Australian Traditional Medicine Society FUNCTIONAL GI SYMPOSIUM 2019 was held in Sydney on Sunday 15th September.

Robert Beson presented a session *Gut Health: Environmental & Internal Profile Consideration*.

Unfortunately, there is very little information regarding his proboitic treatments.

Internet search results for “Robert Beson probiotics” or “Robert Beson gut healthy” lead to the Progurt website which sells probiotic supplements.

https://www.progurt.com.au/

The FAQ section on the website claims that “*Current laws under FSANZ, FDA and EFSA prevent publication of clinical studies supporting probiotic strain health claims.*”

In 2004, an article, *Probiotics and prebiotics in gastrointestinal disorders*, in the journal Gastroenterology[1], stated:

> The use of probiotics and prebiotics as therapeutic agents for gastrointestinal disorders is rapidly moving into the “mainstream.” Mechanisms of action explain the therapeutic effects and randomized; controlled trials provide the necessary evidence for their incorporation into the therapeutic armamentarium.

More recently in 2015 in the same journal, an article, *Prebiotics, probiotics, synbiotics, and the immune system experimental data and clinical evidence*,[2] reviewed the “recent findings relating to probiotics, prebiotics, and synbiotics, specifically their effects on immunological functions.”

There are many papers over the past 15-20 years that extol the virtues of prebiotics and probiotics including random clinic trials.

There does not appear to be much doubt that gut health is vital to our well-being.

Robert states that, “the gut is scientifically known as the second brain”.

It is the ***enteric nervous system*** that is sometimes referred to the “second brain”. The enteric nervous systems is an “apparently autonomous part of the nervous system and includes a number of neural circuits that control motor functions, local blood flow, mucosal transport and secretions, and modulates immune and endocrine functions.”

Reports from his session show dramatic improvement in health outcomes within minutes for taking a Progurt supplement.

Transit times for food that has been eaten vary greatly. It takes perhaps 1-1.5 hours for food to pass through the stomach and 2-6 hours for food to pass through the 6 metres of the small intestine. The large intestine is about 1.5 metres long.

Given that it may take 4-8 hours for the Progurt probiotic to reach the upper part of the large intestine which is where gut bacteria reside, it is difficult to comprehend how such dramatic improvements in health can result from ingesting a probiotic. It cannot be the result of change to our gut bacteria.

Prebiotics are foods that we cannot digest but feed the bacteria in the large intestine. Robert mentions “green leafy plants, herbs, vegetables, fruits and unprocessed foods”. Other important prebiotics are whole-grains and beans.

The Progurt website is a marketing website selling probiotics. Sea salt capsules are also sold. PH Caps – 105 Capsules ($59) which are “Deep Ocean Alkaline Salt Capsules”.

The 105 capsules are approximately a week’s supply with 5 taken 3 times a day.

This is 14.6g/day of salt a day with each capsule containing: Sodium 303 mg; Potassium 8.59 mg; Magnesium 28.1 mg; Calcium 6.25mg; Iron 6.25 mg; Zinc 0.0161 mg; Energy 0.00185 mg.

Whilst there is plenty of evidence regarding the importance of healthy gut bacteria, there does not appear to be much evidence for the necessity of adding sea salt to the diet.

There is a number of papers that relate an increase salt intake to a decrease in gut bacteria. This is particularly true in Japan that has a high salt intake. Potassium is an important mineral and the sodium / potassium ratio in the diet is relevant. Many fruit, starchy vegetables, leafy greens, mushrooms and beans are high in potassium.

> The combination of salt reduction and potassium increase in the diet could lead to a further decline of BP, and thus of CVD risk, in Japan and in the rest of world. [3]

It is well established that microbes in the intestines are essential for the breakdown of complex carbohydrates, the production of short chain fatty acids and synthesis of vitamins. More than 1000 different species have been identified. Despite the vast number of bacteria species and people, there are only two types of bacteriological ecosystems in the gut (enterotypes).

1. *Enterotype 1* dominated by *Bacteroides* genera bacteria which is strongly associated with high-fat, high-protein, animal-based diet

2. *Enterotype 2* dominated by *Prevotella* genus bacteria which is associated with high-fibre, plant-based diets.

Microbiome composition changed within 24 hours of initiating a high-fat/low-fiber or low-fat/high-fiber diet. However, it takes a longer period of time to change the enterotype from one state to the other.[4]

Gut bacteria dominated by *Prevotella* are associated with healthier outcomes. [5] [6] [7] [8] [9] [10]

Changes to gut bacteria change more rapidly when converting to animal-based diet than converting to a plant-based diet. Practitioner probiotics can be make the transition more rapid.

Fibre is the component of the diet that is most significantly associated with *Prevotella* species.

Animal-based diets are associated with an increase in bile-resistant bacteria which is due to the higher fat content. However, any high-fat diet, plant-based or animal-based, increases bile production.

Bile is produced in the liver which assists in the digestion of fats in the small intestine. It is stored in the gallbladder and released into the small intestine. Bile-resistant bacteria genus are *Alistipes*, *Bilophila*, and *Bacteroides*. *Bilophila wadsworthia* is associated with irritable bowel syndrome because it produces hydrogen sulphide – rotten egg gas. [11]

Lactobacillus are not native to humans. These bacteria found in yogurt, cheese and other dairy products are not essential to humans. They will not multiply in the intestine.

Choline is converted by our gut bacteria into trimethylamine (TMA) which is then converted

into trimethylamine N-oxide – $(CH_3)_3NO$ – in our liver. Trimethylamine N-oxide (TMAO) is implicated in a number of detrimental outcomes. TMAO is nasty.

> Recent human studies have established that the levels of TMAO in serum are positively correlated with impaired renal function, colorectal cancer, and cardiovascular disease (CVD). TMAO exacerbates atherosclerosis […]. In addition, TMAO exacerbates impaired glucose tolerance, obstructs hepatic insulin signaling, and promotes adipose tissue inflammation of mice maintained on a high-fat high-sugar diet.[12]

The choline in foods, such as eggs, can be turned by gut bacteria into TMA. However, it is only produced by the bacteria that are prevalent in high-fat, low-fibre, animal-based diets.

Carnitine is similar in structure to choline and the major food source is red meat. Unlike choline, which is an essential nutrient, we have no need to consume carnitine. It is also found in dietary supplements and carnitine-energy drinks.[13]

The production of TMA is absent or greatly reduced in vegans. Feeding people steak or eggs can cause an increase in TMAO within a day – but only those that have a high-fat, low-fibre enterotype.[14]

It seems pointless to continue to eat a diet that is detrimental to our health and the health of our gut bacteria then take expensive supplements to restore equilibrium. A better, healthier and much less expensive solution than purchasing supplements to ensuring our gut bacteria is optimal is to feed our bacteria with whole-grains, legumes, fruits and vegetables. Probiotics can be helpful in ensuring a rapid transition to high-fat, low-fibre enterotype.

Related articles

When Vegan Diets Do Not Work
Rheumatoid arthritis – an autoimmune condition
Eggs and the Benefits of Choline
What is the Problem with Wheat and Gluten?
The Problem With Cow's Milk
Comparison of Dairy Milks with Human Milk

Footnotes

1. Fedorak, R. N. & Madsen, K. L. (2004) Probiotics and prebiotics in gastrointestinal disorders. *Current opinion in gastroenterology*. 20 (2), 146–155.
2. Frei, R. et al. (2015) Prebiotics, probiotics, synbiotics, and the immune system: experimental data and clinical evidence. *Gastroenterology*. 31 (2), 153–158.9
3. Okayama, A. et al. (2016) Dietary sodium-to-potassium ratio as a risk factor for stroke, cardiovascular disease and all-cause mortality in Japan: the NIPPON DATA80 cohort study. BMJ Open. 6 (7), e011632.
4. Wu, G. D. et al. (2011) Linking long-term dietary patterns with gut microbial enterotypes. Science. 334 (6052), 105–108.
5. Brown, K. et al. (2012) Diet-Induced Dysbiosis of the Intestinal Microbiota and the Effects on Immunity and Disease. Nutrients. 4 (12), 1095–1119.
6. Power, S. E. et al. (2014) Intestinal microbiota, diet and health. British Journal of Nutrition. 111 (03), 387–402
7. Tang, W. H. W. & Hazen, S. L. (2014) The contributory role of gut microbiota in cardiovascular disease. Journal of Clinical Investigation. 124 (10), 4204–4211.
8. Zhu, W. et al. (2017) Gut Microbe-Generated Trimethylamine N-Oxide From Dietary Choline Is Prothrombotic in Subjects. Circulation. 135 (17), 1671.
9. Wang, Z. et al. (2011) Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. Nature. 47257.
10. Koeth, R. A. et al. (2013) Intestinal microbiota metabolism of l-carnitine, a nutrient in red meat, promotes atherosclerosis. Nature Medicine. 19 (5), 576–585.
11. David, L. A. et al. (2014) Diet rapidly and reproducibly alters the human gut microbiome. Nature. 505 (7484), 24.

12. Romano, K. A. et al. (2015) Intestinal Microbiota Composition Modulates Choline Bioavailability from Diet and Accumulation of the Proatherogenic Metabolite Trimethylamine- N -Oxide. *mBio*. 6 (2), e02481-14.
13. Koeth, R. A. et al. (2013) Intestinal microbiota metabolism of l-carnitine, a nutrient in red meat, promotes atherosclerosis. Nature Medicine. 19 (5), 576–585.
14. Tang, W. H. W. et al. (2015) Gut Microbiota-Dependent Trimethylamine Oxide (TMAO) Pathway Contributes to Both Development of Renal Insufficiency and Mortality Risk in Chronic Kidney Disease. Circulation Research. 116 (3), 448–455.