

Accelerated Cure Project for MS

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Accelerating research towards a cure for multiple sclerosis

Modifiable Dietary Factors and MS

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Within the last several years, there has been a steady stream of scientific publications focusing on modifiable factors, whether behavioral or lifestyle, on the pathoetiology of MS. Modifiable factors are things we can change, and therefore research on these agents is exceptionally important and particularly of interest to everyone affected by chronic conditions – we all want to know what we can personally do to improve our health. However, understanding the role of non-modifiable factors is critical to understanding the biological mechanisms contributing to onset and outcomes (i.e. genetic analyses to uncover biological processes involved in rapid disability accrual – which may eventually lead to improved screening tests and interventions for those with the greatest risk of severe disability). This month two papers related to diet, which is becoming a popular avenue of research in MS, were published.



www.sansar.org/health_information/risk-factors

The first study, by Tankou et al, published in the *Multiple Sclerosis Journal*, was a pilot study on the influence of probiotics in MS.¹ This study compliments several studies investigating the gut microbiome (e.g. those reported on in the [September 2017 Newsletter](#)). There is enough gathering evidence suggesting the microbiome plays a role in MS risk and progression, but the *how* and *why* are still fuzzy. We also do not know if we can sufficiently alter our microbiome in order to affect some sort of change. For example, can we take a probiotic to alter our gut microbiome? This pilot study of 9 MS subjects and 13 unaffected controls adds to the beginning conversation in MS. All study participants were given a twice daily, oral dose of a probiotic cocktail VSL3 for two months. The daily dose included **3.6 trillion** cells of eight bacteria. Prior studies of VSL3 suggest it may have anti-inflammatory properties and promote neuroprotection, but little is known about the influence on circulating immune cells. First, administration of VSL3 did alter the composition of the gut microbiome (detected in fecal samples) in unaffected controls, and a similar (though not statistically significant trend) was observed in the MS subjects. Upon discontinuation of the VSL3 probiotic, the gut microbiome shifted back to baseline patterns in all study participants. Second, the researchers looked at the composition of circulating immune cells at baseline, during probiotic supplementation, and a few months after the study had stopped. The frequency of several different immune cells were influenced – generally, there were fewer inflammatory cells during supplementation. For a study of 22 individuals, these interesting results beg further investigation in a larger sample size, as well as if other strains of probiotics show similar results, whether disease modifying therapies influence gut microbiomes, and so forth.



Grapefruits are one of my favorite fruits. As a child, I would slice a grapefruit in half and top it with sweetened condensed milk... it's a Caribbean thing, but I haven't done this in decades ㄟ(ツ)_/-. The second study, by Wang et al, published in the *Journal of Nutritional Biochemistry*, focused on the effect of naringenin in the mouse model of MS (experimental autoimmune encephalomyelitis [EAE]). Naringenin is a common flavonoid in tomatoes, and citrus fruits, including grapefruit. Up to **10%** of a grapefruit is naringenin. Prior research in animals and immune cells suggests naringenin may influence the immune system. For this study, there were two groups of EAE mice: 1) fed a normal diet, and 2) fed a normal diet supplemented with a low dose of naringenin. The first result was to determine if naringenin influenced the onset of EAE/MS in the mice – and it did! Only 63% of mice on naringenin developed EAE, while 100% not on naringenin developed EAE. Also, for the mice on naringenin that did develop EAE, their onset was delayed and the disease course was milder than

the untreated counterparts (Figure 1). Naringenin also reduced the development of autoreactive T-cells – those pesky immune cells that attack myelin. In addition, the research showed that treated mice had less inflammation and myelin destruction in their central nervous tissue, as well as less invasion of immune cells into the central nervous system. And lastly, the research showed that naringenin slowed the disease course in mice treated **after** the onset of EAE (Figure 2). For an animal study, this is very exciting. Now we need to study naringenin in humans. Key questions would be: Does it work in humans? If so, what is a safe dose? What does it do in a human body? What genes are activated? Nonetheless, it was a very cool mouse study.

In summary, both these studies address **potential** modifiable dietary opportunities in MS – they generate many intriguing questions, and will serve as a scientific foundation for many future studies.

(Grapefruits contain a compound, furanocoumarins, that blocks an enzyme CYP3A4 needed to metabolize several drugs.³ Speak to a health professional before you add grapefruit-condensed milk combos to your diet!)

1. <https://www.ncbi.nlm.nih.gov/pubmed/29307299>
2. <https://www.ncbi.nlm.nih.gov/pubmed/29331869>
3. <https://www.drugs.com/article/grapefruit-drug-interactions.html>

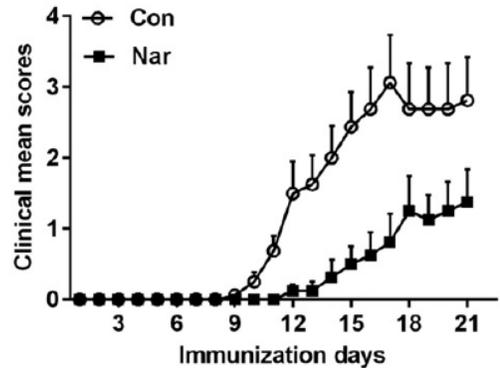


Figure 1: Early administration of naringenin delayed the onset of EAE and the severity in EAE mice.

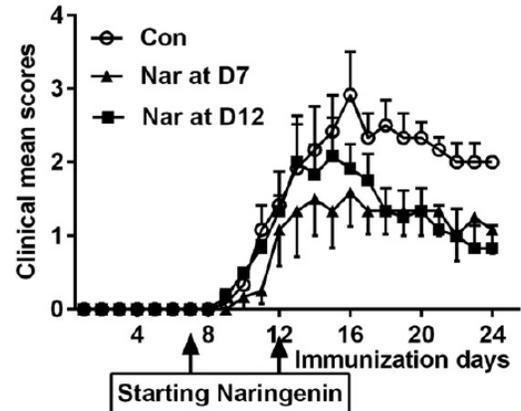


Figure 2: Dietary naringenin administered after EAE onset, on day 7 and day 12 and resulted in reduced severity.