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Reporting on key nutrition indicators as a decision support tool to improve diet quality in remote Indigenous Australian communities

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Introduction: Most food consumed in remote Indigenous Australian communities is purchased at the local store that in many instances belongs to the community and is governed by community representatives (store directors) [1]. These community representatives and remote retailers are therefore key decision makers to optimise stores to support healthier eating and reduce obesity and preventable chronic disease. Reports tracking diet quality indicators can be a powerful decision-support tool for supporting this process [2].

Aim: We aimed to develop timely, accessible, easy to interpret and evidence-informed reports (FoodFox reports) on indicators of community-level diet quality.

Methods: To inform report development we examined best evidence, consulted with Indigenous Australian store directors in 3 communities and collected 18 months of sales data from 31 stores across the Northern Territory, Far North Queensland and the Torres Strait. Reports were made available to participating stores and follow-up interviews occurred with store managers to inform future implementation.

Outcomes: Consultation with Indigenous Australia community representatives (store directors) indicated that they were eager to receive reports to inform their decision-making and that they saw the reports as having potential value in optimising stores to support healthier food and drink choices. They also provided specific feedback regarding cultural relevance of language and data visuals which was used to refine the reports. The resulting reports show key food indicators tracked longitudinally and benchmarked against dietary guideline targets, store goals and the average of participating stores. The indicators (6 food groups and 40 subgroups) and categorisation (Best Choices, Less Healthy Choices and Unhealthy Choices) align with Australian Dietary Guidelines. Store manager interviews identified several potential barriers and enablers to consistent and effective report dissemination and uptake.

Conclusion: These reports have potential as a decision support tool to improve diet quality and reduce obesity and chronic disease risk in remote Indigenous Australian communities.

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Obesity and increased cancer risk: the development of a public health education campaign

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Background: Obesity is recognised as a preventative risk factor for 13 types of cancer [1] and community awareness of this connection is low [2,3]. Currently approximately 63% of Australians are overweight or obese and an important contributor to this problem is excessive sugary drink consumption [4,5]. Cancer Council Victoria is investing in a mass media public education campaign that will be one of the first in Australia to highlight the association between obesity and cancer risk.

Aim: To conduct a public health education campaign that aims to:

Increase community awareness of the link between obesity and risk of cancer.
Increase awareness that sugary drinks can contribute to weight gain.

Methods: The campaign has been informed by existing epidemiological evidence, clinical network engagement and empirical and qualitative research on weight and lifestyle to evaluate advertising. The resultant mass media campaign and supporting website will be launched in October 2018. Stakeholders from eating disorder, mental health and women's health groups were also consulted.

Results: A TV commercial, depicting a surgeon warning of the harmful effects that sugary drinks have on risk of weight gain and in turn increased risk of certain cancers was chosen. The ad was chosen based on focus testing feedback on its believability, sense of personal susceptibility and motivation and urgency for behaviour change. The campaign will be accompanied by supporting digital and outdoor media, and supportive website with information, videos, animations and lifestyle strategies to reduce cancer risk.

Conclusion: The campaign will run in October and November 2018 in Victoria. Evaluation results will be available following the campaign.

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Simulated economic impacts of Australian Obesity Management Algorithm implementation: microsimulation modelling to 2030

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Obesity is a complex, multi-factorial chronic disease, affecting 28% of Australian adults in 2015 [1]. In 2016, The Australian Obesity Management Algorithm, a practical clinical tool for use in primary care, was released. The Algorithm sets target weight loss for four

different sub-groups of persons with obesity. This study aimed to investigate the potential impact if such levels of weight loss could be achieved nationally and the cost implications.

The study used NCDMod [2], a microsimulation model that simulates inter-relationships of risk factors (including obesity), chronic disease and health system costs. NCDMod operates in 5 year cycles projecting to 2030. The main scenario simulated 20% of potential patients with obesity achieving their target weight loss and maintaining weight loss for the 15 year simulation. Sensitivity analysis was done including addition of drop-out/failure to lose weight despite starting treatment as set out by the obesity management algorithm. Key health outcomes were: Decrease in body mass index, cardiovascular disease events averted, quality adjusted life years gained and health system cost offsets.

In comparison to weight trajectories remaining at historical levels, implementation of the Obesity Management Algorithm estimates almost 500,000 less persons with obesity in 2030, resulting in a cost offset in the health system over the 15 years to 2030 of Au$12.7 billion to potentially fund the required weight management programs. Even assuming that 40% of those that start the program either drop-out or fail to lose weight, there still would be 280 000 less persons with obesity in 2030 providing health system cost-offsets of Au$7.7 billion.

The need to facilitate multifaceted, intensive weight management programs described in The Australian Obesity Management Algorithm is illustrated in this simulation, showing the potential decrease in persons with obesity over time and the freeing of health system funding to pay for such services.

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The role of the pro-apoptotic protein BIM in glucose homeostasis and preservation of islet mass in type 2 diabetes

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IL-22-based tissue-targeted therapeutics for type 2 diabetes

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The interplay between insulin and the NPY system

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Insulin has been shown to have effects not only in the peripheral tissues, but also in the brain particularly in the arcuate nucleus of the hypothalamus to regulate glucose and energy homeostasis. The importance of the central neuropeptide Y (NPY) system in the regulation of appetite and energy metabolism is well established. Although insulin is known to influence NPY expression, the precise physiological role of insulin action in these neuronal cells remains unknown. A closely related peptide, PYY released from L-type cells of the gut, is also expressed in α-cells in the pancreatic islets. It has been shown that application of PYY decreases glucose stimulated insulin secretion from rat and mouse islets. PYY can activate Y1, Y2 and Y5 receptor to regulate feeding and energy balance, however, little is known as to which Y receptor(s) mediate PYY's effect in the pancreatic islets. We have recently identified that the Y1 receptors are expressed in the β-cells both in mice and humans. In this presentation, I will discuss the role of insulin action in NPY-expressing neurons and provide evidence addressing the role of Y1 receptor in the regulation of β-cell function and how inhibition of this receptor could improve islet transplantation outcome.

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Cafeteria diet-induced cognitive impairment is prevented by oral minocycline in association with changes in the microbiome

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Obesogenic diets and obesity are associated with cognitive impairment in both humans and animals. While the underlying mechanisms remain controversial, a key potential driver is increased inflammatory signalling associated with obesity. The anti-inflammatory antibiotic minocycline hydrochloride has been routinely used to depress microglial activity as it easily crosses the blood brain barrier. We used a rodent model to test the hypothesis that minocycline would alleviate the cognitive impairment produced by a high-fat high-sugar "cafeteria" diet. The study was a 2×2 design, where rats were exposed to either vehicle (syrup) or minocycline (40 mg/kg/day) while consuming either regular chow or cafeteria diet. Memory was tested using novel object and place recognition tasks (NOR and NPR, respectively) and EchoMRI determined body composition across 6 weeks of intervention. Rats fed the cafeteria diet and vehicle were impaired on the hippocampal-dependent NPR at 2, 4 and 6 weeks but minocycline administration spared NPR performance in cafeteria-fed rats (similar exploration ratios as chow and vehicle). Of interest, chow rats treated with minocycline performed worse than those treated with vehicle. Cafeteria-fed rats irrespective of drug treatment consumed 150% more energy over the experiment and gained 100% more fat mass relative to rats fed chow. Faecal microbiota alpha diversity was reduced by both cafeteria diet and minocycline,