Sydney.*

***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 did was design 25 [not 30] diets*** *that spanned 10 different ratios of protein to fat to carbohydrate… the longest living mice … were those that had throughout their lives a relatively low protein content in their diet, coupled with a relatively high carbohydrate content...*
https://www.abc.net.au/radionational/programs/healthreport/high-protein2c-low-carbohydrate-diet/5309616#transcript

Also on research misconduct as defined below, we have Professor Simpson protecting Professor Jennie Brand-Miller's infamous *Australian Paradox* sugar-and obesity fraud, and indeed assisting her in 2017 to dishonestly expand the fraud into the *American Journal of Clinical Nutrition*. I say dishonestly because Simpson and Brand-Miller knowingly thwarted research-integrity investigator Professor Robert Clark AO's recommendation that she publish a new paper under Faculty supervision that "**specifically addresses and clarifies key factual matters**" including the unreliable data from her 2014 misconduct investigation. My evidence: pp. 5-6 in https://www.australianparadox.com/pdf/USyd-Misconduct-June19.pdf

**20 Definition of research misconduct**

(1) Research misconduct is a serious breach of this policy which is also:
- (a) intentional;
- (b) reckless; or
- (c) negligent.

(2) Examples of conduct which may amount to research misconduct include any of the following on the part of a researcher:
- (a) fabrication, falsification, or deception in proposing, carrying out or reporting the results of research;
- (b) plagiarism in proposing, carrying out or reporting the results of research;
- (c) failure to declare or manage a serious conflict of interests;
- (d) avoidable failure to follow research proposals as approved by a research ethics committee, particularly where this failure may result in unreasonable risk to humans, animals or the environment, or breach of privacy;
- (e) wilful concealment or facilitation of research misconduct by others;
- (f) misleading attribution of authorship;
- (g) intentional, unauthorised taking, sequestration or material damage to any research-related property of another;
- (h) deliberate conduct of research without required human ethics committee approval;
- (i) conduct of research involving animals without required animal ethics committee approval;
- (j) risking the safety of human participants or the wellbeing of animals or the environment; and
- (k) deviations from this policy which occur through gross or persistent negligence.

(3) Repeated or continuing breaches of this policy may also constitute research misconduct, and will do so where these have been the subject of previous counselling or specific direction.

(4) Research misconduct does not include honest differences in judgement, and may not include honest errors that are minor or unintentional. Unintentional errors do not usually constitute research misconduct unless they result from behaviour that is reckless or negligent.

p. 24 of 33 https://sydney.edu.au/policies/showdoc.aspx?recnum=PDOC2013/321&RendNum=0

# 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 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."

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

RELATED STORY: Cold shower may be secret to burning fat

RELATED STORY: Food industry likened to big tobacco in war on sugar

RELATED STORY: Obesity in developing countries growing at alarming rate

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

AUDIO: Listen to Professor David Le Couteur (AM)

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

**Epic fail in University of Sydney's quality control: False mouse-diet claims promoted as "research excellence"**


Source: *The Sydney Morning Herald*, 15 December 2018

## 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

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
While it's widely known that standard lab mice get fat and sick on low-carb diets, Professor Stephen Simpson – Academic Director of the 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 unfamiliar 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-carb, high-fat diets typically become fat and sick - via insulin resistance - whereas humans tend to thrive.

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Nutr Metab (Lond). 2012; 9: 69.
Published online 2012 Jul 28. doi: [10.1186/1743-7075-9-69]
PMCID: PMC3488544
PMID: 22838969

### Response of C57Bl/6 mice to a carbohydrate-free diet

Saihan Borghjid[1,2] and Richard David Feinman[2]

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This article has been cited by other articles in PMC.

Abstract Go to:

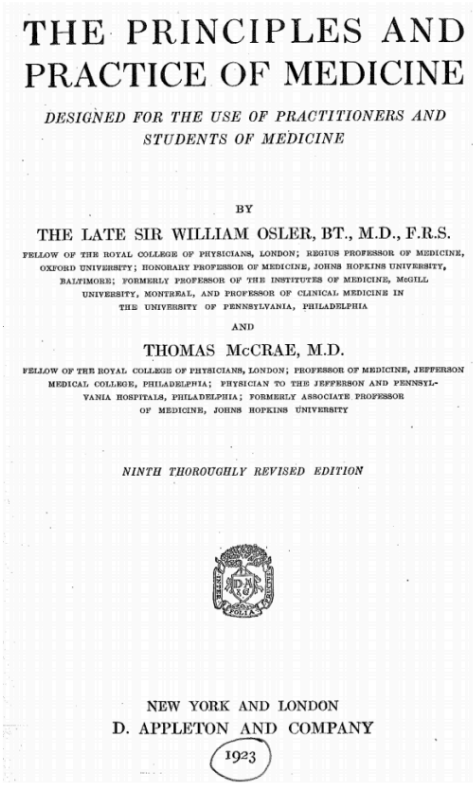
High fat feeding in rodents generally leads to obesity and insulin resistance whereas in humans this is only seen if dietary carbohydrate is also high, the result of the anabolic effect of poor regulation of glucose and insulin. A previous study of C57Bl/6 mice (Kennedy AR, et al.: *Am J Physiol Endocrinol Metab* (2007) **262** E1724-1739) appeared to show the kind of beneficial effects of calorie restriction that is seen in humans but that diet was unusually low in protein (5%). In the current study, we tested a zero-carbohydrate diet that had a higher protein content (20%). Mice on the zero-carbohydrate diet, despite similar caloric intake, consistently gained more weight than animals consuming standard chow, attaining a dramatic difference by week 16 (46.1 ± 1.38 g vs. 30.4 ± 1.00 g for the chow group). Consistent with the obese phenotype, experimental mice had fatty livers and hearts as well as large fat deposits in the abdomino-pelvic cavity, and showed impaired glucose clearance after intraperitoneal injection. In sum, the response of mice to a carbohydrate-free diet was greater weight gain and metabolic disruptions in distinction to the response in humans where low carbohydrate diets cause greater weight loss than isocaloric controls. The results suggest that rodent models of obesity may be most valuable in the understanding of how metabolic mechanisms can work in ways different from the effect in humans.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3488544/ ; https://www.ncbi.nlm.nih.gov/pubmed/16288655

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 p. 18 with the sugary diet harming humans on p. 29.

The following pages tell a tragic story of Group of Eight university science gone wrong.

**The tragedy of modern nutrition "science" and advice is that incompetence and scientific fraud have resulted in "scientists", GPs and dietitians knowing less today about fixing type 2 diabetes than was widely known in 1923**



THE PRINCIPLES AND PRACTICE OF MEDICINE

*DESIGNED FOR THE USE OF PRACTITIONERS AND STUDENTS OF MEDICINE*

BY

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*NINTH THOROUGHLY REVISED EDITION*

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1923

The following are the conditions which influence the appearance of sugar in the urine:

(*a*) EXCESS OF CARBOHYDRATE INTAKE.—In a normal state the sugar in the blood is about 0.1 per cent. In diabetes the percentage is usually from 0.2 to 0.4 per cent. The hyperglycæmia is immediately manifested by the appearance of sugar in the urine. The healthy person has a definite limit of carbohydrate assimilation; the total storage capacity for glycogen is estimated at about 300 gms. Following the ingestion of enormous amounts of carbohydrates the liver and the muscles may not be equal to the task of storing it; the blood content of sugar passes beyond the normal limit and the renal cells immediately begin to get rid of the surplus. Like the balance at the Mint, which is sensitive to the correct weight of the gold coins passing over it, they only react at a certain point of saturation. Fortunately excessive quantities of pure sugar itself are not taken. The carbohydrates are chiefly in the form of starch, the digestion and absorption of which take place slowly, so that this so-called alimentary glycosuria very rarely occurs, though enormous quantities may be taken. The assimilation limit of a normal fasting individual for sugar itself is about 250 gms. of grape sugar, and considerably less of cane and milk sugar. Clinically one meets with many cases in which glycosuria is present as a result of excessive ingestion of carbohydrates, particularly in stout persons and heavy feeders—so-called lipogenic diabetes—a form very readily controlled.

https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf

**Added sugar is 100% carbohydrate**. In 1923, it was widely known by competent GPs across the western world that excessive consumption of added sugar and other carbohydrate is the main driver of (Type 2) diabetes. **Accordingly, a low-carbohydrate, high-fat (LCHF) cure was advised (overleaf).** Today, that LCHF diet cure is almost universally suppressed by "scientists", GPs, dietitians and other public-health careerists. Sadly, the fledgling post-WW2 nutrition "science" space in the 1950s and 1960s was hijacked by mistaken-but-highly influential anti-fat, pro-carbohydrate careerists. For type 2 diabetics today, official advice is worse than useless: "usual care" typically features a diet of 45-65% carbohydrate and a lifetime on ineffective diabetes drugs. With usual care, typically less than 1% of HCPs' customers have their type 2 diabetes "reversed", "cured" or "put into remission" before their untimely, premature deaths.
http://care.diabetesjournals.org/content/early/2014/09/12/dc14-0874.full-text.pdf

https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf

**All sorted a century ago!**

**Pre-eminent medical text in 1923 advised no-sugar, low-carb treatment to cure "lipogenic" (type 2) diabetes**



**QUANTITY OF FOOD** Required by a Severe Diabetic Patient Weighing 60 kilograms. (Joslin.)

| Food | Quantity Grams | Calories per Gram | Total Calories |
|---|---|---|---|
| Carbohydrate | 10 | 4 | 40 |
| Protein | 75 | 4 | 300 |
| Fat | 150 | 9 | 1,350 |
| Alcohol | 15 | 7 | 105 |
| | | | 1,795 |

**STRICT DIET.** (Foods without sugar.) Meats, Poultry, Game, Fish, Clear Soups, Gelatine, Eggs, Butter, Olive Oil, Coffee, Tea and Cracked Cocoa.

**FOODS ARRANGED APPROXIMATELY ACCORDING TO CONTENT OF CARBOHYDRATES**

| | 5% + — | 10% + — | 15% + — | 20% + — |
|---|---|---|---|---|
| VEGETABLES | Lettuce, Spinach, Sauerkraut, String Beans, Celery, Asparagus, Cucumbers, Brussels Sprouts, Sorrel, Endive, Dandelion Greens, Swiss Chard, Vegetable Marrow, Cauliflower, Tomatoes, Rhubarb, Egg Plant, Leeks, Beet Greens, Water Cress, Cabbage, Radishes, Pumpkin, Kohl-Rabi, Sea Kale | Onions, Squash, Turnip, Carrots, Okra, Mushrooms, Beets | Green Peas, Artichokes, Parsnips, Canned Lima Beans | Potatoes, Shell Beans, Baked Beans, Green Corn, Boiled Rice, Boiled Macaroni |
| FRUITS | Ripe Olives (20 per cent. fat), Grape Fruit | Lemons, Oranges, Cranberries, Strawberries, Blackberries, Gooseberries, Peaches, Pineapples, Watermelon | Apples, Pears, Apricots, Blueberries, Cherries, Currants, Raspberries, Huckleberries | Plums, Bananas |
| NUTS | Butternuts, Pignolias | Brazil Nuts, Black Walnuts, Hickory, Pecans, Filberts | Almonds, Walnuts (Eng.), Beechnuts, Pistachios, Pine Nuts | Peanuts; 40% Chestnuts |
| Miscellaneous | Unsweetened and Unspiced Pickle, Clams, Scallops, Fish Roe, Oysters, Liver | | | |

| 30 grams (1 oz.) CONTAIN APPROXIMATELY | Protein | Fat | Carbohydrates GRAMS | Calories |
|---|---|---|---|---|
| Oatmeal | 5 | 2 | 20 | 110 |
| Meat (uncooked) | 6 | 2 | 0 | 40 |
| " (cooked) | 8 | 3 | 0 | 60 |
| Potato | 1 | 0 | 6 | 25 |
| Bacon | 5 | 15 | 0 | 155 |
| Cream, 40% | 1 | 12 | 1 | 120 |
| " 20% | 1 | 6 | 1 | 60 |
| Milk | 1 | 1 | 2 | 20 |
| Bread | 3 | 0 | 18 | 90 |
| Rice | 3 | 0 | 24 | 110 |
| Butter | 0 | 25 | 0 | 240 |
| Egg (one) | 6 | 5 | 0 | 75 |
| Brazil Nuts | 5 | 20 | 2 | 210 |
| Orange (one) | 0 | 0 | 10 | 40 |
| Grape Fruit (one) | 0 | 0 | 10 | 40 |
| Vegetables from 5-6% groups | 0.5 | 0 | 1 | 6 |

1 gram protein contains 4 calories.
1 " carbohydrate contains 4 calories.
1 " fat contains 9 calories.
1 " alcohol contains 7 calories.

1 kilogram—2.2 pounds.
6.25 grams protein contain 1 gram nitrogen.
A patient "at rest" requires 30 calories per kilogram body weight.

CHART XIV.—DIABETIC FOOD TABLES. (JOSLIN.)

https://www.australianparadox.com/pdf/1923-Medicine-Textbook.pdf
http://care.diabetesjournals.org/content/early/2014/09/12/dc14-0874.full-text.pdf

Source: RR's *Submission to ACCC's Scamwatch*

**Society increasingly aware that modern doses of added sugar cause obesity, type 2 diabetes and heart disease**

# Indigenous Affairs Minister Nigel Scullion says sugary soft drinks 'killing the population' in remote communities

By political reporter Anna Henderson
Posted 12 Feb 2016, 2:07pm

**In the wake of this week's progress report on Closing the Gap, the Indigenous Affairs Minister Nigel Scullion has declared sugary soft drinks are "killing the population" in remote Indigenous communities.**

According to evidence provided to Senate estimates today, at least 1.1 million litres of so-called "full sugar" soft drink was sold in remote community stores last financial year.

"I think particularly in remote communities and very remote communities sugar is just killing the population," Senator Scullion said.

"[It's] putting them into that very high risk area before they get to an age where those chronic diseases are evident."

Today's figures were provided by Outback Stores, which runs 36 small supermarkets in remote Aboriginal communities.

The company's chief executive Steven Moore told the committee the figures for soft drink sales are "astounding".

"I think we can all agree that poor diet in communities with consumption of fat, salt and sugar has a large impact on life expectancy in communities," he said.

"Full sugar soft drinks are a major contributor."

PHOTO: The Closing the Gap report said the worst health outcomes, in terms of diabetes, heart disease and other chronic illnesses were found in remote communities. (News Video)

RELATED STORY: Indigenous leaders respond to Closing the Gap

RELATED STORY: Indigenous life expectancy has not improved, Closing the Gap report shows

Key points:

- Closing the Gap report found worst health outcomes found in remote communities
- One remote community store drawing half of total profits from soft drink sales, Senator Scullion says
- Senator Scullion says he thinks attitudes to soft drink are changing

The Closing the Gap report from the Federal Government earlier this week found little progress towards bridging the life expectancy gap between Indigenous and non-Indigenous Australians.

It said the worst health outcomes, in terms of diabetes, heart disease and other chronic illnesses were found in remote communities.

http://www.abc.net.au/news/2016-02-12/scullion-says-sugar-is-killing-remote-communities/7162974

HEART & STROKE™ FOUNDATION

POSITION STATEMENT

# SUGAR, HEART DISEASE AND STROKE

## FACTS

- Heart disease and stroke are leading causes of death in Canada, responsible for 27.3% of all deaths.[1] Over 1.3 million Canadians are living with heart disease[2] and 315,000 Canadians are living with the effects of stroke.[3]
- More than 60% of Canadian adults[4] and 31% of children and youth aged 5 to 17 years are overweight or obese.[5] Children who are obese are at increased risk of remaining overweight or obese as adults.[6]
- Up to 80% of early heart disease and stroke can be prevented through adopting healthy behaviours including eating a healthy diet.
- Sugar is a carbohydrate that provides energy to the body. Other than providing energy, sugar has no other nutritional benefits.
- Sugar can occur naturally in milk, fruit, vegetables, starches, grains and most plant based foods. Sugars can also be added to foods and drinks for flavour, as a sweetener, as a
- Excess sugar consumption is associated with adverse health effects including heart disease,[10-12] stroke,[10] obesity,[13-17] diabetes,[18-22] high blood cholesterol,[23-24] cancer[25] and dental caries (cavities).[26]
- Individuals who consume greater than or equal to 10% but less than 25% of total energy (calories) from added sugar have a 30% higher risk of death from heart disease or stroke when compared to those who consume less than 10%. For those who consume 25% or more of calories from added sugar, the risk is nearly tripled.[10]

https://www.heartandstroke.ca/-/media/pdf-files/canada/2017-position-statements/sugar-ps-eng.ashx

https://www.australianparadox.com/pdf/Letter-to-ACCC.pdf

news.com.au

AAP NOVEMBER 20, 2013 9:45PM

# 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
https://www.news.com.au/national/breaking-news/prof-uses-1000-mice-to-expose-food-folly/news-story/403238e7cccc57b86b689aaa18fa4b95

**Indigenous Australians are perhaps hardest hit by the Charles Perkins Centre's pro-sugar incompetence and fraud. It's tragic that the sorts of outsiders Charlie worked so hard to help often live in misery and die prematurely via type 2 diabetes and CVD, driven by excess consumption of sugar and other carbohydrate**

# Characteristics of the community-level diet of Aboriginal people in remote northern Australia

**Julie K Brimblecombe**
GradDipNut&Diet, MPH, PhD,
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BSc, GradDipNut&Diet, MPH,
Senior Research Officer,[1] and PhD Candidate[1,2]

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BSc, PhD,
Professor, Population Health and Nutrition,[3] and Honorary Professor[4]

1 Wellbeing and Preventable Chronic Disease, Menzies School of Health Research, Darwin, NT.
2 Institute of Advanced Studies, Charles Darwin University, Darwin, NT.
3 School of Population Health, Division of Health Sciences, University of South Australia, Adelaide, SA.
4 Menzies School of Health Research,

## 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.

**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.[1] 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.[2] The 26% of Indigenous Australians living in remote areas experience 40% of the health gap of Indigenous Australians overall.[3] Much of this burden of disease is due to extremely poor nutrition throughout life.[4]

Comprehensive dietary data for Indigenous Australians are not available from national nutrition surveys or any other source. Previous reports on purchased food in remote Aboriginal communities are either dated,[5] limited to the primary store[5,6] and/or short-term or cross-sectional in design.[7,8] These studies have consistently reported low intake of fruit and vegetables, high intake of refined cereals and sugars, excessive

was prohibited in the three study communities at the time of our study.

Monthly electronic food (and non-alcoholic beverage) transaction data

egorised into food groups derived from the Australian Food and Nutrient Database AUSNUT 07 food grouping system[10] and beverages were further

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

**4727.0.55.003 - Australian Aboriginal and Torres Strait Islander Health Survey: Biomedical Results, 2012-13**

**LATEST ISSUE** Released at 11:30 AM (CANBERRA TIME) 10/09/2014 **First Issue**

Summary | Downloads | Explanatory Notes | Related Information | Past & Future Releases

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- + Key Findings
- + Diabetes
- + Cardiovascular disease
- Chronic Kidney Disease
- Liver Function
- Exposure to tobacco smoke
- Anaemia
- Iodine
- Vitamin D
- Feature article: Chronic disease results for Aboriginal and Torres Strait Islander and non-Indigenous Australians
- **Aboriginal and Torres Strait Islander adults experience diabetes 20 years earlier than non-Indigenous adults (Media Release)**
- About this Release
- History of Changes

**MEDIA RELEASE**

10 September 2014 | Embargo: 11:30 am (Canberra Time) | 132/2014

### 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 Jelfs 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 Jelfs.

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 Jelfs.

"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."

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%20200%20years%20earlier%20than%20non-Indigenous%20adults%20(Media%20Release)~130

https://www.australianparadox.com/pdf/Letter-to-ACCC.pdf

**The mobs Charlie Perkins cared about struggle and die early in droves on low-protein, 60%-carb mouse diet**

MJA 100 YEARS The Medical Journal of Australia ·1914-2014

Advanced Search

Research 13.

# Characteristics of the community-level diet of Aboriginal people in remote northern Australia

Julie K Brimblecombe, Megan M Ferguson, Selma C Liberato and Kerin O'Dea

Med J Aust 2013; 198 (7): 380-384. doi: 10.5694/mja12.11407

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**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.[1] 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.[2] The 26% of Indigenous Australians living in remote areas experience 40% of the health gap of Indigenous Australians overall.[3] Much of this burden of disease is due to extremely poor nutrition throughout life.[4]

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

| 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) | 60.7% (0.8) |
| Sugars | 34.3% (0.8) | 28.9% (2.2) | 25.7% (1.8) | 33.4% (0.7) |

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**



Australian Bureau of Statistics

# 4727.0.55.003 - Australian Aboriginal and Torres Strait Islander Health Survey: Biomedical Results, 2012-13

Latest ISSUE Released at 11:30 AM (CANBERRA TIME) 10/09/2014 First Issue

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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 Jelfs.

"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."

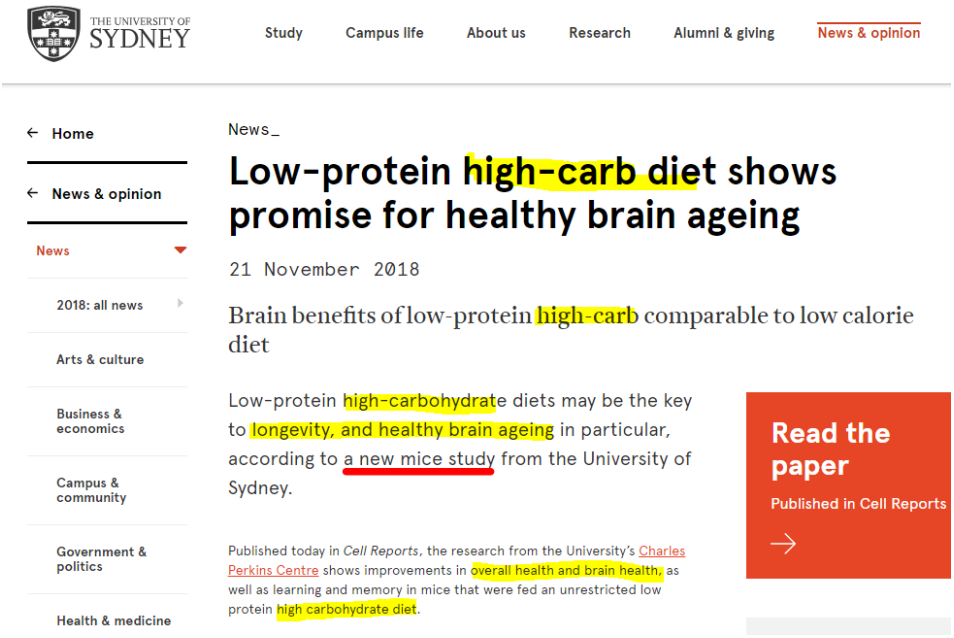
Other results released today suggest that many Aboriginal and Torres Strait Islander adults may not be aware they have high cholesterol, with one in four having high cholesterol levels, yet only one in ten being aware they had it.

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%2020%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**



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# Low-protein high-carb diet shows promise for healthy brain ageing

21 November 2018

Brain benefits of low-protein high-carb comparable to low calorie diet

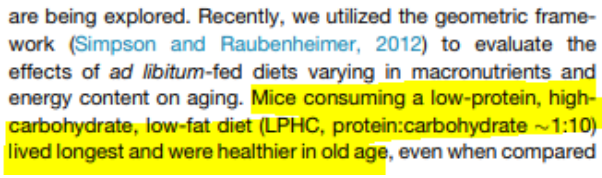
Low-protein high-carbohydrate diets may be the key to longevity, and healthy brain ageing in particular, according to a new mice study from the University of Sydney.

Published today in *Cell Reports*, the research from the University's Charles Perkins Centre shows improvements in overall health and brain health, as well as learning and memory in mice that were fed an unrestricted low protein high carbohydrate diet.

**Read the paper**

Published in Cell Reports

https://sydney.edu.au/news-opinion/news/2018/11/21/low-protein-high-carb-diet-shows-promise-for-healthy-brain-agein.html



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, high-carbohydrate, low-fat diet (LPHC, protein:carbohydrate ~1:10) lived longest and were healthier in old age, even when compared

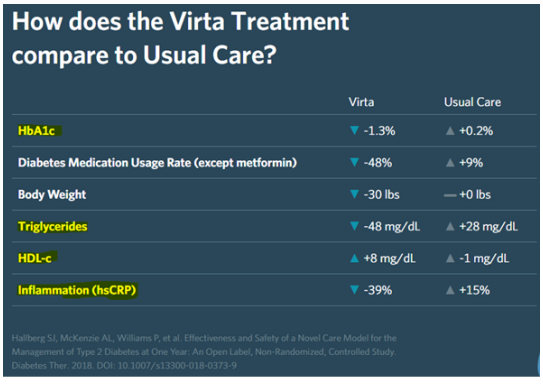
p. 2 https://www.cell.com/cell-reports/pdf/S2211-1247(18)31674-7.pdf

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.

Diabetes Therapy

April 2018, Volume 9, Issue 2, pp 583–612 | Cite as

Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at 1 Year: An Open-Label, Non-Randomized, Controlled Study



**How does the Virta Treatment compare to Usual Care?**

| | Virta | Usual Care |
|---|---|---|
| HbA1c | ▼ -1.3% | ▲ +0.2% |
| Diabetes Medication Usage Rate (except metformin) | ▼ -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% |

Hallberg SJ, McKenzie AL, Williams P, et al. Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at One Year: An Open Label, Non-Randomized, Controlled Study. Diabetes Ther. 2018. DOI: 10.1007/s13300-018-0373-9

**Groundbreaking Clinical Outcomes**

Virta's landmark clinical trial demonstrated rapid type 2 diabetes reversal in as little as 10 weeks, with sustained and improved results at 1 year—all published in peer-reviewed scientific journals.

60% OF PATIENTS REVERSED THEIR TYPE 2 DIABETES

94% OF PATIENTS REDUCED OR ELIMINATED INSULIN

1.3% AVERAGE HBA1C REDUCTION AT ONE YEAR

30 lbs AVG WEIGHT LOSS AT ONE YEAR (12%)

83% CLINICAL TRIAL RETENTION AT ONE YEAR

Hallberg SJ, McKenzie AL, Williams P, et al. Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at One Year: An Open Label, Non-Randomized, Controlled Study. Diabetes Ther. 2018. DOI: 10.1007/s13300-018-0373-9

https://www.virtahealth.com/research ; https://link.springer.com/content/pdf/10.1007%2Fs13300-018-0373-9.pdf

Hard scientific evidence shows Low-carbohydrate, high-fat diet should be first approach to Type 2 diabetes

ELSEVIER

Nutrition

Volume 31, Issue 1, January 2015, Pages 1-13

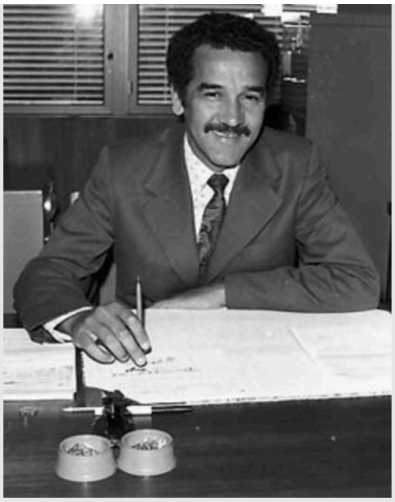
Critical review

# Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base

Richard D. Feinman Ph.D. [a] ✉, Wendy K. Pogozelski Ph.D. [b], Arne Astrup M.D. [c], Richard K. Bernstein M.D. [d], Eugene J. Fine M.S., M.D. [e], Eric C. Westman M.D., M.H.S. [f], Anthony Accurso M.D. [g], Lynda Frassetto M.D. [h], Barbara A. Gower Ph.D. [i], Samy I. McFarlane M.D. [j], Jörgen Vesti Nielsen M.D. [k], Thure Krarup M.D. [l], Laura Saslow Ph.D. [m], Karl S. Roth M.D. [n], Mary C. Vernon M.D. [o], Jeff S. Volek R.D., Ph.D. [p], Gilbert B. Wilshire M.D. [q], Annika Dahlqvist M.D. [r] ... Nicolai Worm Ph.D. [z]


**Fig. 9.** Comparison of low-glycemic index diet with high-cereal diet, and of low-glycemic index diet with low-carbohydrate diet. Data from [6,70]. Redrawn from [75]. CHO, carbohydrate; GI, glycemic index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride; Total-C, total cholesterol.

**What would Charlie think of what's being done under his name, if he hadn't died young, via kidney disease?**

*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

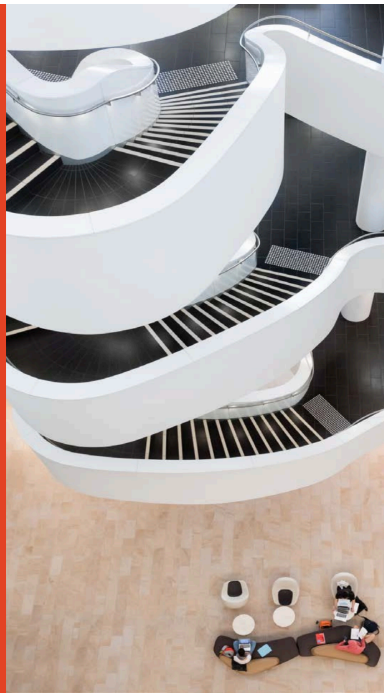
**Key Events**
- Freedom Ride, 1965

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

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

Stephen J. Simpson

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

## Dedication

Charlie Perkins was born in Alice Springs near the red centre of Australia in June 1936. I was born there 30 years later in March 1966. I dedicate my body of work exposing the Charles Perkins Centre's *Australian Paradox* sugar-and-obesity fraud and its low-protein, high-carbohydrate lifespan fraud to my mother, **Elaine Lucas**, who nursed Aboriginal and other Australians in remote places - including Katherine, Alice Springs, Balcanoona, Woorabinda and Baralaba - from the early 1960s to the late 1980s. And to my (late) father, **Alexander "Sandy" Robertson**, who grew up in Scotland and in the Scots Guards, shifted briefly to Melbourne then Coogee in Sydney, before working with cattle, sheep and wheat across country Australia for half a century. He taught me (and my brother and sister) much about what is right and much about what is wrong, often by example. (A longer piece on Dad's life and times can be found in one of the links below.)

I also have firmly in mind people like Bonita and Eddie Mabo, Faith Bandler, Charlie Perkins (who Dad often said he knew briefly - so too his brother Ernie - in The Territory over half a century ago), Waverley Stanley and Lou Mullins of Yalari, and especially Noel Pearson, all of whom worked or are working indefatigably for decades to improve the lot of their mobs, their peoples left behind. Finally, I wonder whatever happened to the many Aboriginal boys and girls I met across country Australia when I was a boy, especially the big Woorabinda mob with whom I shared classrooms and sports fields back in Baralaba, central Queensland, in the late 1970s. Much of the news over the years has been tragic and depressing. https://www.australianparadox.com/baralaba.htm

**Please note**: In this and other documents, I have detailed influential incompetence and worse in nutrition and health "science", and by Group of Eight university senior management. Importantly, if you read anything here or elsewhere from me that is factually incorrect or otherwise unreasonable, please contact me immediately and, if I agree, I will correct the text as soon as possible. This all matters because more than one million Australians today have type 2 diabetes, the number growing rapidly. Many of these vulnerable Australians can expect mistreatment, misery and early death, harmed by high-carbohydrate diabetes advice promoted by a range of respected entities advised by highly influential Group of Eight science careerists. The unfolding diabetes tragedy can be seen most clearly in the quiet suffering of short-lived Indigenous Australians.


## Rory Robertson
**economist and former-fattie**
**https://twitter.com/OzParadoxdotcom**

**+61 414 703 471**
strathburnstation@gmail.com


**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

**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** http://www.australianparadox.com/pdf/Big-5-year-update-Feb-2017.pdf

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

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

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

**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**