Committee Secretariat
PO Box 6021
Parliament House
CANBERRA
Canberra ACT 2600
+61 2 6277 4145
Health.Reps@aph.gov.au

To the Honourable members of the Standing Committee on Health, Aged Care & Sport,

I write in response to your request for written submissions as you inquire into the state of the diabetes epidemic. My educational background includes a Bachelor of Medicine and Surgery, a Bachelor of Physiotherapy and a Master degree in Occupational Health. I have completed a 4-year full time fellowship via the Australasian College of Sports and Exercise Medicine, and am now an accredited practising Specialist Sports and Exercise Medicine Physician. I am a recognised expert in nutritional science, having been an invited speaker on nutrition at many medical conferences nationally and internationally. I also act as an expert peer reviewer for submitted journal articles relating to nutrition.

I currently hold the role of Chief Medical Officer for Defeat Diabetes, a science-based program providing nutritional support for those with and at risk of type 2 diabetes. I am also the invited lead author of an academic chapter on nutrition, Nutrition for health[1], published in an eminent sports medicine textbook. This academic chapter challenges several long-established nutritional conventions. The book chapter is extensively referenced with 306 peer reviewed citations and underwent meticulous analysis prior to publication. I am also co-author on a recent extensively referenced paper which reviews the health impacts of low carbohydrate diets, including with respect to lipid changes and diabetes risk[2]. This paper has been well received, its Altmetric score demonstrating a level of interest placing it in the top 1% of published articles.

My submission covers the five elements listed in the five areas of your investigation. I am available at your convenience to respond to any questions that you might have, and thank you for your efforts to address this enormous challenge.

Sincerely,

Dr Paul Mason

MBBS (Honours) University of Sydney
B. Physio (Latrobe University)
Masters Occ. Health (Latrobe University)
Fellow Australasian College Sport and Exercise Physicians
Orthosports

[1] Brukner & Khan's Clinical Sports Medicine Volume 2: The medicine of exercise, Fifth Edition, Peter Brukner, Karim Khan, McGraw-Hill Education Australia, 2019.

[2] Diamond, David M.a; Bikman, Benjamin T.b; Mason, Paulc. Statin therapy is not warranted for a person with high LDL-cholesterol on a low-carbohydrate diet. Current Opinion in Endocrinology & Diabetes and Obesity 29(5):p 497-511, October 2022. | DOI: 10.1097/MED.0000000000000764

Executive summary

1. The causes of diabetes (type 1, type 2 and gestational) in Australia, including risk factors such as genetics, family history, age, physical inactivity, other medical conditions and medications used

In Australia, approximately 85% of diabetes cases are type 2, and diet plays a pivotal role in its development and management. The Australian dietary guidelines are influential and likely have contributed to an increased burden of diabetes. They influence diets in schools, aged care facilities, hospitals, and the general public. Despite technically not applying to those with specific illness, in practice the dietary guidelines influence the diets of millions of Australians who are not in optimal metabolic health.

Understanding type 2 diabetes as a condition rooted in insulin resistance is crucial. Dietary sugars, particularly fructose-containing sugars, have been conclusively linked to insulin resistance, and therefore diabetes. Restricting fructose intake can has been proven to result in significant improvements in insulin resistance and liver fat in periods as short at 9 days. High carbohydrate diets, as recommended by the dietary guidelines, worsen blood sugar control once insulin resistance has developed. All carbohydrates break down into glucose, which cannot be effectively removed from the blood in an insulin resistant state, resulting in elevated blood sugar levels and the subsequent medical complications of diabetes.

A significant issue with the Australian Dietary Guidelines is the failure to recommend the restriction of both added sugars and those naturally present in foods, despite causing identical metabolic harms. That the guidelines promote starchy high-carbohydrate foods further compounds the problem.

Low carbohydrate diets, despite not aligning with the Australian Dietary Guidelines, have shown numerous benefits in managing diabetes. For example, more than 50% of diabetic individuals needing insulin were able to discontinue it during a five-year prospective trial on low carbohydrate diets. A re-evaluation of these guidelines in light of the latest scientific evidence is crucial for improving public health outcomes related to diabetes.

2. New evidence-based advances in the prevention, diagnosis and management of diabetes, in Australia and internationally

Recent research has challenged the conventional belief that type 2 diabetes is an irreversible, progressive condition. A substantial body of evidence supports the effectiveness of low carbohydrate diets in managing and even achieving remission of type 2 diabetes. This research is extensive and consistent, with systematic reviews and meta-analyses serving as the gold standard for evaluating scientific evidence.

Significantly, 12 identified systematic reviews and meta-analyses focusing on therapeutic carbohydrate restriction or low carbohydrate diets in diabetes management all unanimously conclude the benefits of employing such diets. These findings underscore the importance of considering low carbohydrate approaches as a viable and effective strategy for managing type 2 diabetes.

3. The broader impacts of diabetes on Australia's health system and economy;

Type 2 diabetes is strongly linked to various other health conditions, and understanding this interconnectedness is crucial. Associated maladies include cardiovascular disease including atherosclerosis, hypertension, atherogenic dyslipidaemia, obesity, non-alcoholic fatty liver disease (NAFLD) and dementia, often referred to as "type 3 diabetes."

Insulin resistance plays a central role in the development of these conditions, having long been recognised as a key factor in the onset of cardiovascular disease. Insulin resistance contributes to atherosclerosis through multiple mechanisms including hypertension, hyperglycaemia, pro-thrombosis, endothelial dysfunction and impaired nitric oxide synthesis, all which collectively harm blood vessel structure and function. Insulin resistance is a primary driver of hypertension through various pathways, including sodium retention and sympathetic nervous system activation. Insulin resistance is also associated with serum lipid profiles known to increase cardiovascular disease risk.

The brain, which accounts for a small percentage of body mass but a significant amount of energy consumption, is particularly vulnerable to metabolic diseases. The close link between dementia and metabolic disease is evident from research showing that obesity increases dementia risk, and brain volume decreases with higher body mass index. Indeed, individuals with diabetes for over a decade by age 70 are at a doubled

risk of dementia. Compelling evidence now shows that low carbohydrate diets have the potential to reduce the risk of dementia and improve cognitive function. Significantly, they outperform pharmacological interventions.

Recognizing that type 2 diabetes exists as part of a broader constellation of metabolic diseases caused by insulin resistance, with diet as a major risk factor, emphasises the significant benefits achievable through clear dietary recommendations grounded in scientific literature. Improved dietary guidelines would predictably lead to substantial savings in healthcare costs related to diabetes, cardiovascular disease, and dementia, which collectively consume a significant portion of the healthcare budget. The current Australian Dietary Guidelines, which discourage the utilisation of low carbohydrate diets, is contributing both to unnecessary suffering and expenditure.

4. Any interrelated health issues between diabetes and obesity in Australia, including the relationship between type 2 and gestational diabetes and obesity, the causes of obesity and the evidence-base in the prevention, diagnosis and management of obesity; and

Metabolic syndrome encompasses a group of interconnected conditions, with insulin resistance as a major risk factor, including hypertension, diabetes, obesity, and atherogenic dyslipidaemia. A significant problem in current dietary advice for managing metabolic syndrome is the misrepresentation of study findings to support researchers' initial hypotheses, contrary to the scientific method.

While the dietary guidelines are purported to be based on scientific evidence, certain research publications have clearly misrepresented their findings. This includes three large-scale and influential randomized controlled trials. The Women's Health Initiative Randomized Controlled Dietary Modification Trial, the Sydney Diet Heart study, and the Minnesota Coronary Experiment. The Women's Health Initiative trial aimed to find evidence supporting saturated fat restriction but found that females with prior heart disease following a low-fat diet had a 26% higher risk of complications such as heart attack. However, this significant finding was omitted from the results and conclusion, being couched in vague language in a single sentence on page 661 of the publication.

The intervention in both the Sydney Diet Heart study and the Minnesota Coronary Experiment was to reduce saturated fat intake. Both found evidence of harm from this intervention. The Sydney Diet Heart study for example, found risk of death to be increased by 62% after reducing saturated fat intake. These findings however, were destined to never be published. The Minnesota Coronary Experiment's lead investigator is on record admitting his reluctance to publish was due to disappointment in the results. It was only after a chance discovery of the records for both studies led to their eventual publication after more than three decades. Notably, the current iteration of the dietary guidelines does not reflect the evidence from these studies, with detrimental consequences for public health.

5. The effectiveness of current Australian Government policies and programs to prevent, diagnose and manage diabetes.

The prevailing increase in type 2 diabetes and related diseases is evidence of the failure of current policies and programs. A significant contributor to this is the unscientific nature of dietary recommendations in the Australian Dietary Guidelines. These guidelines have long promoted high-carbohydrate, low-saturated fat diets based on the diet-heart hypothesis, which is not supported by contemporary research.

Intake of saturated fat continues to be discouraged primarily on the basis of concern regarding LDL cholesterol levels. This is despite clear evidence to the contrary. For example, a systematic review published in the British Medical Journal found that 16 of 19 prospective studies established that subjects with higher LDL levels lived longest, even when controlling for reverse causation. Importantly, influential experts are revising their views on dietary saturated fat, including those responsible for previous iterations of the American Dietary Guidelines. This shift has not yet reached the Australian Dietary Guidelines.

Another area of policy failure is the tacit approval of recommendations to limit the intake of red meat. There are several arguments made against red meat, the most prominent which is a putative link to colorectal cancer much of which stems back to an influential WHO report from 2015. Notably, this report only referenced 6 experimental studies before concluding red meat causes cancer. Three of these conducted on rats both fed red meat AND injected with cancer causing chemicals. Despite this, none of the rats developed cancer. While this sounds unbelievable, it is true. Discouraging the intake of red meat reduces a nutrient dense food while necessarily being replaced by less healthy foods. All food policy should be based on robust scientific evidence.

1. The causes of diabetes (type 1, type 2 and gestational) in Australia, including risk factors such as genetics, family history, age, physical inactivity, other medical conditions and medications used

The majority of cases of diabetes in Australia, ~85% as per the Australian Bureau of Statistics is type 2. While there are several risk factors contributing to the development of type 2 diabetes, the most significant of these is diet.

Importantly, the dietary pattern as recommended by the Australian dietary guidelines has been shown by research to contribute both to the development of, and worsening control of type 2 diabetes. For example, the earliest randomised controlled trial I have identified studying seed oil consumption[3] reported not only an increased risk of death with seed oils, but also describes in clear detail the development of glucose in the urine (glycosuria), a hallmark of diabetes, secondary to seed oil consumption. Cessation of the seed oil resulted in resolution of the glycosuria, only to return again when the seed oils were resumed.

Likewise, the Women's Health Initiative (WHI) study, a landmark research project (involving over 48,000 subjects and costing $700 million USD) found no benefit of reducing saturated fat intake and increasing seed oil intake with respect to diabetes.

Further, the understanding that type 2 diabetes is a disease underpinned by insulin resistance – whereby insulin is no longer effectively able to remove glucose from the blood – now has widespread acceptance[4]. This is critical to understanding the role of diet in causing type 2 diabetes. It cannot be overemphasised that consumption of dietary sugars has been conclusively demonstrated to contribute to insulin resistance, again, the major risk factor for type 2 diabetes.

One study for example, found that simply by that restricting the fructose containing sugar intake, including fruit juices, in young American subjects resulted in significant improvements in insulin resistance within just 9 days[5]. There was also an almost 50% reduction in liver fat, also strongly associated with type 2 diabetes, over this 9-day period.

The problems of high carbohydrate diets, including of starches such as pasta, breads (including wholemeal and wholegrain), cereals and root vegetables only compounds the problem once insulin resistance has developed. All carbohydrates are made of chains of glucose, the sugar in the blood which is elevated in diabetes. With insulin unable to remove this digested sugar effectively from the blood, as in the case of insulin resistance, blood sugar levels elevate, and the complications of diabetes ensue.

---

[3] ROSE GA, THOMSON WB, WILLIAMS RT. CORN OIL IN TREATMENT OF ISCHAEMIC HEART DISEASE. Br Med J. 1965 Jun 12;1(5449):1531-3. doi: 10.1136/bmj.1.5449.1531. PMID: 14288105; PMCID: PMC2166702.

[4] Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C. Pathophysiology of Type 2 Diabetes Mellitus. Int J Mol Sci. 2020 Aug 30;21(17):6275. doi: 10.3390/ijms21176275. PMID: 32872570; PMCID: PMC7503727.

[5] Schwarz JM, Noworolski SM, Erkin-Cakmak A, Korn NJ, Wen MJ, Tai VW, Jones GM, Palii SP, Velasco-Alin M, Pan K, Patterson BW, Gugliucci A, Lustig RH, Mulligan K. Effects of Dietary Fructose Restriction on Liver Fat, De Novo Lipogenesis, and Insulin Kinetics in Children With Obesity. Gastroenterology. 2017 Sep;153(3):743-752. doi: 10.1053/j.gastro.2017.05.043. Epub 2017 Jun 1. PMID: 28579536; PMCID: PMC5813289.

It is a significant problem that the Australian Dietary Guidelines do not treat all sugars in food equally, differentiating between added sugars and those naturally present in foods. The fact is these sugars are molecularly identical with identical deleterious impacts.

Equally problematic is that the Australian Dietary Guidelines promote the consumption of foods high in carbohydrates, proven to adversely impact blood glucose levels in those with insulin resistance which underpins metabolic disease. While technically the Australian Dietary Guidelines technically do not apply to those with specific medical issues, most of the population is metabolically unhealthy. For example, a national study on the US population, whom has similar levels of obesity and metabolic illness to Australia, found that only 12.2% of the adult population was metabolically healthy[6].

That these diets are problematic for diabetes has also been proven by numerous studies demonstrating the benefits of following low carbohydrate diets not allowed for by the Australian Dietary Guidelines. For example, a 5-year prospective trial on low carbohydrate diets found that more than 50% of diabetic subjects needing insulin were able to completely cease it[7].

It is difficult to overstate the barrier that the Australian Dietary Guidelines presents to both the prevention and management of type 2 diabetes. They inform the content of recommended diets in schools, aged care facilities and hospitals as well as diets consumed by the public.

[6] Araújo J, Cai J, Stevens J. Prevalence of Optimal Metabolic Health in American Adults: National Health and Nutrition Examination Survey 2009-2016. Metab Syndr Relat Disord. 2019 Feb;17(1):46-52. doi: 10.1089/met.2018.0105. Epub 2018 Nov 27. PMID: 30484738.

[7] https://www.virtahealth.com/press/virta-sustainable-health-improvements-5-year-diabetes-reversal-study

2. New evidence-based advances in the prevention, diagnosis and management of diabetes, in Australia and internationally

Until recently, type 2 diabetes was generally considered to be an irreversible, progressive disease. This understanding has been overturned in the face of an enormous volume of research that low carbohydrate diets consistently prove to be beneficial in the management of type 2 diabetes, including leading to its remission.

It is difficult to appreciate the full extent of this research as well as the consistency of findings. Systematic reviews and meta-analyses are methods of synthesising scientific evidence on a specific topic in an objective and methodological manner. Each systematic review or meta-analysis sets out to consider the entirety of the evidence base before establishing a conclusion. That both systematic reviews and meta-analyses can each evaluate multiple, sometimes hundreds, of studies both on their findings and underlying methodological quality (assessing for risk of bias resulting in unreliable findings), they are considered the highest level of evidence (1A). Both systematic reviews and meta-analyses provide clear and unambiguous support for strong clinical recommendations which clinicians should follow unless a clear rationale for an alternative approach is present[8]. This is a well-accepted principle in the field of evidence-based medicine.

It is therefore significant that I have identified 12 systematic reviews or meta-analyses covering therapeutic carbohydrate restriction, or low carbohydrate diets, in the management of diabetes [9] [10] [11]

[8] Burns PB, Rohrich RJ, Chung KC. The levels of evidence and their role in evidence-based medicine. Plast Reconstr Surg. 2011 Jul;128(1):305-310. doi: 10.1097/PRS.0b013e318219c171. PMID: 21701348; PMCID: PMC3124652.

[9] Li S, Ding L, Xiao X. Comparing the Efficacy and Safety of Low-Carbohydrate Diets with Low-Fat Diets for Type 2 Diabetes Mellitus Patients: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. International Journal of Endocrinology. 2021;2021:e8521756. doi:10.1155/2021/8521756

[10] Yuan X, Wang J, Yang S, et al. Effect of the ketogenic diet on glycemic control, insulin resistance, and lipid metabolism in patients with T2DM: a systematic review and meta-analysis. Nutrition & Diabetes. 2020;10(1):1-8. doi:10.1038/s41387-020-00142-z

[11] Meng Y, Bai H, Wang S, Li Z, Wang Q, Chen L. Efficacy of low carbohydrate diet for type 2 diabetes mellitus management: A systematic review and meta-analysis of randomized controlled trials. Diabetes Research and Clinical Practice. 2017;131:124-131. doi:10.1016/j.diabres.2017.07.006 ABSTRACT

[12] [13] [14] [15] [16] [17] [18] [19] [20]. That each of these reviews concludes benefit of utilising low carbohydrate diets in the management of diabetes ought not be ignored. I cannot place enough emphasis on this point.

[12] Alarim RA, Alasmre FA, Alotaibi HA, Alshehri MA, Hussain SA. Effects of the Ketogenic Diet on Glycemic Control in Diabetic Patients: Meta-Analysis of Clinical Trials. Cureus. 12(10). doi:10.7759/cureus.10796

[13] Turton J, Brinkworth GD, Field R, Parker H, Rooney K. An evidence-based approach to developing low-carbohydrate diets for type 2 diabetes management: a systematic review of interventions and methods. Diabetes, Obesity and Metabolism. doi:10.1111/dom.13837

[14] Snorgaard O, Poulsen GM, Andersen HK, Astrup A. Systematic review and meta-analysis of dietary carbohydrate restriction in patients with type 2 diabetes. BMJ Open Diabetes Res Care. 2017;5(1):e000354. doi:10.1136/bmjdrc-2016-000354

[15] Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr. 2013;97(3):505-516. doi:10.3945/ajcn.112.042457

[16] Goldenberg JZ, Day A, Brinkworth GD, et al. Efficacy and safety of low and very low carbohydrate diets for type 2 diabetes remission: systematic review and meta-analysis of published and unpublished randomized trial data. BMJ. 2021;372:m4743. doi:10.1136/bmj.m4743

[17] Nicholas AP, Mota AS, Lambert H, Collins AL. Restricting carbohydrates and calories in the treatment of type 2 diabetes: a systematic review of the effectiveness of 'low carbohydrate' interventions with differing energy levels. medRxiv. Published online May 14, 2021:2021.05.07.21256843. doi:10.1101/2021.05.07.21256843

[18] Nicholas AP, Soto-Mota A, Lambert H, Collins AL. Restricting carbohydrates and calories in the treatment of type 2 diabetes: a systematic review of the effectiveness of 'low-carbohydrate' interventions with differing energy levels. Journal of Nutritional Science. 2021;10. doi:10.1017/jns.2021.67

[19] Huntriss R, Campbell M, Bedwell C. The interpretation and effect of a low-carbohydrate diet in the management of type 2 diabetes: a systematic review and meta-analysis of randomised controlled trials. Eur J Clin Nutr. 2018;72(3):311-325. doi:10.1038/s41430-017-0019-4 ABSTRACT

[20] Valenzuela Mencía J, Fernández Castillo R, Martos Cabrera MB, Gómez-Urquiza JL, Albendín García L, Cañadas de la Fuente GA. Diets low in carbohydrates for type 2 diabetics. Systematic review. Nutr Hosp. 2017;34(1):224-234. doi:10.20960/nh.999 ABSTRACT

3. The broader impacts of diabetes on Australia's health system and economy;

The full impact of type 2 diabetes can only be fully appreciated with the understanding that type 2 diabetes causally co-exists with numerous other maladies. This extensive list includes cardiovascular disease secondary to atherosclerosis, hypertension, atherogenic dyslipidaemia (high triglycerides and low HDL cholesterol, obesity, non-alcoholic fatty liver disease (NAFLD) as well as dementia, itself increasingly referred to as type 3 diabetes.

Insulin resistance is perhaps the most important risk factor for all these conditions. The causal association of insulin resistance to cardiovascular disease has been understood for decades[21] [22] and confirmed by contemporary research finding insulin resistance to be a strong and independent predictor of cardiovascular disease [23] [24] [25] [26] [27].

There are myriad mechanisms whereby insulin resistance contributes to the pathogenesis of atherosclerosis, including hypertension [28] [29] [30] [31] [32] [33] [34], glycocalyx disruption secondary to hyperglycaemia[35], pro-thrombosis[36], advanced glycation end product associated endothelial

[21] Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988; 37:1595–1607.
[22] Kraft J. Diabetes epidemic & you. Bloomington: Trafford Publishing; 2008.
[23] Haffner SM, Stern MP, Hazuda HP, et al. Cardiovascular risk factors in confirmed prediabetic individuals. Does the clock for coronary heart disease start ticking before the onset of clinical diabetes? JAMA 1990; 263:2893–2898.
[24] Lu MC, Fang WC, Li WC, et al. The Association between insulin resistance and cardiovascular disease risk: a community-based cross-sectional study among Taiwanese people aged over 50 years. Int J Environ Res Public Health 2020; 17.
[25] Hill MA, Yang Y, Zhang L, et al. Insulin resistance, cardiovascular stiffening and cardiovascular disease. Metabolism 2021; 119:154766.
[26] Adeva-Andany MM, Fernandez-Fernandez C, Carneiro-Freire N, et al. Insulin resistance underlies the elevated cardiovascular risk associated with kidney disease and glomerular hyperfiltration. Rev Cardiovasc Med 2020; 21:41–56.
[27] Pyorala M, Miettinen H, Laakso M, Pyorala K. Hyperinsulinemia predicts coronary heart disease risk in healthy middle-aged men: the 22-year follow-up results of the Helsinki Policemen Study. Circulation 1998; 98:398–404.
[28] Slivnick J, Lampert BC. Hypertension and heart failure. Heart Fail Clin 2019; 15:531–541.
[29] Reaven GM. Insulin resistance/compensatory hyperinsulinemia, essential hypertension, and cardiovascular disease. J Clin Endocrinol Metab 2003; 88:2399–2403.
[30] Reaven G. Insulin resistance, hypertension, and coronary heart disease. J Clin Hypertens (Greenwich) 2003; 5:269–274.
[31] Soleimani M. Insulin resistance and hypertension: new insights. Kidney Int 2015; 87:497–499.
[32] Esler M, Rumantir M, Wiesner G, et al. Sympathetic nervous system and insulin resistance: from obesity to diabetes. Am J Hypertens 2001; 14:304S–309S.
[33] Egan BM. Insulin resistance and the sympathetic nervous system. Curr Hypertens Rep 2003; 5:247–254.
[34] Facchini FS, Stoohs RA, Reaven GM. Enhanced sympathetic nervous system activity. The linchpin between insulin resistance, hyperinsulinemia, and heart rate. Am J Hypertens 1996; 9:1013–1017.
[35] Nieuwdorp M, van Haeften TW, Gouverneur MCLG, et al. Loss of endothelial glycocalyx during acute hyperglycemia coincides with endothelial dysfunction and coagulation activation in vivo. Diabetes 2006; 55:480–486.
[36] Springer International Publishing, Ghosh K. Kartha CC, Ramachandran S, Pillai RM. Diabetes as a prothrombotic state. Mechanisms of vascular defects in diabetes mellitus 2017. 361–376.

dysfunction[37] and impaired nitric oxide synthesis [38].These mechanisms all contribute to adverse effects on blood vessel structure and function [39] [40] [41].

Through multiple distinct mechanisms, IR is often the primary driver for hypertension[42], including stimulation of sodium retaining channels within the nephron [43] [44], as well as activation of the sympathetic nervous system[45] [46] [47]. The chronic hyperinsulinemia that occurs concurrently in IR promotes chronically elevated epinephrine, which elicits cardiovascular activation, including increased cardiac output and systemic vasoconstriction[48], as well as an enhancement of platelet aggregation[49].

Insulin resistance associated hyperinsulinemia is also associated with CVD risk through increased macrophage lipid accrual in blood vessels. As macrophages accrue lipids, they become 'foam cells'. Foam cells are a staple feature of atherosclerotic plaques, not only constituting a major portion of the plaque itself, but also contributing to atherosclerosis by aggressively secreting pro-inflammatory cytokines[50]. Park et al. [51] demonstrated that insulin increased macrophage oxidized LDL uptake by more than 80% and produced almost three times greater total lipid uptake into the macrophage in as little as 16 h. Insulin resistance is also associated with serum lipid components which are well established risk factors for cardiovascular disease.

Compelling evidence is now mounting that low carbohydrate diets may be effective in both reducing the risk of incident dementia as well as improving cognitive function in those already with dementia. Representing only 2% of the body's mass, yet consuming 20% of its energy, the brain is particularly susceptible to metabolic disease. Focal insulin resistance within the brain is a hallmark of dementia,

---

[37] Tan KCB, Chow WS, Ai VHG, et al. Advanced glycation end products and endothelial dysfunction in type 2 diabetes. Diab Care 2002; 25:1055–1059.
[38] Tessari P, Cecchet D, Cosma A, et al. Nitric oxide synthesis is reduced in subjects with type 2 diabetes and nephropathy. Diabetes 2010; 59:2152–2159.
[39] Lu MC, Fang WC, Li WC, et al. The Association between insulin resistance and cardiovascular disease risk: a community-based cross-sectional study among Taiwanese people aged over 50 years. Int J Environ Res Public Health 2020; 17.
[40] Hill MA, Yang Y, Zhang L, et al. Insulin resistance, cardiovascular stiffening and cardiovascular disease. Metabolism 2021; 119:154766.
[41] Domingues N. Insulin resistance as a predictor of cardiovascular diseases. Revista Portuguesa De Cardiologia 2021; 40:545–546.
[42] Reaven GM. Insulin resistance/compensatory hyperinsulinemia, essential hypertension, and cardiovascular disease. J Clin Endocrinol Metab 2003; 88:2399–2403.
[43] Reaven G. Insulin resistance, hypertension, and coronary heart disease. J Clin Hypertens (Greenwich) 2003; 5:269–274.
[44] Soleimani M. Insulin resistance and hypertension: new insights. Kidney Int 2015; 87:497–499.
[45] Esler M, Rumantir M, Wiesner G, et al. Sympathetic nervous system and insulin resistance: from obesity to diabetes. Am J Hypertens 2001; 14:304S–309S.
[46] Facchini FS, Stoohs RA, Reaven GM. Enhanced sympathetic nervous system activity. The linchpin between insulin resistance, hyperinsulinemia, and heart rate. Am J Hypertens 1996; 9:1013–1017.
[47] Egan BM. Insulin resistance and the sympathetic nervous system. Curr Hypertens Rep 2003; 5:247–254.
[48] Tack CJ, Lenders JW, Willemsen JJ, et al. Insulin stimulates epinephrine release under euglycemic conditions in humans. Metabolism 1998; 47:243–249.
[49] Henning RJ. Obesity and obesity-induced inflammatory disease contribute to atherosclerosis: a review of the pathophysiology and treatment of obesity. Am J Cardiovasc Dis 2021; 11:504–529.
[50] Yu XH, Fu YC, Zhang DW, et al. Foam cells in atherosclerosis. Clin Chim Acta 2013; 424:245–252.
[51] Park YM, Kashyap SR, Major JA, Silverstein RL. Insulin promotes macrophage foam cell formation: potential implications in diabetes-related atherosclerosis. Lab Invest 2012; 92:1171–1180.

resulting in regions of the brain deprived of energy. This leads to areas of impaired functioning manifesting as cognitive deficiency[52].

The close association between dementia, of which 70% of cases are caused by Alzheimer's disease, and metabolic ill health is well demonstrated by research which shows that subjects in the highest quintile for obesity are at nearly 3 times greater risk of dementia than those in the lowest quintile[53]. It is also a reliable observation that brain volume reduces inversely with body mass index, a relationship that holds true for those aged between 40-50 as much as it does for those over 60 [54] [55] [56]. This research underpins why dementia is now often referred to as Type 3 diabetes, and supported by more recent research finding having diabetes for more than 10 years by the age of 70 more than doubles the risk of dementia[57].

One of the pathological hallmarks of Alzheimer's disease are clumps of peptides known as beta amyloid plaques[58], which can now be visualised in living subjects using advanced brain imaging techniques[59]. It is now understood that glycation and oxidation of beta amyloid peptides precedes their clumping together in plaque formations [60] [61]. Factors which increase glycation and oxidation stresses predictably will therefore increase beta amyloid plaque deposition within the brain. This is why HbA1c, a marker of glycation of red blood cells often used in diagnosing type 2 diabetes, is strongly predictive of dementia risk[62].

Low carbohydrate diets have also been shown to be therapeutic in those suffering dementia[63]. For example, one high quality experimental trial which comparing low carbohydrate diets to the

---

[52] Murray, J. , H. Tsui, W. , Li, Y. , McHugh, P. , Williams, S. , Cummings, M. , Pirraglia, E. , Solnes, L. , Osorio, R. , Glodzik, L. , Vallabhajosula, S. , Drzezga, A. , Minoshima, S. , J. de Leon, M. and Mosconi, L. (2014) FDG and Amyloid PET in Cognitively Normal Individuals at Risk for Late-Onset Alzheimer's Disease. Advances in Molecular Imaging, 4, 15-26.

[53] Whitmer RA, Gustafson DR, Barrett-Connor E, Haan MN, Gunderson EP, Yaffe K (2008) Central obesity and increased risk of dementia more than three decades later. Neurology 71, 1057–1064.

[54] Hamer, M., & Batty, G. D. (2019). Association of body mass index and waist-to-hip ratio with brain structure. Neurology, 10.1212/WNL.0000000000006879.

[55] Jagust W, Harvey D, Mungas D, Haan M. Central obesity and the aging brain. Arch Neurol 2005;62:1545–1548.

[56] Ward MA, Carlsson CM, Trivedi MA, Sager MA, Johnson SC. The effect of body mass index on global brain volume in middle-aged adults: a cross sectional study. BMC Neurol 2005;5:23.

[57] Barbiellini Amidei C, Fayosse A, Dumurgier J, et al. Association Between Age at Diabetes Onset and Subsequent Risk of Dementia. JAMA. 2021;325(16):1640–1649. doi:10.1001/jama.2021.4001

[58] Cheng, Y., Tian, D.-Y., & Wang, Y.-J. (2020). Peripheral clearance of brain-derived Aβ in Alzheimer's disease: pathophysiology and therapeutic perspectives. Translational Neurodegeneration, 9(1).

[59] Valotassiou, V., Malamitsi, J., Papatriantafyllou, J., Dardiotis, E., Tsougos, I., Psimadas, D., Georgoulias, P. (2018). SPECT and PET imaging in Alzheimer's disease. Annals of Nuclear Medicine.

[60] Iannuzzi, C., Irace, G., & Sirangelo, I. (2014). Differential effects of glycation on protein aggregation and amyloid formation. Frontiers in Molecular Biosciences, 1.

[61] Guilbaud, A., Niquet-Leridon, C., Boulanger, E., & Tessier, F. (2016). How Can Diet Affect the Accumulation of Advanced Glycation End-Products in the Human Body? Foods, 5(4), 84.

[62] Ramirez A, Wolfsgruber S, Lange C, Kaduszkiewicz H, Weyerer S, Werle J, Pentzek M, Fuchs A, Riedel-Heller SG, Luck T, Mösch E, Bickel H, Wiese B, Prokein J, König HH, Brettschneider C, Breteler MM, Maier W, Jessen F, Scherer M; AgeCoDe Study Group. Elevated HbA1c is associated with increased risk of incident dementia in primary care patients. J Alzheimers Dis. 2015;44(4):1203-12. doi: 10.3233/JAD-141521. PMID: 25524954.

[63] Rusek, M., Pluta, R., Ułamek-Kozioł, M., & Czuczwar, S. J. (2019). Ketogenic Diet in Alzheimer's Disease. International journal of molecular sciences, 20(16), 3892. https://doi.org/10.3390/ijms20163892

American Heart Association recommended diet which is very similar to that advocated by the Australian Dietary Guidelines found marked benefit with the low carbohydrate approach[64]. These findings have been substantiated by numerous high-quality studies, one such study finding for example that low carbohydrate diets led to structural repair of brain deterioration after just one week[65], or another finding significant improvements in daily function and quality of life[66]. It is significant that the magnitude of benefit from these studies surpasses by a wide margin those achieved to date by any pharmacological intervention.

Appreciation of the fact that across the population, type 2 diabetes does not exist in isolation, but rather as a component of metabolic disease ultimately caused by insulin resistance, for which diet is a major risk factor facilitates an understanding of the breadth of benefit which may be obtained from clear unambiguous dietary advice reflective of the scientific literature. In addition to reductions in health spending on diabetes itself, responsible for 2.2% of the health budget [67], savings can be anticipated with respect to the 9.1% of health spending allocated to cardiovascular disease [68] as well as the more than $15 billion annual price tag for the management of dementia, projected by modelling from the National Centre for Social and Economic Modelling (NATSEM) to exceed $26 billion within 20 years[69].

Reduced carbohydrate diets have proven effectiveness in improving multiple medical issues which represent a substantial health burden on the Australian population. That their effective utilisation is effectively discouraged by the current Australian Dietary Guidelines has resulted in much unnecessary suffering and expenditure.

---

[64] Neth, B. J., Mintz, A., Whitlow, C., Jung, Y., Sai, K. S., Register, T. C., Craft, S. (2019). Modified Ketogenic Diet Is Associated With Improved Cerebrospinal Fluid Biomarker Profile, Cerebral Perfusion, And Cerebral Ketone Body Uptake In Older Adults At-Risk For Alzheimer's Disease: A Pilot Study. Neurobiology of Aging.

[65] Lilianne R. Mujica-Parodi, Anar Amgalan, Syed Fahad Sultan, Botond Antal, Xiaofei Sun, Steven Skiena, Andrew Lithen, Noor Adra, Eva-Maria Ratai, Corey Weistuch, Sindhuja Tirumalai Govindarajan, Helmut H. Strey, Ken A. Dill, Steven M. Stufflebeam, Richard L. Veech, Kieran Clarke. Diet modulates brain network stability, a biomarker for brain aging, in young adults. Proceedings of the National Academy of Sciences Mar 2020, 117 (11) 6170-6177.

[66] Phillips MCL, Deprez LM, Mortimer GMN, Murtagh DKJ, McCoy S, Mylchreest R, Gilbertson LJ, Clark KM, Simpson PV, McManus EJ, Oh JE, Yadavaraj S, King VM, Pillai A, Romero-Ferrando B, Brinkhuis M, Copeland BM, Samad S, Liao S, Schepel JAC. Randomized crossover trial of a modified ketogenic diet in Alzheimer's disease. Alzheimers Res Ther. 2021 Feb 23;13(1):51. doi: 10.1186/s13195-021-00783-x. PMID: 33622392; PMCID: PMC7901512.

[67] https://www.aihw.gov.au/reports/diabetes/diabetes/contents/impact-of-diabetes/health-system-expenditure

[68] https://www.aihw.gov.au/reports/heart-stroke-vascular-diseases/hsvd-facts/contents/impacts/expenditure-cvd

[69] https://www.dementia.org.au/about-us/media-centre/media-releases/266-billion-cost-alzheimers-disease-projected

4. Any interrelated health issues between diabetes and obesity in Australia, including the relationship between type 2 and gestational diabetes and obesity, the causes of obesity and the evidence-base in the prevention, diagnosis and management of obesity; and

Metabolic syndrome refers to a constellation of inter-related conditions which all feature insulin resistance as a major risk factor. This includes hypertension, diabetes, obesity and atherogenic dyslipidaemia. One of the major problems with respect to currently accepted dietary advice for the management of metabolic syndrome is the misrepresentation of study findings to support the study investigators original hypothesis. While this is clearly contrary to the scientific method, it has occurred in several large and influential studies.

That current dietary guidelines recommend high carbohydrate diets is a necessary consequence of recommendations to limit saturated fat intake. Thus research on the health impacts of dietary saturated fats is a central supporting tenet to our current misguided dietary guidelines.

I submit that important research providing putative support for our current dietary guidelines has been misrepresented. Despite the record being corrected within the literature, no meaningful changes have resulted with respect to dietary guidelines. I present three large scale randomised controlled trials investigating the consequences of dietary fat. These are the Women's Health Initiative Randomized Controlled Dietary Modification Trial, the Sydney Diet Heart study and the Minnesota Coronary Experiment . The Women's Health Initiative trial tried to find evidence supporting restriction of saturated fat. The only statistically significant result found however was that females with prior heart disease exposed themselves to a 26% higher risk of complications such as further coronary events by following a low-fat diet. Despite this being the only significant finding from what was a $700 million UDS publicly funded study, it was omitted from the results section and the conclusion, only being mentioned in obscure language within text on page 661 of the published journal. This omission has been attributed to an editorial oversight by the lead investigator, though no formal attempts have been made to disseminate this information.

Two other studies where the actual findings challenged the diet heart hypothesis were the Sydney Diet Heart study and the Minnesota Coronary Experiment. In both cases, the findings from these well conducted experiments, while paradigm challenging, were almost never published in entirety. Strikingly, transparency of their findings only occurred after recovery of study data from basements and eventual publication in the British Medical Journal, some 40 years after their conclusion. Both these studies utilised an intervention of reducing saturated fat intake, and both found evidence of harm.

In the case of the Sydney Diet Heart study, mortality in the intervention population increased by 62%. Predictably, these paradigm challenging findings were subject to much scrutiny and criticism. As a result, claims regarding the trans-fat content of the intervention diets have been used to discredit both studies. Trans-fats consumption is thought to increase the risk of cardiovascular disease. These criticisms however, amount to little more than mischievous subterfuge. For example, in the Sydney Diet Heart study, it is claimed that the soft margarine used in the intervention group led to an increase in trans-fat consumption. Unlike the hard margarines at the time however, soft margarines contained little, if any trans fats. And consumption of trans-fat containing foods, such as biscuits, cakes, pastries and puddings was expressly discouraged in the intervention group. Additionally, safflower oil, again low in trans fats, was used in the intervention group, further displacing sources of

trans fats. On balance, the trans-fat intake in the intervention group was almost certainly lower than both their baseline consumption and that of the control group. This argument therefore is little more than a smoke-screen attempting to obscure the true findings of these important studies.

The Minnesota Coronary Experiment randomised 9,423 residents of six mental hospitals and a nursing home to either a control diet high in saturated fat or an experimental diet rich in polyunsaturated fat. Like the Sydney Diet Heart study, increased mortality was found with cholesterol lowering diets, directly challenging the a priori hypothesis of the study investigators. When asked in an interview by science journalist Gary Taubes, the lead investigator (Frantz) admitted that the delay in publishing the study findings was because 'we were just so disappointed in the way they turned out'. In addition to delaying publication of data, Frantz also appears to have misrepresented the findings, promoting the idea that there may have been a 'favourable trend' for low saturated fat diets in 'younger age groups'. This is not to suggest that researchers regularly or wilfully misrepresent study findings. Rather it is likely these issues arise from cognitive biases. Unfortunately, regardless of intent, the potential consequences of these types of scientific deceptions have proven catastrophic for public health. Moving forward, dietary guidelines should reflect only scientific data, and steps should be taken to minimise the impact of poor scientific practice.

5. The effectiveness of current Australian Government policies and programs to prevent, diagnose and manage diabetes.

The continued increase in the prevalence of type 2 diabetes and associated disease is evidence of the failure of current policies and programs in this regard. Central to this is the continued unscientific dietary recommendations promoted by the Australian Dietary Guidelines. Throughout various iterations since their introduction, the Australian Dietary Guidelines have recommended high carbohydrate diets low in saturated fats. These recommendations arise from the diet-heart hypothesis, which was first proposed in the 1950's. Widespread acceptance of this hypothesis led many doctors to concern themselves with their patients' cholesterol levels. Importantly, it is now well accepted that dietary cholesterol and saturated fat is not a risk factor for cardiovascular disease[70], something which is not reflected in the Australian Dietary Guidelines.

What is commonly referred to as cholesterol by doctors are in fact complex structures called lipoproteins. These lipoproteins include a phospholipid outer shell containing embedded functional proteins and carry a cargo of hydrophobic lipid around the circulation. Cholesterol molecules simply form a part of this cargo. There are several different types of lipoprotein particles, which can be defined based on size and density. This includes chylomicrons, very low-density lipoproteins (VLDLs), intermediate density lipoproteins (IDLs), low density lipoproteins (LDLs) and high density lipoproteins (HDLs).

The concept of what may be referred to as total cholesterol, or the sum total of the individual lipo-protein subfractions, is now generally recognised to be a sub-optimal metric for cardiovascular risk. Even low density lipoprotein which is often perjoratively referred to as 'bad cholesterol' appears to associate inversely with all cause mortality. That is, those with higher levels of LDL tend to live longer.

One recent meta-analysis of this topic, published in the British Medical Journal, found that of 19 prospective studies addressing this question, 16 of them found an inverse relationship – in effect those with higher LDL levels lived longest[71]. Attempts to cast doubt on this finding by proponents of the diet heart hypothesis frequently cite the potential issue of reverse causation. The concept of reverse causality is basically that illness lowers LDL levels, meaning that those with the lowest LDL levels are simply those who are already ill. Indeed, the observation that LDL levels tend to reduce in the last 2 years of life is well documented[72], however the average duration of observation of studies included within this meta-analysis was far more than 2 years. Furthermore, even when those in the lowest quartile of LDL levels are removed, and the all-cause mortality of the 2nd quintile is compared to the 4th quintile (thereby eliminating those presumed to be carrying pre-existing illness), a clear mortality benefit is still evident in those with the highest LDL levels.

---

[70] Soliman G. A. (2018). Dietary Cholesterol and the Lack of Evidence in Cardiovascular Disease. Nutrients, 10(6), 780. https://doi.org/10.3390/nu10060780

[71] Ravnskov U, Diamond DM, Hama R, et al. Lack of an association or an inverse association between low-density-lipoprotein cholesterol and mortality in the elderly: a systematic reviewBMJ Open 2016;6:e010401. doi: 10.1136/bmjopen-2015-010401

[72] Charlton J, Ravindrarajah R, Hamada S, Jackson SH, Gulliford MC. Trajectory of Total Cholesterol in the Last Years of Life Over Age 80 Years: Cohort Study of 99,758 Participants. J Gerontol A Biol Sci Med Sci. 2018;73:1083-1089. doi: 10.1093/gerona/glx184.

Even when, in one study, patients with a history of terminal disease, cardiovascular disease, diabetes or statin prescriptions were excluded, the association between higher LDL levels and longevity was both evident and statistically significant to $p< 0.001$, with an effect size in the order of a 50% reduced chance of death, when comparing the highest and lowest quartiles[73]. The science associating higher serum LDL levels with an increased lifespan is therefore robust.

Another reason why low serum LDL levels are not necessarily desirable is that some therapies which have been proven to lower serum LDL levels offer no cardiovascular benefits. This includes hormone replacement therapy [74] and cholesteryl ester transport protein inhibitors[75]. The corollary is also true, with sodium-glucose cotransporter type 2 inhibitors being shown to both increase serum LDL levels and reduce cardiovascular events[76], [77].

Indeed, in a finding that will surprise many, not only has it been shown that regression of atherosclerosis is possible, but it also appears to be facilitated by higher LDL levels. For example, a 2010 study found that patients with progression of atherosclerosis had lower LDL levels than those with regression [78].

A recent expert review article published in the Journal of the American Academy of Cardiology[79] also addressed the issue of saturated fat consumption as it related to serum LDL levels. It was concluded that while saturated fat consumption could increase serum LDL levels, this did not appreciably increase cardiovascular risk. Indeed, this expert review provided a degree of nuanced commentary

---

[73] Bathum L, Depont Christensen R, Engers Pedersen L, et al. Association of lipoprotein levels with mortality in subjects aged 50+without previous diabetes or cardiovascular disease: a population-based register study. Scand J Prim Health Care 2013;31:172–80. doi:10.3109/02813432.2013.824157

[74] Manson JE, Hsia J, Johnson KC, et al. Estrogen plus progestin and the risk of coronary heart disease. N Engl J Med 2003;349:523-34.

[75] Armitage J, Holmes MV, Preiss D. Cholesteryl ester transfer protein inhibition for preventing cardiovascular events: JACC review topic of the week. J Am Coll Cardiol 2019;73:477-87.

[76] Zaccardi F, Webb DR, Htike ZZ, Youssef D, Khunti K, Davies MJ. Efficacy and safety of sodium-glucose cotransporter 2 inhibitors in type 2 diabetes mellitus: Systematic review and network meta-analysis. Diabetes Obes Metab. 2016.

[77] Bays HE, Sartipy P, Xu J, Sjostrom CD, Underberg JA. Dapagliflozin in patients with type II diabetes mellitus, with and without elevated triglyceride and reduced high-density lipoprotein cholesterol levels. J Clin Lipidol. 2017;11:450 e1–458 e1.

[78] Spence JD, Hackam DG. Treating arteries instead of risk factors: a paradigm change in management of atherosclerosis. Stroke. 2010 Jun;41(6):1193-9. doi: 10.1161/STROKEAHA.110.577973. Epub 2010 Apr 22. PMID: 20413738.

[79] Astrup, A., Magkos, F., Bier, D. M., Brenna, J. T., de Oliveira Otto, M. C., Hill, J. O., Krauss, R. M. (2020). Saturated Fats and Health: A Reassessment and Proposal for Food-based Recommendations: JACC State-of -the-Art Review. Journal of the American College of Cardiology.

often missing from the discussion, concluding that free consumption of meat and eggs ought not be discouraged[80].

While it is true that 'on average' serum LDL level correlates inversely with ill health, some LDL particles do play a causal role in the pathogenesis of atherosclerotic cardiovascular disease[81]. These are what are known as 'small dense' LDL particles, which have been damaged by a process of glyco-oxidation. These small dense LDL particles are relatively depleted of cholesterol and have a much greater association with cardiovascular disease, than the larger, cholesterol rich undamaged LDL particles[82].

This pattern of LDL is associated with low HDL levels and high triglyceride levels, commonly referred to as atherogenic dyslipidaemia, and has not been shown to be caused by the consumption of saturated fat. Restriction of saturated fat consumption does not reduce the levels of serum 'small dense' LDL particles[83], while low carbohydrate and ketogenic diets, often high in saturated fats, have been shown to improve atherogenic dyslipidaemia. It is therefore apparent that a degree of nuance must be applied to the evaluation of cardiovascular risk based on serum lipid levels, with the triglyceride:HDL ratio being a more reliable marker than serum LDL.

It is an implication of discouraging saturated fat intake on the back of the lipid hypothesis that recommended diets must then necessarily be high in carbohydrates. This is despite an abundance of high-quality research proving the benefits of diets high in saturated fat. This includes numerous systematic reviews have been conducted on the question of dietary saturated fat, even extending to two 'umbrella reviews' which reviewed the systematic reviews. One of these umbrella reviews considered 17 systematic analyses[84] and concluded that reducing saturated fat did not convincingly reduce cardiovascular events or mortality. The other umbrella review, considered 19 meta-analyses[85] and found no association between saturated fat intake and heart disease, ultimately concluding recommendations to limit saturated fat intake need re-evaluation.

The weight of this evidence is now beginning to alter the perspective of influential experts, even those who have previously held contrary views. An expert review article[86] published in the Journal of

---

[80] Astrup, A., Magkos, F., Bier, D. M., Brenna, J. T., de Oliveira Otto, M. C., Hill, J. O., Krauss, R. M. (2020). Saturated Fats and Health: A Reassessment and Proposal for Food-based Recommendations: JACC State-of - the-Art Review. Journal of the American College of Cardiology.

[81] Boren J, Chapman MJ, Krauss RM, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J 2020.

[82] Bergeron N, Chiu S, Williams PT, S MK, Krauss RM. Effects of red meat, white meat, and nonmeat protein sources on atherogenic lipoprotein measures in the context of low compared with high saturated fat intake: a randomized controlled trial. Am J Clin Nutr 2019;110:24-33.

[83] Bergeron N, Chiu S, Williams PT, S MK, Krauss RM. Effects of red meat, white meat, and nonmeat protein sources on atherogenic lipoprotein measures in the context of low compared with high saturated fat intake: a randomized controlled trial. Am J Clin Nutr

[84] DuBroff R., de Lorgeril M. Fat or fiction: The diet-heart hypothesis. BMJ Evid. Based Med. 2019;26:3–7. doi: 10.1136/bmjebm-2019-111180.

[85] Jeffery L Heileson, Dietary saturated fat and heart disease: a narrative review, Nutrition Reviews, Volume 78, Issue 6, June 2020, Pages 474–485, https://doi.org/10.1093/nutrit/nuz091

[86] Astrup A, Magkos F, Bier D, et al. Saturated Fats and Health: A Reassessment and Proposal for Food-Based Recommendations. J Am Coll Cardiol. 2020 Aug, 76 (7) 844–857.

the American Academy of Cardiology concluded that scientific evidence, including that from meta-analyses of randomised controlled trials (the gold standard of medical evidence), now undermines the longstanding recommendation to limit dietary saturated fat. Three of the authors of this paper were directly involved in the drafting of a previous iteration of Dietary Guidelines for Americans, recommending restrictions on saturated fat intake. Their authorship of this paper, and ownership of the ideas contained demonstrate the progressive acceptance of the idea that recommending the restriction of saturated fat is not required. Unfortunately, this understanding of the science has not yet percolated into the Australian Dietary Guidelines.

Another area in which the government could ensure evidence-based policy is with respect to recommendations relating to red meat consumption. Various claims alleging adverse health impacts, chief amongst them the putative risk of colo-rectal cancer have been used as a basis for the limiting of red meat consumption. This,however, is a significant distortion of the science, and risks exacerbating nutrient deficiencies as well as encouraging the intake of higher carbohydrate foods associated with increased risk of type 2 diabetes.

Much of the concern regarding a link between red meat consumption and cancer for example arises directly from a two-page World Health Organisation report published in 2015[87]. This widely publicised report claimed that processed meat definitely causes colorectal cancer in humans, and that red meat 'probably' causes colorectal cancer in humans. It listed only 20 references in support of these contentions. These references however, appear to have been selectively chosen. Multiple studies which showed either no connection between meat and cancer or even a protective effect of meat on colon cancer risk were not referenced. Indeed, the report itself states that more than 800 epidemiological studies investigation the association between red meat and cancer were considered, with a mere 29 being judged to be 'informative'. And the majority of these 29 studies, 15 of them, found no association between red meat and colorectal cancer. As for processed meats, only 27 studies were deemed 'informative' with only 14 associating a higher risk of colorectal cancer. And this is in the context that epidemiological studies such as these are not able to demonstrate causation, and hypotheses arising from them require validation with experimental trials[88], [89].

It is therefore significant that only six experimental studies were cited to support the contention that red meat consumption is causally related to colorectal cancer, and all of these had major methodological failings. Three were conducted solely on rats injected with carcinogenic chemicals,

[87] Bouvard V, Loomis D, Guyton KZ, Grosse Y, Ghissassi FE, Benbrahim-Tallaa L, Guha N, Mattock H, Straif K; International Agency for Research on Cancer Monograph Working Group. Carcinogenicity of consumption of red and processed meat. Lancet Oncol. 2015 Dec;16(16):1599-600.

[88] David M. Klurfeld, Research gaps in evaluating the relationship of meat and health, Meat Science, Volume 109, 2015, Pages 86-95.

[89] Ioannidis JPA. The Challenge of Reforming Nutritional Epidemiologic Research. JAMA. 2018;320(10):969–970. doi:10.1001/jama.2018.11025

euphemistically referred to as treatment with 'colon cancer initiators', prior to being fed meat[90] [91] [92]. One does not need scientific training to realise that extrapolation of this kind of research to humans is inappropriate. The three experimental studies which included human subjects (one was a rat / human study) were also fundamentally flawed. Methodological limitations included but were not limited to, small numbers of participants [93] [94] [95], the use of unreliable or outdated biomarkers[96], the presence of confounders such as co-consumption of sugary juices [97] and lack of adequate controls[98]. It was on this background that a group of 23 international cancer experts gathered to examine the science presented in this report, concluding that the conclusions drawn were unjustified[99].

The subsequent publication of the heavily promoted EAT-Lancet report promoted by the World Health Organisation again linked red meat consumption to cancer[100]. As with the 2015 report, scientific justification was lacking, this time every single one of the 16 references in the relevant section being an epidemiological study. It is therefore clear that discouraging red meat intake on the spurious notion that it may cause an increase in colorectal cancer is without basis. Australian government policy ought to clearly repudiate non-scientific dietary recommendations in all their forms.

---

[90] Pierre FH et al. Beef meat promotion of dimethylhydrazine-induced colorectal carcinogenesis biomarkers is suppressed by dietary calcium. Br J Nutr. 2008;99:1000–1006.

[91] Pierre FH et al. Beef meat and blood sausage promote the formation of azoxymethane-induced mucin-depleted foci and aberrant crypt foci in rat colons. J Nutr. 2004;134:2711–2716.

[92] Santarelli RL et al. Meat processing and colon carcinogenesis: cooked, nitrite-treated, and oxidized high-heme cured meat promotes mucin-depleted foci in rats. Cancer Prev Res. 2010;3(7):852-864.

[93] Pierre FH et al. Calcium and alpha-tocopherol suppress cured-meat promotion of chemically induced colon carcinogenesis in rats and reduce associated biomarkers in human volunteers. Am J Clin Nutr. 2013;98: 1255–1262.

[94] Le Leu RK et al. Butyrylated starch intake can prevent red meat-induced O6-methyl-2-deoxyguanosine adducts in human rectal tissue: a randomised clinical trial. Br J Nutr. 2015;114:220–230.

[95] Lewin MH et al 2006. Red meat enhances the colonic formation of the DNA adduct O6-carboxymethyl guanine: implications for colorectal cancer risk. Cancer Res. 2006;66:1859–1865.

[96] Pierre FH et al. Calcium and alpha-tocopherol suppress cured-meat promotion of chemically induced colon carcinogenesis in rats and reduce associated biomarkers in human volunteers. Am J Clin Nutr. 2013;98: 1255–1262.

[97] Le Leu RK et al. Butyrylated starch intake can prevent red meat-induced O6-methyl-2-deoxyguanosine adducts in human rectal tissue: a randomised clinical trial. Br J Nutr. 2015;114:220–230.

[98] Lewin MH et al 2006. Red meat enhances the colonic formation of the DNA adduct O6-carboxymethyl guanine: implications for colorectal cancer risk. Cancer Res. 2006;66:1859–1865.

[99] Oostindjer M, Alexander J, Amdam GV et al. The role of red and processed meat in colorectal cancer development: a perspective. Meat Sci. 2014; 97(4):583-96. https://www.ncbi.nlm.nih.gov/pubmed/24769880.

[100] Willett W, Rockström J, Loken B, Springmann M, Lang T, Vermeulen S, Garnett T, Tilman D, DeClerck F, Wood A, Jonell M, Clark M, Gordon LJ, Fanzo J, Hawkes C, Zurayk R, Rivera JA, De Vries W, Majele Sibanda L, Afshin A, Chaudhary A, Herrero M, Agustina R, Branca F, Lartey A, Fan S, Crona B, Fox E, Bignet V, Troell M, Lindahl T, Singh S, Cornell SE, Srinath Reddy K, Narain S, Nishtar S, Murray CJL. Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. Lancet. 2019 Feb 2;393(10170):447-492. doi: 10.1016/S0140-6736(18)31788-4. Epub 2019 Jan 16. Erratum in: Lancet. 2019 Feb 9;393(10171):530.