

perspectives

NUTRITION NEWS AND VIEWS

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Treating adolescent obesity in 'Eat Smart': What they want works by Helen Truby



The current and potential health, social and economic consequences of childhood obesity across Australia and New Zealand are enormous. Provision of an evidence base for effective weight management programmes in this group is critical.

KEY POINTS

- Reduced carbohydrate diets appear just as effective as low fat diets for weight reduction in obese adolescents.
- Structured diets with portion control are preferred for weight reduction by families of obese adolescents.
- Tailored dietary advice is essential in this group.
- Dietary advice is preferred over physical activity advice in this group.

The 2007 Australian National Children's Nutrition and Physical Activity Survey reported that 22% of boys and 24% of girls aged 2-16 years were overweight or obese⁽¹⁾. The 2002 National Children's Nutrition Survey in New Zealand reported that 29% of boys and 33.5% of girls aged 5-14 years were overweight or obese⁽²⁾. Childhood obesity tracks into adulthood⁽³⁾. There is a clear requirement to develop effective weight management programmes, in particular for severely obese adolescents where the evidence base remains weak.

FEASIBILITY STUDY: EAT SMART

To understand how to engage adolescents and their families in behaviour change, the salient factors that are important barriers and facilitators of change need to be identified first; in essence to define what solutions they and their families are seeking. To inform intervention, we conducted a 9-month feasibility study called 'Eat Smart', commencing in March 2007 at the Queensland Children's Hospital, Brisbane⁽⁴⁾. In this pilot work, implemented as a 12-week trial, we aimed to test the use of a reduced carbohydrate eating plan and to test the acceptability of using structured eating plans (low fat (55% carbohydrate, 25% fat) and reduced carbohydrate (35% carbohydrate, 40% fat)) compared to a more standard model of care – a low fat unstructured approach⁽⁴⁾.

The feasibility study suggested that Australian families and adolescents preferred tailored energy prescription and structured advice for treatment.

Only 7% of participants elected the unstructured approach. The option to decrease high carbohydrate foods presented as a suitable option to pursue and would broaden the choice of dietary patterns that could be offered in practice. Psychological issues uncovered in this feasibility phase led us to consider psychological screening system and intervention program prior to attempting dietary change.

REFINING EAT SMART

The next phase of development of the 'Eat Smart' program compared the two structured approaches over 12 weeks, dropping the unpopular 'standard care' approach and adding an untreated control group. We aimed to determine the metabolic and cardiovascular effects of a structured reduced carbohydrate diet versus low fat in a short term study design. A preparation phase was added where subjects completed a 6 week validated group based program, 'FRIENDS for life'⁽⁵⁾, aiming to prepare for change, reduce anxiety, and increase self efficacy and coping skills. A psychologist screened subjects to ensure they scored below clinical cut-offs on the Anxiety Disorder Interview Schedule⁽⁶⁾ before being randomised to group stratified by sex and pubertal stage.



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This issue of Perspectives highlights the underlying intricacies of nutrition recommendations and the ability to choose a healthy diet.

Ostan and colleagues have asked the question “Why do people, who have enough income to choose healthier food, behave in a self-destructive way by choosing unhealthy nutrition?”^(1, p443) – one that may simultaneously offend and challenge. I’d like to reflect on food choice in context of some of the nutrition issues brought to us in the current *Perspectives*.

If dietary recommendations are made based on science and communicated as such, how are they incorporated into one’s diet? Truby et al suggest that what practitioners may regard as ‘best practice’ is not always what their patients wish to receive^(2, p3). There is complexity in putting dietary advice into practice and the need for practitioners to consider tailoring the dietary advice package that individuals receive according to individual circumstances and desires, as well as what we think they need. In this issue of *Perspectives*, Helen Truby reports on the success of providing structured advice (what clients wanted) for adolescent weight management.

Evolution in scientific thinking and changes in recommendations can mean wise food choice and advising in line with these is challenging. Jane Rycroft informs us on the changing direction on antioxidants theory and points to evidence for their action in vitro vs in vivo. The material reviewed for the Heart Foundation Position Statement on antioxidants for cardiovascular health and subsequent recommendations was scoped based on queries on this topic received from professionals and the public (see Tuesday Udell’s report).

Providing food and nutrition advice in an environment where there is a call for sustainable food production has caused local debate and controversy⁽³⁾. Williams reports that population growth will require food production to double in the next fifty years and points to the challenge of managing natural resources with food and nutrition security. He calls for change in government policy, increased investment in agro-science, and a change in the view and practice of farming. How does/will this impact food and nutrition recommendations and following on from this, food choice?

Even if dietary recommendations are understood and delivered to the client in their preferred way, and are environmentally conscientious, then what

about the impact of biological basis for food choice? Russell Keast gives an insight into another biological mechanism impacting food choice: oral sensitivity to fat. This promising area of research demonstrates a potential for oral hyposensitivity to fat driving increased fat and energy intake (and BMI).

Food choice and nutrition science cannot be viewed without the context of the time⁽⁴⁾ – a time where health, political and environmental problems impact on the ability to address food and nutrition issues and where ‘knowledge integration from a range of disciplines is required’^(4, p2). Issues presented in this *Perspectives* (and many others) that impact food choice and consumption are interwoven, requiring holistic and contextual thinking in approach.

Janelle Gifford, PhD, Accredited Practising Dietitian

Note: I encourage you to visit Unilever’s new nutrition information website www.unileverhealthcarenutrition.com.au for healthcare professionals where you can find information on a range of topics and download reports from the DAA National Conference breakfast symposiums ‘The Latest on Kids Snacking’ and ‘Antioxidants: Panacea or Placebo’.

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Dietary antioxidants: *In vitro* vs *in vivo* evidence by Jane Rycroft



So-called dietary antioxidants encompass a wide range of compounds (including, vitamin C, vitamin E, zinc, selenium, and flavonoids) that are naturally present in foods and beverages.

In the past fifteen years the preventative and therapeutic potential of molecules referred to as antioxidants, from natural sources and supplements has been thoroughly researched. With this research, understanding of the physiological role of dietary antioxidants in human health has shifted.

In theory, dietary antioxidants were proposed to have health benefits by protecting the body against free radical damage. Plentiful evidence has accumulated that dietary antioxidants can quench free radicals *in vitro*. However, evidence that dietary antioxidants reduce free radical damage *in vivo* has not been found⁽²⁻⁴⁾.

WHAT IS AN ANTIOXIDANT?

It is a substance that when present at low concentrations, compared to that of an oxidisable substrate, significantly delays

KEY POINTS

- Antioxidant effects *in vitro* do not necessarily predict antioxidant action *in vivo* or related health effect.
- The mechanism by which dietary antioxidant compounds exert their health benefits is not via antioxidant/free radical quenching activity, but via other mechanisms.
- The use of the term antioxidant should be reconsidered to instead refer specifically to the molecules themselves, such as vitamins and polyphenols, their mechanisms and health benefits.

or prevents oxidation of that substrate⁽⁵⁾. The definition of dietary antioxidant (proposed by the Food Nutrition Board)⁽⁶⁾ is a substance in food that significantly decreases the adverse effects of reactive oxygen species, reactive nitrogen species, or both on normal physiological function in humans. Antioxidants can inhibit oxidation reactions by being oxidized themselves (for review of antioxidant mechanisms see Niki, (2010)⁽⁷⁾).

EVIDENCE FOR ANTIOXIDANT ACTIVITY *IN VITRO* VS *IN VIVO*

The 'antioxidant hypothesis' that high intake of dietary antioxidant may protect against coronary heart disease emerged from the widely postulated role of LDL oxidation in the initiation of vascular disease⁽⁸⁾. Dietary antioxidants including vitamin C, E and β -carotene have received the greatest attention with regard to coronary heart disease, due to early observational studies that have suggested the importance of dietary antioxidants in heart disease prevention⁽⁹⁻¹¹⁾.

Flavonoid intake has also been associated to lower risk of coronary heart disease in early observational studies⁽¹²⁾, and a role as antioxidants has been suggested. *In vitro* studies show that, for example antioxidants from fruit and vegetables⁽¹³⁾ and wine⁽¹⁴⁻¹⁶⁾ demonstrate antioxidant activity. However, whilst the molecules that act as antioxidants *in vitro* are absorbed, the levels found in plasma may be low^(17,18) and may not be physiologically relevant.

Many dietary compounds have powerful antioxidants effects in various in vitro test systems; however, these effects do not predict antioxidant actions in vivo or a related health effect.

There has been much research on the potential role of *in vitro* antioxidants in the prevention of disease development in the last two decades. Whilst some evidence has developed in support of *in vivo* antioxidant effects, it is inconsistent. For example, people with diets rich in fruit and vegetables have an increase in the concentration of β -carotene in the blood and a decreased risk of cancer, however supplements

of β -carotene do not have an anti-cancer effect, rather the opposite in smokers⁽¹⁹⁾. Fruit and vegetable consumption decreases the amount of free-radical damage to DNA in the human body (a risk factor for cancer development), but supplements of ascorbate, vitamin E, or β -carotene do not decrease DNA damage in most studies⁽²⁰⁻²⁶⁾. A recent meta-analysis on use of antioxidant supplements found no preventive effect on cancer, and even suggested an increased risk of bladder cancer⁽²⁷⁾. Despite more than fifty studies showing the potent antioxidant activity of flavonoids *in vitro*, an *in vivo* antioxidant action has not been demonstrated^(2,3). Flavonoids have, however, been shown *in vivo* to enhance synthesis and release of nitric oxide, which plays a pivotal role in blood vessel tone and reactivity^(28,29).

This does not mean dietary antioxidants do not have health benefits. It just means the mechanism by which dietary antioxidant compounds exert their health benefits is not via antioxidant/free radical quenching activity, but via other mechanisms.

WHAT CAN WE UNDERSTAND FROM THE TERM ANTIOXIDANT?

Refuting the antioxidant hypothesis should not be interpreted to indicate that antioxidant systems in the body are unimportant. On the contrary, it would seem that the human body is so well regulated that dietary antioxidants do not provide any additional value to the body's antioxidant defence system. The use of the term 'antioxidant' should be reconsidered; instead we should start to refer to the specific molecules themselves such as vitamins and polyphenols, and focus on their health benefits which are likely to be the result of alternative mechanisms of action.

References (see p2)

Dr Jane Rycroft is Nutrition Manager at Unilever Research & Development, United Kingdom

Farming without harming by John Williams



As world population is set to expand from 6.7 billion to 9.2 billion by 2050, projected demand for food will require agricultural and fisheries production to double over the next fifty years.

Substantial increase in production must be achieved with a decreasing impact on the natural resources and environment^(1,2). Producing our foods and ensuring that we also reduce the number of people who are malnourished in light of all the environmental pressure that must be managed is a huge challenge.

THE ISSUES....

Some issues we face include^(1,2):

1. The rising price of oil, fertilisers and pesticides, diminishing supplies of phosphorus, and a crisis in water supply. Genetically improved crops have increased yields but were successful due to access to relatively cheap oil based fertilisers, pesticides and abundant water. Monoammonium and Diammonium Phosphate, two fertilisers of choice for Australian cereal crops, more than doubled in price over 12 months prior to the financial crisis. 'Round-up' herbicide more than tripled in the same year. Phosphorus

KEY POINTS

- There is a need for incentives to farmers for sustainable practices
- The economic costs to the environment and for sustainable production needs to be internalised in food prices
- We need an increased investment in agro-ecological research
- We need to invest in economic valuation of ecosystem services

is essential for food production; however reserves will be depleted in the next 50-100 years according to Dana Cordell (University of Technology Sydney, 2008).

2. Many of our soils are tired, impoverished and need rehabilitation and there is urban encroachment onto fertile productive agricultural land. Urban expansion also drives the increasing trend for water to be moved from agricultural production to urban and industrial use.
3. Traditional low food prices have not included the cost of this environmental damage. Failure to cost and price environmental damage into the price of food will mean the natural resource base for producing more food in the future will decline.
4. Pressure to increase food production by further expansion of agriculture into rainforests, wetlands, peat lands, savannahs and grasslands will mean further loss of biodiversity at a time when the mitigation of climate change requires repair of this function and increased carbon sequestration.
5. Climate change will impact by increasing uncertainty in agricultural production.
6. The rising price of oil will continue to push the growth of bio-fuels where food producing land will be converted to bio-fuel production and further clearing of forests and natural habitat will be lost to bio-fuels.

'GREEN REVOLUTION' FADING

Climate change creates increased production risk in many farming systems through affecting productivity via changes in temperature, precipitation, carbon dioxide fertilisation, climate variability and surface water runoff^(1,2). The distribution of plants, invasive species, pests and disease systems may also be affected^(1,2).

Williams and McKenzie^(1,2) report that the 'Green Revolution' in the 1960's, based on high input systems sustained by a suite of new seed varieties, pesticides and fertilisers, was thought to be the solution. However, the productivity of many of these systems is being undermined by pollution, soil degradation and

pest and weed build-up^(1,2). Today, billions of hectares of land (and its people) are affected by significant land degradation via salinity, erosion and acidification⁽³⁾. Salinisation alone affects more than 2.1 million hectares of agricultural land⁽³⁻⁵⁾. Although there is an urgent need to increase productivity, the land we rely on for food production is under threat.

WHAT CAN BE DONE?

The cost of maintaining and improving the natural resource base in agricultural systems has to be included in the price of food for farmers to be able to farm sustainably and profitably^(1,2).

We need governments to adopt policies that create incentives for sustainable practices and result in costs to the environment being internalised^(1,2). This may mean dearer food, but it will also mean ensuring that we can continue to produce enough food.

We need increased investment in agricultural and agro-ecological research^(1,2). The focus in agricultural science needs to shift from only production to hydrological, ecological, and energy systems as a whole (a core message from the recent International Assessment of Agricultural Science & Technology report⁽⁶⁾).

We need investment in the economic valuation of ecosystem services^(1,2). With a market for these services, farmers in the future will be paid for the goods they produce as well as for the services they deliver through the management of healthy landscapes, rivers, wetlands and estuaries for the public good.

FOOD PRODUCTION SENATE

In August 2010, the Select Senate Committee on Agricultural and Related Industries released its report on Food Production in Australia⁽⁷⁾. Their goal was to investigate how food is produced that is affordable, viable for farmers and environmentally sustainable. Recommendations made by the Committee highlighted the importance of ownership of commercial agricultural, pastoral land and water by sovereign-owned companies for future security, investment in agriculture research,

and re-establishment of the Committee to 'further examine issues relating to food production, including the implications of any proposed emissions trading scheme for affordable, sustainable food production and viable farmers' (5, p ix).

The Committee View (see Table 1) and the issues raised by the report are consistent with concern examined by myself⁽²⁾ and others but the recommendations are somewhat disappointing. The hard work to be done is to produce greatly increased amounts of food which has high nutritional value AND at the same time does no further damage to land, rivers, oceans and their biodiversity. It is the damage to ecological systems that must stop because to keep producing from the planet's ecosystems require them to be healthy and fully functional. This emphasis is lost to a large extent in the Senate report. The emphasis of the report is on how to increase production with little emphasis on how to do that and keep the ecosystems from which production comes in healthy functional condition.

The Committee recommended the establishment of a broad strategic food plan to encompass these complex issues but also stated that some issues needed to be explored in more depth than the timing of the report submission allowed.

CAN WE SECURE OUR FOOD WHILST MAINTAINING OUR ENVIRONMENT?

'Governments around the world, including Australia's, must plan for the food needs of the population into the long term future. Such planning should begin in earnest as of now.' (7,p14)

We must learn better to farm without harming. Past efforts in agricultural science have not included adequate attention to a more holistic integration of natural resource management with food and nutritional security. Australian agricultural science can contribute significantly to international leadership and could contribute much to the global problem we now face.

For further information see www.wentworthgroup.org, and in particular:

<http://www.wentworthgroup.org/uploads/Speaking%20Notes-Our%20Food%20%20versus%20Our%20Environment%20250610.pdf>

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TABLE 1: THE COMMITTEE VIEW ⁽⁷⁾

1. The global community faces an enormous challenge to feed itself by the middle of this century as the demand for food increases significantly, perhaps doubling, while our capacity to produce food is constrained by water scarcity, declining arable land, declining nutrient inputs, declining agricultural research and development and deteriorating climatic conditions in key food growing regions of the world. If the challenge is not met, the consequences for global peace and security could be grave and Australia will not be immune.
2. From Australia's perspective, it is imperative that we maintain a productive base capable of meeting the food needs of the domestic population to ensure food security in the event that other countries become unwilling to trade food grown within their borders. Even more important, however, is the need for Australia, as a major food exporter, to contribute to meeting the global food task and thereby prevent the potentially disastrous consequences of major food shortages.
3. The committee is therefore of the view that governments around the world, including Australia's, must plan for the food needs of the population into the long term future. Such planning should begin in earnest as of now. The views expressed by the committee in the remainder of this report reflect changes to our current approach to agricultural food production that must occur if Australia is to meet its food production objective of producing food that is affordable and can be produced viably by farmers in an environmentally sustainable way.

The National Heart Foundation of Australia puts forward its position and recommendations in relation to antioxidants

by Tuesday Udell



There is now a substantial body of research relating to vitamin C, vitamin E, β-carotene and polyphenols and their purported health benefits. While the scientific platform has been building public (and professional) confusion has also grown about foods traditionally thought to have antioxidant effect and their potential for cardiovascular health benefit.

The National Heart Foundation of Australia (Heart Foundation) recently released their position statement⁽¹⁾ to review the evidence on cardiovascular benefits of foods commonly understood to be sources of antioxidants and to make updated recommendations. Whereas the previous position statement focussed only on supplements, the scope of the current review was expanded to respond to enquiries received by the Heart Foundation from the public and health professionals around fruit

The Heart Foundation's review found that a balanced diet that contains a wide variety of plant-based food sources, rather than supplements, would provide nutrients beneficial to cardiovascular health⁽⁴⁾.

and vegetables, tea, coffee, cocoa, red wine and supplements. Given that it is food and not isolated nutrients that we consume, this review looked at foods and drinks traditionally thought to have a positive cardiovascular health benefit as well as supplement sources, where possible⁽²⁾. Outcomes considered were⁽²⁾:

- cardiovascular events and mortality;
- cardiovascular risk factors, such as high blood pressure, diabetes, overweight and abnormal lipid profile; and
- other clinical outcomes related to the risk of CVD, such as endothelial function, platelet function and inflammation.

An expert working group* convened by the Heart Foundation developed the evidence paper from February 2009 – November 2009. They identified studies from PubMed for all years with the exception of studies on supplements, which were searched from 2003⁽²⁾ since the Heart Foundation's previous statement included studies published up to this time. Other evidence considered included

recommendations from the Heart Foundation working group, and bibliographic references of key reviews, journal articles and related articles⁽²⁾. National Health and Medical Research Council levels of evidence⁽³⁾ were assigned where possible (see Table 2).

The Heart Foundation recommends that fruit and vegetables (including nuts and seeds) and warm black or green tea, but not more than five cups of coffee (paper-filtered, percolated, café-style or instant) per day, be incorporated into a healthy, balanced diet. High polyphenol cocoa drinks, but not chocolate, may be included⁽⁴⁾ (see Table 2).

So whilst there were some positive findings for cardiovascular health from the epidemiological evidence on fruit and vegetables and tea, consumption of coffee, red wine, dark chocolate and supplements for cardiovascular health was not supported⁽²⁾.

References (see p7)

Tuesday Udell is National Policy Officer-Food Supply at the Heart Foundation, Australia

TABLE 2: HEART FOUNDATION RECOMMENDATIONS AND EVIDENCE STATEMENTS RELATING TO 'ANTIOXIDANTS' FOR FOOD, BEVERAGE AND SUPPLEMENT CONSUMPTION FOR ADULT AUSTRALIANS AND CARDIOVASCULAR HEALTH^(4,4)

| | RECOMMENDATIONS | EVIDENCE STATEMENTS |
|-----------------------------------|--|--|
| FRUIT AND VEGETABLES | <ul style="list-style-type: none"> • At least 2 serves of fruit per day. • At least 5 serves of vegetables per day. | <ul style="list-style-type: none"> • Consuming a diet rich in fruit and vegetables causes a modest fall in systolic blood pressure in normotensive and hypertensive individuals (Level II). • Increasing fruit and vegetable consumption is associated with a reduction in CVD mortality, a reduced risk of stroke and a lower risk of coronary heart disease (Level III-2). |
| TEA | <ul style="list-style-type: none"> • Either black or green tea made with leaves or tea bags. • May add reduced, low or no fat milk. | <ul style="list-style-type: none"> • Drinking tea or consuming tea-flavonoids improves endothelial function (Level II). • There is limited evidence that drinking green tea or consuming tea-flavonoids reduces visceral fat (Level II). • Regular tea drinking is associated with a reduced risk of cardiovascular disease (Level III-2). |
| COFFEE | <ul style="list-style-type: none"> • People who already drink coffee should drink <5 cups/ day of paper-filtered, percolated, café-style or instant coffee in preference to boiled or plunger coffee. | <ul style="list-style-type: none"> • Consuming approximately five cups per day of coffee causes a small elevation in systolic blood pressure (Level I). • Boiled coffee increases low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) levels. Filtered and instant coffee have very little effect on LDL-C and TC levels (Level I). • Coffee consumption has little impact on the risk of coronary heart disease (Level III-2). • Coffee consumption is not associated with increased rates of heart failure hospitalisation or mortality, or cardiovascular mortality (Level III-2). • Coffee consumption is not associated with an increased risk of hypertension (Level III-2). • Regular coffee consumption is associated with a lower risk of type 2 diabetes (Level III-2). |
| RED WINE | <ul style="list-style-type: none"> • The amount of alcohol consumed has more impact on cardiovascular health than the type of alcohol consumed. • Healthy Australians who already drink alcohol should drink no more than 2 standard drinks per day. | <ul style="list-style-type: none"> • Conflicting and insufficient evidence exists regarding the cardiovascular health benefits of polyphenols in red wine (Level: not allocated). |
| HIGH POLYPHENOL COCOA / CHOCOLATE | <ul style="list-style-type: none"> • Use raw cocoa powder in drinks and cooking. • Most commercial cocoa and chocolate will be poor sources of antioxidants. | <ul style="list-style-type: none"> • Acute intake of high polyphenol cocoa and/or chocolate increases endothelial function (Level I). • Consuming high polyphenol (500 mg) cocoa and/or high polyphenol (500 mg) chocolate can modestly reduce systolic blood pressure (Level II). • Cocoa and/or chocolate intake reduces platelet reactivity (Level II). |
| SUPPLEMENTS | <ul style="list-style-type: none"> • Combination or individual antioxidant supplements are not recommended for the prevention of cardiovascular disease. | <ul style="list-style-type: none"> • Consuming vitamin E supplements does not decrease all-cause mortality or cardiovascular mortality, or prevent cardiovascular events (Level I). • Consuming β-carotene supplements has a small effect on increasing cardiovascular mortality and all-cause mortality in smokers (Level II). • There is no evidence for the effectiveness of combination antioxidant supplements for the prevention of cardiovascular disease (Level II). • There is conflicting evidence regarding the effect of supplemental vitamin C intake and the risk of coronary heart disease (Level III-2). |

Influence of oral fatty acid sensitivity on fat consumption

by Russell Keast



Emerging research suggests that inter-individual differences in oral sensitivity to fat may partially explain differences in fat intake and assist in the understanding of why fat consumption may be excessive in the development of overweight and obesity.

KEY POINTS

- Differences in oral sensitivity to fat may partially explain differences in fat and energy intake between people.
- Preference for fat consumption can be habituated, which means there is potential to change preference and consumption of fat.
- The link between oral fat sensitivity to consumption and BMI is a promising avenue in obesity research.

Fat perception may be influenced by olfactory, textural, nociceptive, thermal and gustatory qualities⁽¹⁾. However there is evidence that even when these factors are controlled for, fat can be detected in the oral cavity.

Oral exposure to fats, without consumption of fats, prolongs the elevation of postprandial serum triacylglycerol in human subjects consuming butter versus odour and textured controlled fat replacers⁽²⁾ and fat-free products⁽³⁾. The chemical moiety for fat taste is not the triacylglycerol, but rather free fatty acids, and there are multiple putative receptor mechanisms located on taste receptor cells that respond to fatty acids. In animals, fat taste is thought to be mediated via a range of receptors including G-protein receptors, fatty acid transporters (CD36, FAT) and fatty acid sensitive ion channels⁽⁴⁻⁷⁾.

Work with two rat strains, one classified as fat hypersensitive, the other as fat hyposensitive revealed that when exposed to a high fat diet, fat hypersensitive rats consumed less dietary fat and resisted weight gain compared to fat hyposensitive rats that consumed more fat and became obese⁽⁸⁾. In humans, Kamphuis et al⁽⁹⁾ investigated fat specific satiety by replacing habitual dietary fat with linoleic acid, gamma-linoleic acid and oleic acid, showing that subjects ate significantly less total fat at dinner following 2 weeks' consumption of oils high in linoleic acid vs oleic acid (45.0 ± 9.4 vs 48.3 ± 8.3 E%; $P < 0.05$).

Importantly, studies demonstrate that fat preference and consumption (amount and frequency) can be habituated^(10,11).

Our laboratory⁽¹²⁾ recently conducted a study in humans demonstrating an association between oral fatty acid sensitivity and lower energy, fat consumption and BMI. Fifty-four subjects were screened for oral sensitivity to oleic acid and categorised as either hypo- or hypersensitive to fat. They ranked samples of custard containing 0, 2, 6, and 10% fat, recorded habitual diet for one week day and one weekend day, and BMI was calculated from self-reported height and weight. Fat hypersensitive subjects reported mean \pm SEM energy intake of 6362 ± 452 kJ/day, whilst hyposensitive subjects reported 7695 ± 344 kJ/day ($P < 0.05$). Additionally the fat intake of hypersensitive subjects was significantly lower (51 ± 3 g/day versus 72 ± 6 g/day, $P < 0.05$). Fat hypersensitive subjects had significantly lower

BMI ($P < 0.05$) and performance ranking fat content of custard was associated with sensitivity ($P < 0.05$).

The link between oral fat sensitivity, energy and fat consumption and BMI is exciting in the context of obesity research. These data build on previous research in rat model, showing that hyposensitivity to fat is linked to consumption of fat and potentially development of overweight and obesity. The area of fat taste is a promising avenue to further explore the link between consumer preference for fat or oral fatty acid sensitivity, and its role in energy balance and body weight regulation.

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Editor's note: The National Heart Foundation of New Zealand is currently reviewing and updating their equivalent nutrition statements.

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TABLE 3: CHANGE IN WAIST CIRCUMFERENCE AND BLOOD PARAMETERS OVER TIME

| | Control n=11 | Low Fat n=36 | Reduced Carbohydrate n=37 | P |
|--------------------------|--------------|--------------|---------------------------|--------|
| Waist circumference (cm) | 1.2 (1.6) | -3.0* (2.5) | -3.3*(4.3) | <0.001 |
| Insulin (mU/L) | 5.4 (6.7) | -1.8* (3.7) | -2.5* (1.0) | <0.001 |
| HDL (mmol/L) | 0.04 (0.18) | -0.06 (0.15) | -0.01 (0.16) | 0.12 |
| LDL (mmol/L) | -0.03 (0.49) | -0.18 (0.29) | -0.01 (0.46) | 0.15 |
| VLDL (mmol/L) | -0.04 (0.14) | 0.03 (0.16) | -0.89# (0.22) | 0.03 |
| Triglycerides (mmol/L) | -0.01 (0.36) | 0.07 (0.34) | -0.16 (0.48) | 0.06 |

Data are presented as Mean (SD). Post hoc: *diff between control and active groups; # diff between reduced carbohydrate and low fat group only

Eighty-four adolescent boys and girls participated in this phase. Over the 12 week period a control group of 11 subjects continued to gain weight whilst both the low fat and reduced carbohydrate groups reduced their BMI significantly more than control ($P < 0.01$) but were not statistically different from each other (see Figure 1). Both dietary interventions also resulted in favourable and significant reductions in waist circumference and insulin levels compared to the control group (see Table 3).

THE NEXT PHASE.....

Our next phase will build our understanding of factors affecting uptake into weight management programmes, options for successful weight management to enable tailoring advice and further, how these affect metabolic and body compartment changes⁽⁷⁾.

We aim to address the gap in the evidence base for optimal weight

management interventions in obese Australian adolescents in a clinical setting. The intervention will examine effect of macronutrient manipulation on body composition and cardiovascular risk markers in adolescents presenting for weight management over 2.5 years, integrating psychological support as a group based activity, individual face to face counselling and telephone support. We are building in a longer term management phase 'Eat Smart for Success' to test whether adding pharmacotherapy can help in weight maintenance after an initial period of weight loss.

CONCLUSION

In the group of severely obese adolescents that are referred to us, the chronic and remitting course of childhood obesity and its subsequent morbidities are abundantly clear. We plan to develop and test a model of care that acknowledges the premise that childhood obesity has no 'quick fix' but requires a

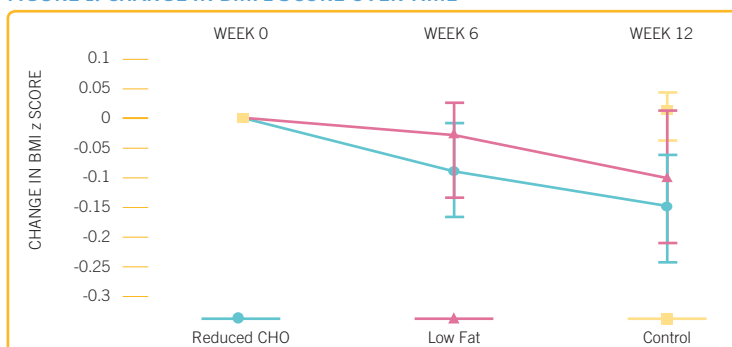
continuum of care. By building interventions that bolt onto each other we can provide a framework that supports long term engagement in treatment for the obese child and their family.

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FIGURE 1: CHANGE IN BMI z SCORE OVER TIME



Post hoc $P < 0.01$ between control and active groups

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