GI News - September 2020

GI News is published online bi-monthly (during 2020) by the University of Sydney, School of Life and Environmental Sciences and the Charles Perkins Centre, and delivered to the mailboxes of our 97,000 subscribers. Our goal is to help people choose the high-quality carbs that are digested at a rate that our bodies can comfortably accommodate and to share the latest scientific findings on food and diet with a particular focus on available carbohydrates (maltodextrins, starches, sugars), dietary fibres, blood glucose, the glycemic index and glycemic load.

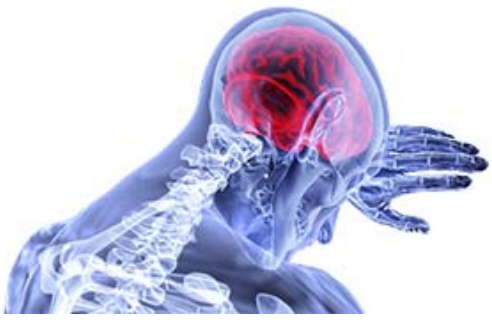
Publisher: Professor Jennie Brand-Miller, AM, FAA, FAIFST, FNSA, PhD
Editor: Dr Alan Barclay, PhD, APD

Contact GI News: glycemic.index@gmail.com

Sydney University Glycemic Index Research Service
Manager: Dr Roslyn Muirhead, PhD, APD
Contact: sugirs.manager@sydney.edu.au

FOOD FOR THOUGHT
GLUCOSE LEVELS AND DEMENTIA

Developing dementia as we age is one of our worst fears. We know people with diabetes are at greater risk (1), raising the question of whether high glucose levels are a driver of the abnormalities that are found in the brain of dementia patients. If true, it would mean glucose levels *intermediate* between healthy and indicative of diabetes, also increase the risk of developing the disease. And if glucose plays a role, then perhaps dietary changes can reduce the chances that we'll fall victim to this insidious disease.



What we already know

Most studies have revealed links between high glycated hemoglobin levels (or HbA1c - a marker of average glucose over 3 months) or after-meal (postprandial) glucose levels, but not fasting glucose levels. One of the most convincing to date was the study by Crane and colleagues published in the *New England Journal of Medicine* in 2013 (2). It was the first to examine changes in glucose levels *per se* across time and long-term follow-up of elderly people using a battery of cognitive tests.

This cohort of over 2000 Americans had no evidence of dementia at baseline when they were an average of 76 years old. Only 10% had diabetes. During the next 7 years they carefully tracked glucose levels and the development of objective signs of dementia. They assessed dementia using a screening questionnaire, followed by a large number of neuro-psychological tests. They measured glycated hemoglobin as well as fasting and random glucose levels.

Over the next 7 years, 1 in 4 of this cohort developed some form of dementia. They found higher glucose levels increased the risk in a dose-response fashion, even in those without diabetes. Glucose levels at the lower end of the spectrum – ie pre-diabetes – were implicated. The higher the glucose level, the greater the chance of a diagnosis, even after taking into account other risk factors such as blood pressure and exercise.

Some of us carry gene changes that increase the risk of dementia, particularly one form of the APOE gene, which also increases the risk of stroke and heart disease. But in Crane's study, it made no difference which form of the gene was carried.

How does glucose increase the likelihood of dementia?

High glucose levels could contribute to dementia via several mechanisms. Vascular (blood vessel) dementia is caused by problems with the supply of blood to the brain, typically a series of mini strokes. Glucose itself may have a toxic effect on vascular walls and neurones in the brain. We know that poorly controlled glucose levels increase the risk of kidney disease and blindness in people with type 1 and type 2 diabetes. And the mechanisms are likely to be the same in dementia.

This process begins with the glycation of proteins in the kidney and eye, whereby glucose molecules bind irreversibly in a way that interferes with the function of enzymes and other proteins. One particular glycated protein called beta-amyloid leads to a gradual increase in deposits in the brain that are detected using techniques such as CT and MRI. Alzheimer's dementia is characterised by widespread amyloid deposits in the brain.

Interestingly, there's a connection with diet composition. In animal models, refined carbohydrates (starches and sugars) have been shown to worsen Alzheimer's disease, although evidence in humans is lacking. However, the recent study by Melissa Gentreau and colleagues in France (3) found that an afternoon snack based on high glycemic carbohydrates was associated with more dementia, particularly in those that carried one of the high-risk APOE genes.

The French group did not find any association with the glycemic load of breakfast, lunch or dinner and dementia. The study was well designed with a large number of elderly individuals followed up for an average of 11 years. Unfortunately, observational studies such as this can't tell us whether high glucose is the cause or effect (i.e., the driver or the passenger).

We need long term randomised controlled trials to answer this question, including those in animal models of dementia. In the meantime, eating a healthy diet based on high quality carbohydrates (starches and sugars) with a low GI is the best bet for reducing risk of **all** chronic diseases.

REFERENCES:

1. Wium-Andersen and colleagues. Risk of dementia and cognitive dysfunction in individuals with diabetes or elevated blood glucose.
2. Crane and colleagues. Glucose Levels and Risk of Dementia.
3. Gentreau and colleagues. Refined carbohydrate-rich diet is associated with long-term risk of dementia and Alzheimer's disease in apolipoprotein E ε4 allele carriers.

Professor Jennie Brand-Miller holds a Personal Chair in Human Nutrition in the Charles Perkins Centre and the School of Life and Environmental Sciences, at the University of Sydney. She is recognised around the world for her work on carbohydrates and the glycemic index (or GI) of foods, with over 300 scientific publications. Her books about the glycemic index have been bestsellers and made the GI a household word.

WHAT'S NEW?

LIFESTYLE MAY HELP REDUCE THE RISK OF ALZHEIMER'S DISEASE

It's estimated that there are currently 50 million people living with dementia globally. Major risk factors for dementia include age, genetics and family history. For example, people who have a gene called APOE4 are at much higher risk of developing Alzheimer's disease – the most common form of dementia. As explained by Prof. Jennie Brand-Miller in FOOD FOR THOUGHT, there's also increasing evidence for a link between diabetes and dementia, particularly type 2 diabetes. In fact, some researchers have described Alzheimer's disease as type 3 diabetes.

The good news is that there are many things you can do to reduce your risk. In fact, evidence suggests that at least half of the risk of dementia can be attributed to lifestyle factors including diet, exercise and smoking. What's more, studies show that people at higher risk of Alzheimer's disease due to having the ApoE4 genotype may benefit even more from making lifestyle changes.

This was demonstrated in a study published last year in the *Journal of the American Medical Association* which followed more than 196,000 adults aged 60 year or older for around 8 years. They were divided into low, intermediate or high-risk categories, based on measurement of their genetic risk. The researchers then gave participants a healthy lifestyle score based on whether they smoked, drank alcohol only in moderation, ate a healthy diet and were physically active, dividing them into favourable, intermediate, and unfavourable lifestyle groups. Not surprisingly, those at higher genetic risk were more likely to develop dementia. However, the study found that in those at high genetic risk, following a favourable lifestyle reduced the risk of developing dementia by 32% compared to an unfavourable lifestyle.

In this study, a healthy diet was classified as one that included higher intakes of fruit, vegetables, wholegrains and fish but lower intakes of processed meats, red meats and refined grains. Other research has shown that a higher intake of saturated fat can increase the risk of dementia, particularly in those with the APOE4 gene, while unsaturated fat intake appears to be protective. However, the types of carbohydrate (starches and sugars) in our diet may also play a role. A 2017 study found that in older adults with normal cognitive function (meaning they didn't have dementia), those consuming a high glycaemic load diet had higher levels of amyloid plaques in their brain. Amyloid plaques are thought to play a role in the development of Alzheimer's disease. A previous study found an association between the glycaemic load of the diet, blood glucose levels and cognitive performance.

Read more:

- Association of Lifestyle and Genetic Risk With Incidence of Dementia.
- A high-glycemic diet is associated with cerebral amyloid burden in cognitively normal older adults.
- Blood glucose, diet-based glycemic load and cognitive aging among dementia-free older adults.

Dr Kate Marsh is an is an Advanced Accredited Practising Dietitian, Credentialled Diabetes Educator and health and medical writer with a particular interest in plant-based eating and the dietary management of diabetes and polycystic ovary syndrome (PCOS).
Contact: Via her website www.drkatemarsh.com.au.

PERSPECTIVES: DR ALAN BARCLAY
FOOD AND MOOD

Most of us are aware that certain foods and drinks, like chocolate, or a nice hot cuppa, can provide comfort when feeling down, but many would be surprised to learn that there is increasing evidence that the types of foods and drinks we consume on a regular basis can have a more prolonged effect on our mood and brain function.

Why? There are many possible reasons, relating to both the structure and function of our brains and nervous systems:

Fats on the brain

Did you know that the human brain and nervous system is around 60 % fat? A large proportion of the nerves that comprise the brain and nervous system are surrounded by an insulating substance known as myelin that dramatically increases the rate at which nerve impulses are sent throughout the body. Myelin itself is about 80% fat, and most of the fat is of the poly-unsaturated kind, in particular, omega-3.

Long-chain varieties of omega-3 fat from seafood (e.g., herring, mackerel, sardine, salmon and tuna) are the best for our brains and nervous system. Shorter-chain varieties like those found in certain nuts and seeds (flaxseed, canola, walnut, wheat germ and soybean oils), and margarines and oils derived from these, can be converted to the long-chain varieties by our body – though not very efficiently.

Unfortunately, most people do not eat enough essential omega-3 fat these days. Aim to eat oily fish at least 2-3 times a week and small amounts of nuts, seeds and their oils each day. If you don't like eating seafood, consider taking a fish oil supplement.

Brain function

Fuel up with smart carbs

Glucose is the preferred fuel for our brain and nervous system, with the typical adult requiring around 110-130 grams a day for optimal mental functioning. About 1/3rd of the glucose is used for brain fuel, and the rest is used for the production of amino acids, neuropeptides, and fats which in turn are used to produce essential neurotransmitters and hormones. The brain has limited ability to store glucose, and therefore optimal brain functioning is dependent upon a continuous supply. Therefore, it should come as no surprise that consuming the right type and amount of carbohydrate throughout the day is essential for everyone interested in optimal mental health and performance.

The amount of carbohydrate you need depends on many factors such as your age, gender, physical activity level, ethnic background, type of diabetes (if you have it), and medication use (if any). Needless to say that the optimal amount for you is best worked out in conjunction with your Accredited/Registered Dietitian, and other members of your health-care/performance team. As far as the type of carbohydrate is concerned, there is growing evidence that a healthy low glycemic index (GI) diet can improve mental performance, memory and mood. The average GI of most people's diet is higher than recommended (ideally, the daily average should be less than 45). Therefore, aim to eat at least one serve of low GI carbohydrate with each meal and snack (if you have them), to help reduce your diets average GI.

Increase serotonin - the "feel-good" neurotransmitter

The "feel-good" brain chemical serotonin is manufactured in the body from the essential amino acid tryptophan, along with essential nutrients like vitamin B6, vitamin C, folate and zinc. Tryptophan is often found to be low in people suffering from depression, and several of the drugs commonly used to treat depression work by increasing brain serotonin levels. Luckily, it is commonly found in high protein foods such as eggs, meat (in particular Turkey and chicken) and beans (legumes). Interestingly, high carbohydrate foods, even though they may not contain any

tryptophan, actually increase its availability, because the insulin released when carbohydrates are digested puts other competing amino acids to a different use, easing tryptophan's entry into our brains.

Do food additives affect moods?

A popular urban myth is that red cordial makes some children go "ballistic" or hyperactive (often incorrectly described as going "hypo" by some people). While food additives are rarely the cause of hyperactivity in most children, it is worth pointing out that some people (children and adults) may have abnormal reactions to natural and added chemicals in foods. For example, foods high in amines like chocolate, tasty cheese, and many strong seasonings have a negative effect on some people. Others may be sensitive to preservatives (e.g., benzoates (food additive numbers 210-218), gallates (food additive numbers 310-312) and other fat preservers (food additive numbers 319-321)), colours (e.g., Tartrazine (102) Erythrosine (127), and Brilliant Blue (133)) and flavour enhancers (e.g., glutamates (food additive numbers 620-625)). If you suspect you may be sensitive to natural or added food chemicals, discuss it with your doctor, and perhaps arrange a visit to an Accredited/Registered Dietitian with experience in elimination diets.

Don't lose your balance

While I have talked about specific nutrients and foods that may improve our mood, it is important that these are consumed as part of a healthy diet that is in line with current Dietary Guidelines.

Also, it is important to note that the evidence so far does not show that depression or other mental conditions can be prevented or "cured" by diet alone. But a healthy diet may help relieve the symptoms of certain mental illnesses; improve the effectiveness of medication for some of these conditions; and reduce the unpleasant side-effects of some of the medications used to treat these conditions. Of course, see your doctor and Accredited/Registered Dietitian for further advice.

Read more:

- Sugar for the brain: the role of glucose in physiological and pathological brain function
- Subjective Mood and Energy Levels of Healthy Weight and Overweight/Obese Healthy Adults on High-and Low-Glycemic Load Experimental Diets
- The effect of using isomaltulose (Palatinose™) to modulate the glycaemic properties of breakfast on the cognitive performance of children

Alan Barclay PhD is a consultant dietitian and chef. He is author of *Reversing Diabetes* (Murdoch Books), and co-author of 30-plus scientific publications, *The Good Carbs Cookbook* (Murdoch Books), *Managing Type 2 Diabetes* (Hachette Australia) and The *Ultimate Guide to Sugars and Sweeteners* (The Experiment Publishing).

Contact: Follow him on Twitter, LinkedIn or check out his website.

DIABETES CARE
BRAIN HEALTH

The brain is the organ in the body that scientists know the least about. What we do know is that for its size (around 1.4 kg for an adult) it uses a disproportionate amount of resources. The brain is 2% of body mass but consumes 20% of the oxygen we inhale, and this is largely spent on transmission of electrical impulses from one brain region to another or out to muscles and internal organs. In close concert to oxygen consumption in the brain is the use of glucose as the main energy source. The delivery of glucose and oxygen to the brain via the blood is tightly correlated with brain activity, and remains almost constant throughout the day and our lives. This scenario is quite distinct from organs such as muscles, the liver and fat tissue where glucose uptake is controlled by the hormone insulin. This is not to say that insulin doesn't have a role in the brain, but we will return to that story shortly.

The latest figures for Australia have two chronic brain diseases, Alzheimer's disease (AD) and Stroke, as second and fourth leading causes of death respectively (1). The main risk factor for both diseases is ageing, with the increase in human longevity afforded by modern medicine and good cardiovascular health, contributing to the rapid rise of AD as an unintentional side effect. Brain diseases can be broken down into those affecting the blood supply and those affecting the brain tissue itself. Stroke is a vascular disease and the risk factors for, and drugs effective against, are very similar to heart disease. AD is the result of the loss of brain tissue from areas that contribute to memory and spatial awareness although AD shares some of the same risk factors as the vascular diseases such as smoking, high blood pressure, obesity and diabetes (2).

Indeed, AD has been referred to by some researchers as type 3 diabetes (3). This is based on experiments that showed similarities in the downstream molecular features of AD brains and the liver, for example, in those with type 2 diabetes. It might seem strange to be talking about insulin resistance in the brain when we know that glucose uptake into the brain is independent of insulin. However, insulin still affects the utilisation of glucose within the brain and probably acts as a neuron protective factor. Although there is evidence for changes in the AD brain consistent with type 2 diabetes, it seems more likely that the disease is a culmination of small effects of many different factors and processes that include diabetes, being female and having fewer years of education (4).

In the AD brain there is build-up of two proteins called beta-amyloid and tau. Beta-amyloid was thought to be the main problem but experimental drugs that remove amyloid have so far failed to stop disease progression. This has led to researchers and drug companies to redirect their focus to tau, oxidative stress and inflammation but there is no other obvious target to date. A likely scenario is that a successful future drug will be a combination of medicines that address multiple targets including insulin resistance.

In the absence of a cure, is it possible to slow or prevent AD at present? Well not smoking, eating a balanced healthy diet, maintaining physical and mental activity into old age are all proven ways of reducing your risk. Yet, the answer may ultimately lie in your genes – individuals with one or both parents with AD have an incrementally higher risk of developing the disease themselves. In the not too distant future, your GP armed with your sequenced genome, will be able to advise you what food, drinks or exposures to avoid from an early age, and also what drugs you might need in middle to old age to slow AD. This is referred to as precision medicine and will be a common approach in the future management of all diseases.

References:

1. Causes of Death, Australia, 2018 Australian Bureau of Statistics; 2019.
2. Livingston and colleagues. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission.
3. de la Monte and colleagues. The 20-Year Voyage Aboard the Journal of Alzheimer's Disease: Docking at 'Type 3 Diabetes', Environmental/Exposure Factors, Pathogenic Mechanisms, and Potential Treatments.
4. Chami and colleagues. The rise and fall of insulin signaling in Alzheimer's disease.

A/PROF GREG SUTHERLAND is a research and teaching academic in the Charles Perkins Centre (CPC) at the University of Sydney. His research interest is neurodegenerative diseases including alcohol-related brain damage and Alzheimer's disease (AD). In the CPC he leads the 'Brain and Body' node that aims to understand how diseases like diabetes increase the risk of brain disorders such as AD.

Contact: via the CPC Brain and body node.

YOUR GI SHOPPING GUIDE
MINDFUL CARBS

Researchers have been trying to identify foods that are able to slow the decline of our brains as we age and the development of Alzheimer's disease. Berries, beans and whole grains are just 3 of the 10 foods that have been identified as brain-healthy foods in the MIND diet (a combination of the Mediterranean and Dietary Approaches to Stop Hypertension (DASH), diets) that also contain good carbs. One of the things these foods also have in common is their low GI, as you'll see below.

For people with diabetes – To those of you who count your carbohydrate intake in grams, exchanges or portions, we have included the details for each of these.

15g carbohydrate exchange – a serve containing 12-18g carbohydrate.
10g carbohydrate portion – a serve containing 7.6-12.5g carbohydrate.

Berries

Serving size, ½ cup (approximately 75g/2½ oz)

	GI	Energy	Available carbohydrate	Exchanges	Portions	Glycemic load
Blueberries	53	163kJ/ 39Cal	8.5g	0.5	1	5
Strawberries	40	79kJ/19Cal	2.9g	<0.5	<0.5	1

Beans

Serving size, ½ cup (approximately 85g/3oz)

	GI	Energy	Available carbohydrate	Exchanges	Portions	Glycemic load
Black-eye beans	33-50	422kJ/101Cal	16g	1	1.5	5-8
Butter beans	26-36	395kJ/94Cal	12g	1	1	3-4
Chickpeas	31-33	431kJ/103Cal	12g	1	1	4

Whole grains

	GI	Serving size	Energy	Available carbohydrate	Exchanges	Portions	Glycemic load
Pearl Barley	22-35	½ cup (95g/3 ½ oz)	562kJ/134Cal	24g	1.5	2	9
Bulgur, cracked wheat	46-53	½ cup (91g/3oz)	309kJ/74 Cal	14g	1	1	6-7
Brown long-grain rice	48-66	½ cup (80g/3oz)	509kJ/122Cal)	25g	2	2	13-17
Sourdough rye bread	48-57	1 slice (40g/1 ½oz)	439kJ/105Cal	18g	1	1.5	9-10

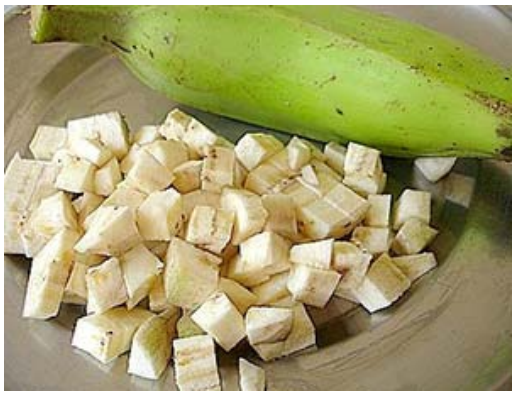
Read more:

- www.glycemicindex.com

Kaye Foster-Powell is an Accredited Practising Dietitian who has worked with people with diabetes for 30 years. She was co-author of the original series of international, best-selling books on the glycemic index. She conducts a specialized private practice for people with diabetes in the Blue Mountains, west of Sydney, Australia.
Contact: Via her website.

GOOD CARBS FOOD FACTS
PLANTAINS

I once visited a friend and found a giant bunch of what I thought were bananas on her back patio. I learned they were actually plantains. A Filipino friend had given them to her from his own garden. She was keen to give some away as she had way too many and unsure what to do with them. Well, I love a food challenge and can't bear to let food go to waste so I gratefully accepted her offer and snapped off a dozen or so from the bunch and let the World Wide Web guide me on a journey of discovery.



Plantains are also known as cooking bananas and are starchy rather than sweet. As the name suggests they are always eaten cooked. They can be eaten ripe or unripe (green) and the starchy unripe form has a neutral flavour similar to potato. They are a major staple in central and West Africa, the Caribbean, Central America and the northern parts of South America, and indigenous to tropical South East Asia. They provide a surprising 25% of the carbohydrate requirements of 70 million people in Africa alone. They are a useful food crop because they bear fruit all year.

In Africa, plantains are usually fried or roasted, while in the Caribbean they are boiled and mashed. In Central and South America plantains can be boiled and mashed, or made into chips, patties or dumplings and fried. In these cuisines, plantains provide a neutral palette on which to add flavoursome savoury dishes. In India, Indonesia and the Philippines they tend more to the sweeter side of things, such as steamed plantain and coconut cake, or simply fried and sprinkled with sugar or syrup. Plantains can also be dried and ground into flour.

Nutritionally, plantains are around one fifth carbohydrate, of which around half is starch and half is sugars. They are very low in protein and fat and a source of fibre. They also provide useful amounts of vitamin A, C, B6 and potassium. The glycemic index of plantain varies according to the cultivar, how ripe it is and how it is prepared. Unripe, green plantain is generally low GI but some cultivars can be medium or even high when boiled.

You might wonder what I did with my plantains. I went savoury with a Cayeye and Cabeza de Gato (Colombian mashed green plantain) and then sweet with Caramelised Plantains. If you're not lucky enough to have a neighbour growing them to share, you can find them in greengrocers and markets, especially in places where immigrants who traditionally eat them live.

Good Carbs Food Facts **Plantains**	
★ ★ ★ ★	
Glycemic index 39 - 66	
Serving size – 1 medium (about 89g or 3.1oz) plantain boiled in salt water for 10 minutes.	
Kilojoules	328

Calories	78
Protein	1g
Fats – Total Includes: –Saturated fat –Polyunsaturated fat –Mono-unsaturated	0.3 0.1 0.1 0.1
Saturated : unsaturated fat ratio	0.5
Carbohydrates – Total *Available* Includes: –Natural sugars –Natural starches –Added sugars –Added starches *Unavailable* Includes: –Dietary fibre	20g 18g 11g 7g 0 0 2g 2g
Sodium	206mg
Potassium	285mg
Glycemic load	10
Diabetes exchange	0.5
Ingredients: Plantain, water, salt	

Source: USDA, 2020

Nicole Senior is an Accredited Practising Dietitian, author, consultant, cook and food enthusiast who strives to make sense of nutrition science and delights in making healthy food delicious. **Contact**: You can follow her on Twitter, Facebook, Pinterest, Instagram or check out her website.

THE GOOD CARBS KITCHEN

BARBECUED SALMON AND KALESLAW WITH HONEY & LIME DRESSING

0:15 Prep • 4 Serves • Main • Every day

INGREDIENTS

4 x 100g boneless, skinless salmon fillets
2 teaspoons lime juice, plus extra lime wedges, to serve
¼ teaspoon ground coriander
¼ teaspoon ground cumin
1 x 400g packet fresh kaleslaw, dressing discarded
1 x 400g can no-added-salt chickpeas, rinsed, drained
¼ cup coriander leaves, plus extra, to garnish
Lime & honey dressing
Zest and juice of 2 limes
2 teaspoons honey
¼ teaspoon ground cumin

METHOD

Season the salmon fillets with lime juice, ground coriander and cumin. Stand for 5 minutes.

Combine kaleslaw, chickpeas and coriander in a large bowl. Make lime and honey dressing: Combine all of the ingredients in a small bowl and whisk until mixed. Pour the dressing over the salad and toss to mix well. Cover and chill.

Meanwhile, heat a barbecue hot plate or chargrill pan over a high heat. Spray salmon with olive oil and cook for 2 minutes, then reduce heat to medium. Turn the salmon and cook for a further 2–3 minutes, or until cooked to your liking.

Divide the prepared kaleslaw between four serving plates and top with the barbecued salmon. Serve with coriander leaves and lime wedges.

NUTRITION

Per serve 1828kJ/437 calories; 29g protein; 24.8g fat (includes 4.4g saturated fat; saturated : unsaturated fat ratio 0.2); 19g available carbohydrate (includes 8g sugars and 11g starch); 8.6g fibre; 89mg sodium

RECIPE AND IMAGE

Courtesy of *Australian Healthy Food Guide* magazine.

For more healthy recipe inspiration and expert advice, visit healthyfoodguide.com.au

SESAME AND MOCHI MOON CAKES

0:45 Prep • 0:20 Cook • 5 Servings • Special occasion

INGREDIENTS

Outer layer of moon cake:

1 ½ tablespoons of unsalted butter
1 tablespoon stevia or other non-nutritive sweetener
1 teaspoon matcha powder
2 teaspoons condensed milk
1 tablespoon whipping cream
1 teaspoon milk powder
1/3 cup plain flour
1 ½ tablespoon cornstarch
1 whole egg

Sesame filling:

70g cooked/canned white kidney beans
40g black sesame powder
1 tablespoon stevia or other non-nutritive sweetener
10g honey

1 tsp peanut oil

Mochi:
25g milk
10g stevia or other non-nutritive sweetener
25g glutinous rice flour
5g peanut oil

METHOD
You will need cling wrap, food processor and moon cake molds.

To make the outer layer of moon cake:

First, whisk an egg. Use a mixer to whisk the butter, stevia and condensed milk together. Add the matcha powder whipping cream into the mixture. Add the other dry ingredients (milk powder, plain flour, cornstarch) and combine well. Use a sheet of cling wrap to wrap the mixture, place in the fridge for two hours.

To make the sesame filling:

Put the sesame powder, white kidney beans, stevia, and honey into the food processor and puree the mixture. Heat a pan with the peanut oil, and pan fry the bean and sesame mixture until it turns black and shiny. Set aside and let cool. Once the mixture has cooled down, divide it into 5 serves.

To make the mochi:

Combine milk, stevia, glutinous rice flour and peanut oil in a bowl and mix well. Microwave on low power for 5 minutes or until the surface turns to a milky colour. Let cool. Divide the mixture into 5 serves.

To make the mooncake:

Whisk 1 whole egg.

Take the outer layer of moon cake out of the fridge, cut into 5 serves. Then, fold the mochi into the black sesame fillings. After that, fold the black sesame fillings into the outer layers of moon cake. Pre-heat the oven to 200 degrees. Bake the moon cakes for 5 minutes. Brush 1 tablespoon of whisked egg onto the moon cakes. Bake the moon cakes at 180 degrees for another 5 minutes. Brush the remaining whisked egg onto the moon cakes. Bake for another 3 minutes or until the surface is no longer moist. Let cool. Serve cold.

TIPS

- Stevia and other non-nutritive sweeteners do not contain added sugars, so they do not raise blood glucose or insulin levels. They are also low in calories compared to added sugars.

NUTRITION

Per serve 894 kJ/213 calories; 4.5g protein; 12.0g fat (includes 5.2g saturated fat; saturated : unsaturated fat ratio 0.76); 21g available carbohydrate; 3.7g fibre; 64mg sodium; 133mg potassium; sodium : potassium ratio 0.5

RECIPE

Shannon Shanshan Lin is an is an Accredited Practising Dietitian and Credentialled Diabetes Educator with a particular research interest in culturally and linguistically and indigenous populations. She has been actively involved in the various committees both national and internationally, including the Australian Diabetes Educators Association, Global Chinese Diabetes Association and Beijing Key Laboratory of Nutrition Intervention for Chronic Disease.

Contact: You can contact her via Wechat (ID= shannon033)

COPYRIGHT AND PERMISSION

the articles so published. GI News provides links to other World Wide Web sites as a convenience to users, but cannot be held responsible for the content or availability of these sites. All recipes that are included within GI News have been analysed however they have not been tested for their glycemic index properties by an accredited laboratory according to the ISO standards.