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Can Thylakoids Replace Bariatric Surgery for Long Term Maintenance of Weight Loss in Obesity Giving A More Physiological Approach

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Abstract

We reviewed the literature regarding thylakoids as the naturally physiologically occurring agents from green leafy food plants like spinach. We carried out a search in PUBMED DATABASE for detailed information using MeSH terms like satiety, fat metabolism, weight loss, glucose metabolism, lipid metabolism, gut microbiota for studying the efficacy of these thylakoids in utilizing for treatment for medical management of obesity Results- We found 70-80 articles pertaining to the same relating to obesity, diabetes, weight loss .Of these we used 65 articles excluding the duplicate articles .No meta-analysis was carried out.

Since bariatric surgery acts by acting on various targets and remain the most effective therapy for obesity management in contrast to current pharmacotherapeutic agents , use of thylakoids appears to be a promising method for tackling both obesity and preventing its associated co morbidities like type 2 diabetes mellitus(T2DM),non alcoholic fatty liver disease(NAFLD),decreasing both appetite as well as hedonic aspect of hunger reducing the cravings which make an obese patient revert to snacking in between meals and find it very difficult to control food intake simply by lifestyle interventions like diet control and exercise. Most modern medicines have not been found to be useful for long term maintenance of weight loss .In view of their cardiovascular (CVS) side effects a lot had to be withdrawn. The only drug approved is the lipase inhibitor orlistat for longterm obesity treatment which has its limitations because of steatorrhea which is very cumbersome for the patient .Thylakoids seem to be acting on multiple targets like preventing hedonic hunger ,improve fat digestion along with causing loss of weight along with fat mass as measured by DEXA,besides increasing transit time in intestine by delaying fat digestion and thus mimicking the effect of natural satiety hormones like increasing GLP1,CCK,decreasing ghrelin,an appetite stimulating hormone and thus does the work of multiple aniobesity drugs like orlistat,GLP1agonists like liraglutide ,with its probiotic effect it further promotes the weight lowering effect although whether SCFA increase is part of this mechanism is still not clear. Still AppethylTM the patented form of spinach thylakoids offers a great option for treatment of obesity and metabolic syndrome. Greater BMI experiments are needed in humans than the ones carried out till now to study if these effects seem to translate into practice in humans with morbid obesity.

Keywords: obesity; metabolic syndrome; hedonic control;satiety;GLP1;CCK,Ghrelin;probiotic effect; weight loss; fat loss

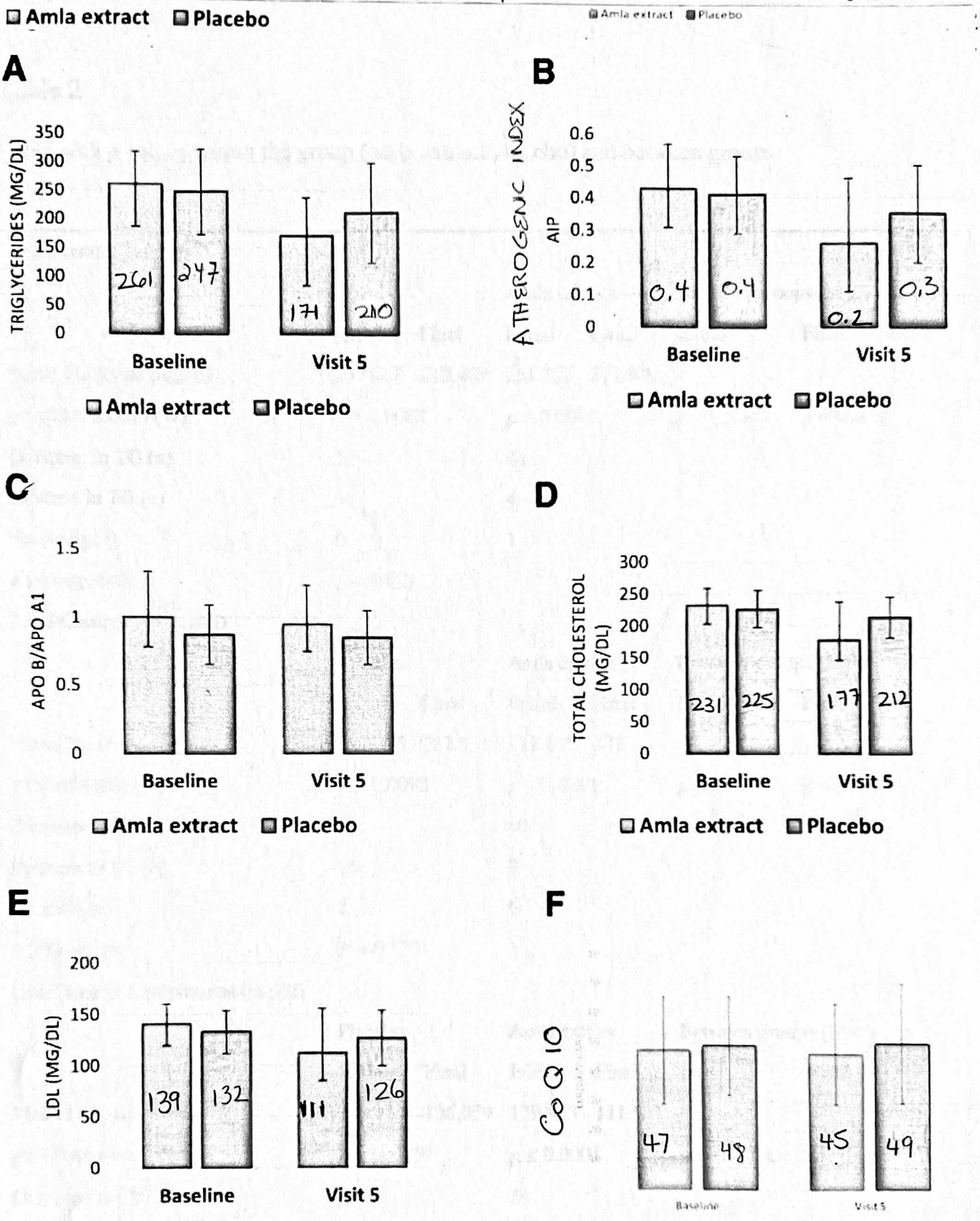
Introduction

There is an increase in an obesity epidemic currently extending globally. We had previously reviewed the aetiopathogenesis, medical treatment of obesity followed by further updates and latest the novel pathways to be targeted medically [1-8]. In this review we stress on more physiological approach in targeting various aetiopathogenetic factors by the more physiologically obtained thylakoids from natural green leafy foods like spinach which act by multiple mechanisms like acting on fat digestion, involving satiety, interacting with various factors like glucagon like peptide 1(GLP1), cholecytokinin (CCK), as well as act as probiotics along with improving glucose and lipid metabolism.

The various mechanisms which contribute to this obesity epidemic include

1. Greater consumption of energy dense and nutrient poor food which contain high levels of fat and sucrose [9]
2. Two systems apparently control appetite namely the

12 weeks



(a) Triglyceride level in amla extract and placebo group ($p = 0.0003$ between the groups); **(b)** Atherogenic index of plasma in amla extract and placebo group ($p = 0.0177$ between the groups); **(c)** Ratio of Apo B to Apo A1 at baseline and visit 5 in amla extract and placebo group ($p = 0.0866$ between the groups); **(d)** TC level at baseline and visit 5 in amla extract and placebo group ($p = 0.0003$ between the groups); **(e)** LDL-C level at baseline and visit 5 in amla extract and placebo group ($p = 0.0064$ between the groups); **(f)** CoQ10 level at baseline and visit 5 in amla extract and placebo group ($p = 0.4581$ between the groups)

Milk Consumption and Prostate Cancer: A Systematic Review

Alex Sargsyan and Hima Bindu Dubasi

Abstract

Prostate cancer is the third most common cancer in men globally, and the most common cancer among men in the United States. Dietary choices may play an important role in developing prostate cancer; in particular, a higher dairy product intake has been associated with an increased risk of developing prostate cancer. The overall positive association between milk consumption and the risk of prostate cancer development and prostate cancer mortality has been well documented in multiple epidemiological studies. However, there is limited literature on the association between types of milk, as classified by fat content (skim, low fat, and whole), and the risk of developing prostate cancer. When further examining current state of the literature on this topic, there is a number of epidemiologic studies assessing the relationship between prostate cancer and milk consumption. On the contrary, very few experimental studies explore this topic. Further experimental research may be necessary to examine the relationship between dairy and dairy products consumption and the increased risk of development of prostate cancer. At this time, there are no formal clinical recommendations regarding dairy products consumption for patients who are at risk of prostate cancer development or who have a history of prostate cancer. In this manuscript, we sought to systematically review the existing literature on the association between milk consumption classified by fat content, and the risk of developing prostate cancer. These findings may be useful for the clinicians who provide recommendations for the patients at risk of developing prostate cancer.

Keywords: Carcinogenesis, Diet, cariogenic, Diet, western, Prostatic neoplasms

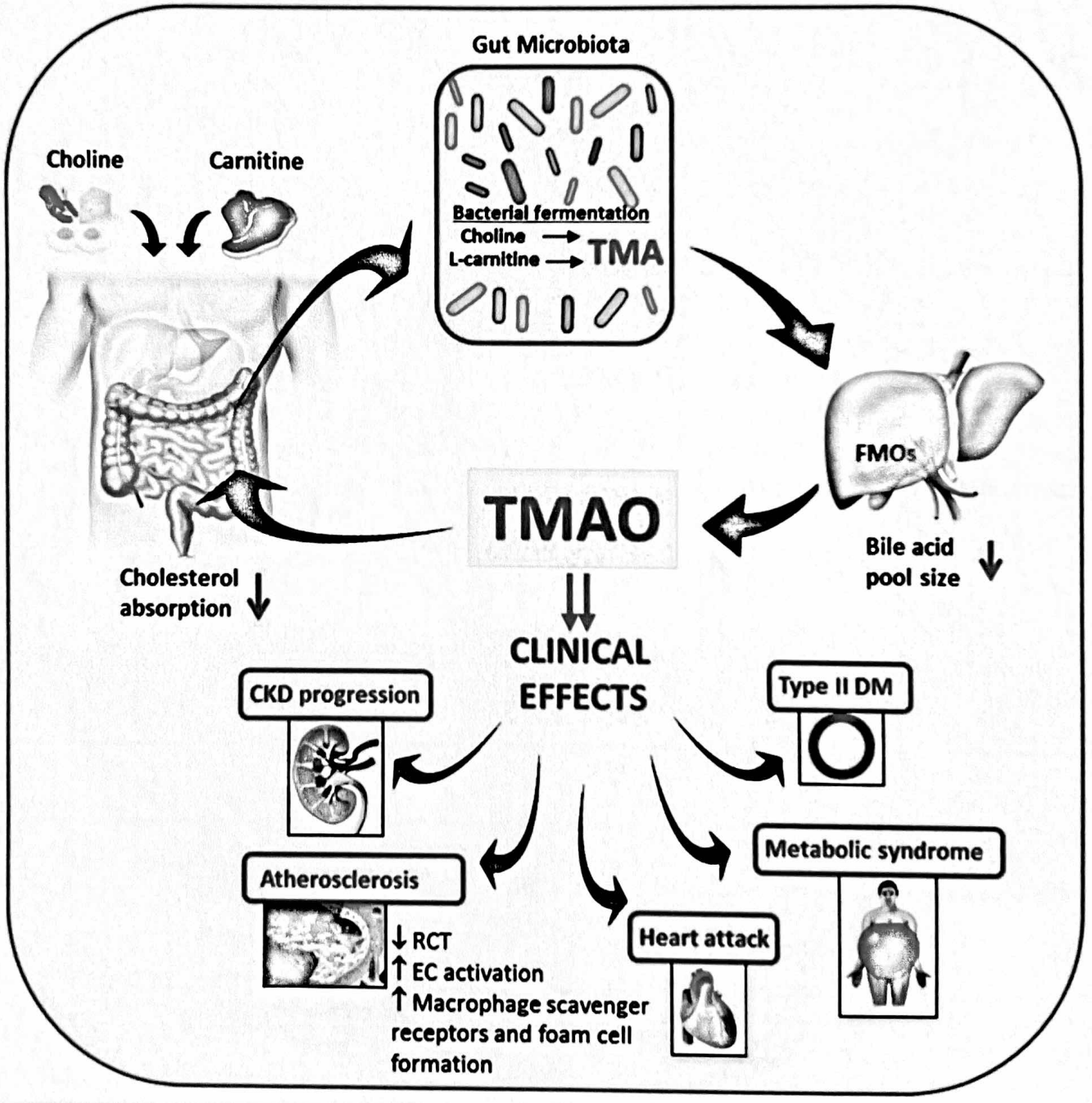
INTRODUCTION

Despite treatment advances, prostate cancer related mortality rates remain high in the United States. While surgical treatments may play a significant role in reducing a disease progression, prostatectomy may not significantly decrease the mortality related to a localized prostate cancer when compared to an observational approach [1]. Global cancer incidence estimates reveal that, prostate cancer has become the third most common cancer in men, and half a million new cases are being reported every year [2]. The global burden of prostate cancer is going to be 1.7 million new cases by 2030 [3]. According to the Center for Disease Control, prostate cancer is the most common cancer among men in the United States and one of the leading causes of cancer deaths among men of all races. Prostate cancer mostly occurs in the elderly people with three quarters of the cases occurring in men who are above 65 years of age, which is suggestive of the fact that, developed countries having a higher proportion of elderly population report a higher incidence rate of prostate cancer (≈15%), compared to the developing countries [2]. Incidence rates in the developing countries are continuing to increase, while mortality rates are staying at a relatively stable level [4].

A review of prostate cancer incidence data from 42 countries suggested that there may be a correlation ($r=0.711$) between milk consumption and prostate cancer incidence [5]. It has been observed that total dairy product intake and calcium from the dairy products has been positively associated with the risk of developing prostate cancer and it was observed that the low fat milk intake was associated with greater risk of non-aggressive form of the disease and whole milk was associated with greater risk of fatal prostate cancer [6]. The association between the risk of developing prostate cancer and dairy products has been linked to the fact that dairy products raise the concentrations of insulin like growth factors [7]. The high fat content in the dairy products has been associated with c-peptide concentration which leads to development of aggressive form of prostate cancer [8]. Metanalysis conducted by Lu and colleagues [9] suggested that increased whole milk consumption may contribute to higher prostate cancer mortality rate (2016). Another metanalysis suggested that high intake of whole milk and low fat milk may contribute to an increased risk to develop prostate cancer [10]. A study of Swedish men suggested that there is an association between whole milk consumption and cancer progression; also, this study suggested that low-fat milk intake may reduce mortality in patients with localized prostate cancer [11]. While the data suggesting that whole milk may contribute to prostate cancer development may appear rather convincing, the effects of skim and low-fat milk are not clear.

TMAO: Why It Increases & Factors That May Reduce TMAO levels

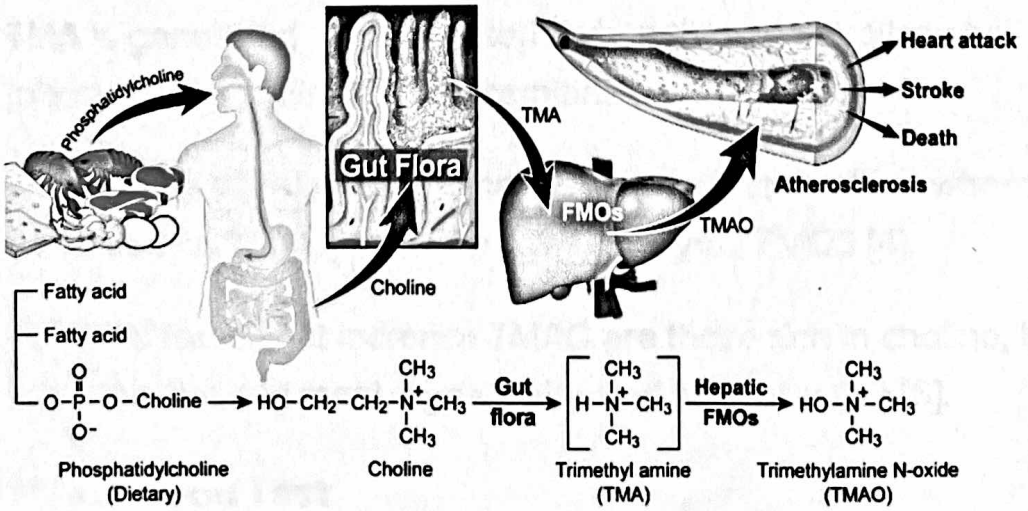
Medically reviewed by Biljana Novkovic, PhD, Puya Yazdi, MD | Written by Carlos Tello, PhD (Molecular Biology) | Last updated: January 22, 2021



CONTENTS

TMAO (trimethylamine N-oxide) is produced by gut bacteria from choline, lecithin, and L-carnitine-rich foods (mainly fish, meat, egg, and dairy). High levels are associated with heart disease, hardening of the arteries, diabetes, and colon cancer. However, the role of TMAO in these diseases is still controversial. Read more to find out how TMAO levels may affect health and what factors may lower its levels.

What Is TMAO?



Taking a Closer Look at TMAO

What Is TMAO?

TMAO (or trimethylamine N-oxide) is a metabolite produced by gut bacteria. Briefly, nutrients such as phosphatidylcholine (also known as lecithin), choline, and L-carnitine are abundant in animal-derived products such as red meat, egg yolk and full-fat dairy products. When consumed, these nutrients are processed by gut bacteria resulting in the release of various metabolites including TMA (trimethylamine) into the blood. TMA is then transported to the liver where it is converted into TMAO which has been shown to regulate various physiological processes involved in the development of atherosclerosis[1,2].

What foods are rich in dietary TMAO precursors?

Red Meat	Full-Fat Dairy Products	Others
Beef	Whole milk	Energy drinks
Pork	Eggs	Dietary supplements
Ham	Yogurt	
Lamb	Cream cheese	
Veal	Butter	
Processed meats		

What dietary modification may help reduce an elevated TMAO?

The composition of the diet can have a dramatic effect on the composition of the gut microbiome. Through dietary modifications, including the elimination of TMAO precursors, the gut bacteria may be altered and TMAO levels reduced. Foods commonly found in the Mediterranean diet such as cold-pressed olive oil, balsamic vinegar, and red wine are rich in the compound DMB (or 3,3-dimethyl-1-butanol), which has been shown to inhibit TMAO production[3].

My patient is taking a fish oil/krill oil supplement, will it falsely elevate their TMAO results?

To date, we know that TMAO is found in high levels in certain types of seafood. A comprehensive list of the contents in supplements are rarely listed, so it is possible that TMAO may be present in fish oil/krill oil supplements. To avoid potential false elevations in TMAO results, we recommend the patient abstain from eating fish and taking a fish oil/krill oil supplement 24 hours prior to their blood draw for the TMAO test.

If a patient has an elevated TMAO and they modify their diet, how long should the physician wait to repeat the test?

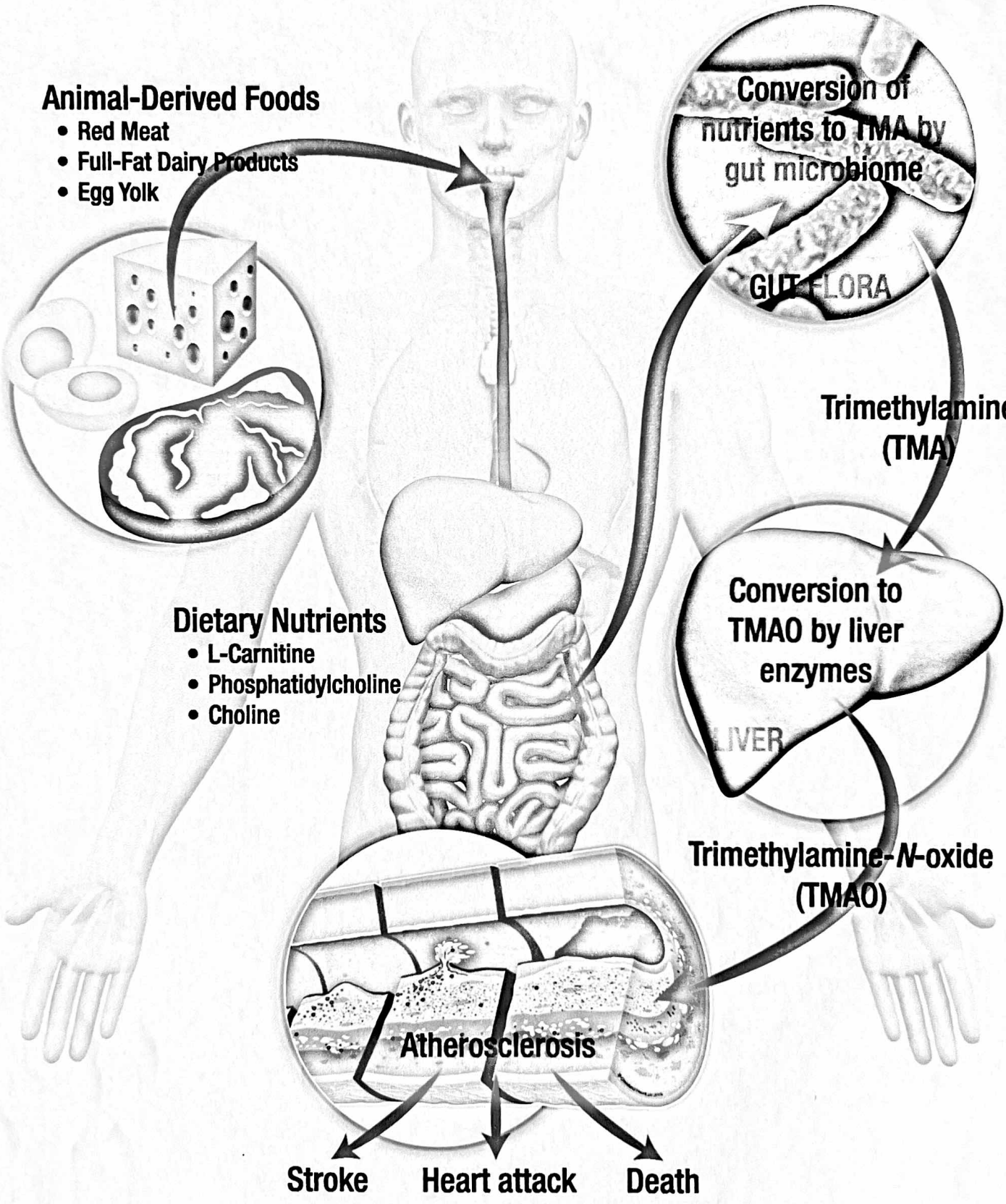
To date, there is no evidence published demonstrating how long it takes to lower baseline TMAO levels. Dramatically changing the gut microbiome through diet alone may take weeks to months. Therefore, a meaningful shift in the gut community affecting TMAO production may take weeks to months as well. We are currently suggesting a 3 to 6 month follow-up time, but this may change as we gather more information.

If a patient begins taking pre/probiotics, how long until they change their gut bacteria?

Similar to dietary modifications, pre/probiotics may take weeks to months to make a meaningful shift in the gut bacteria community. Pre/probiotics have been proven to help maintain a diverse 'healthy' gut[4], but have not been scientifically shown to reduce TMAO levels.

Is TMAO a marker for Small Intestinal Bacterial Overgrowth (SIBO)?

To date, the literature has not demonstrated a link between TMAO and SIBO. There are many types of disorders that fall under the umbrella of gut dysfunction. SIBO is a type of gut dysfunction and is primarily attributable to GI nerve dysregulation. Excess TMAO production is a type of gut dysfunction indicating the intestine (primarily large intestine) has excess bacteria capable of producing TMAO. The TMAO test does not indicate the location of TMAO producing bacteria in the gut, only the presence.



References

1. Wang Z et al. Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. *Nature*. 2011; 472: 57–63. 2. Koeth RA et al. Intestinal microbiota metabolism of L-carnitine, a nutrient in red meat, promotes atherosclerosis. *Nat Med*. 2013; 19: 576-585. 3. Wang Z et al. Non-lethal Inhibition of Gut Microbial Trimethylamine Production for the Treatment of Atherosclerosis. *Cell*. 2015; 163: 1585-1595. 4. Bäckhed F et al. Defining a healthy human gut microbiome: current concepts, future directions, and clinical applications. *Cell host & microbe* 2012; 5: 611-622.

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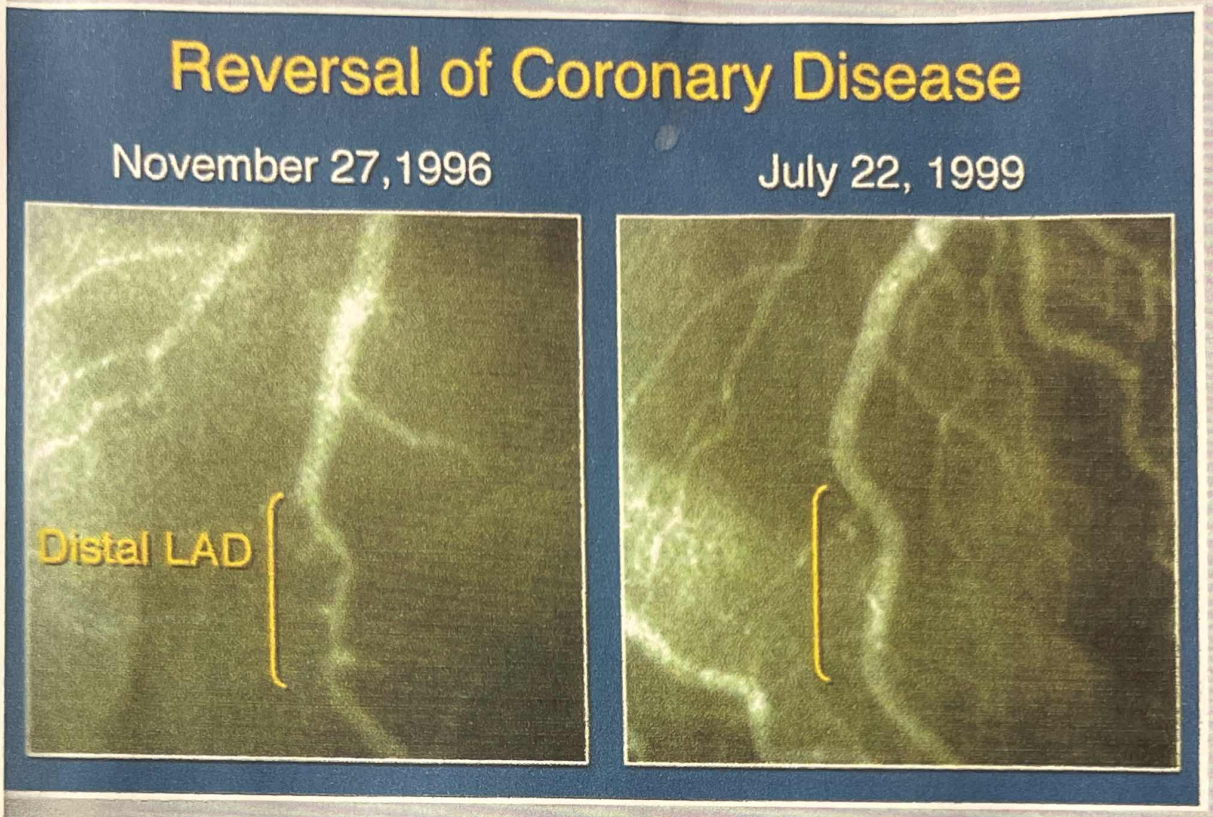

FIGURE 1 *Coronary angiograms of the distal left anterior descending artery before (left bracket) and after (right bracket) 32 months of a plant-based diet without cholesterol-lowering medication, showing profound improvement.*

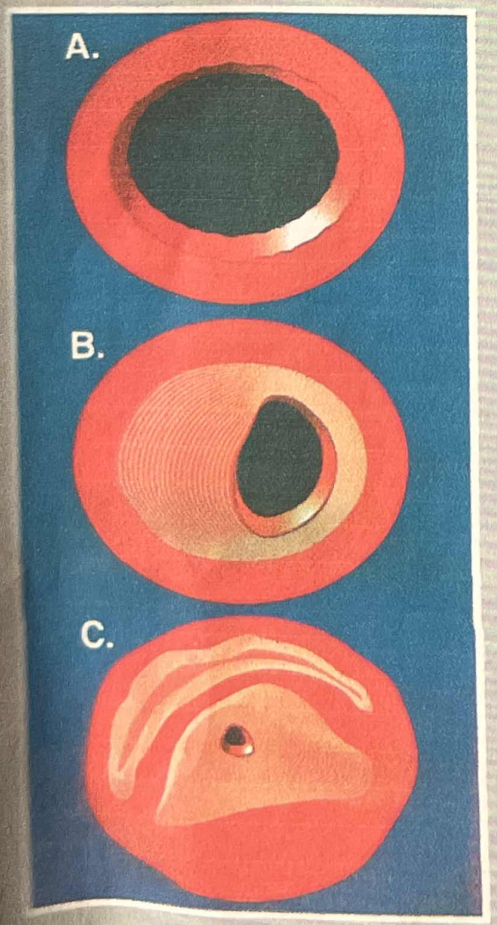

FIGURE 2 *Gradually progressive coronary artery narrowing, which accounts for 12.5 percent of heart attacks.*