

# How to Reduce the Effect of Western Diet Induced Worldwide Escalation of Neuropsychiatric Diseases Along With the Obesity Epidemic Secondary to Imbalanced Fatty Acid Metabolism

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Our dietary habits, in addition to the dietary pattern make up have been realized to be strong modifiers with regards to brain health. Over the last few years lot of information has been gained regarding the critical role of the trillions of bacteria that our gastrointestinal (GIT) hosts. A bidirectional gut –brain crosstalk occurs via a complicated communication system involving sympathetic nervous system (SNS) along with parasympathetic nervous system (PNS), hypothalamus-pituitary adrenal (HPA) axis along with enteric nervous system and vague nerve .Further enter endocrine cells liberate variety of hormones like GLP1, PYY, CCK and serotonin, that have influence on nutrient absorption, metabolism and appetite, with manipulation of anxiety like behaviors [1-3].

Recently it has been realized that changes in the gut micro biota (GM) ecosystem, might be associated with altered metabolism of dietary fibres altered production of eicosanoids and n6 PUFA metabolism through ALA[reviewed in 4]. This GM landscape can get drastically altered by dietary fats ,or even in the reverse direction by lipids of various kinds like advantages of n3polyunsaturated fatty acids( PUFAs) while disadvantage of n6 –PUFAs . Saturated fatty acids (SFA) can result in dysbiosis along with tendency towards neuropsychiatric diseases (NPD), with selected lipids like n3 PUFAS along with their metabolites can offer disease resilience or help in resolving the systemic and brain inflammation that has been found to be involved in schizophrenia (SCZ), autistic spectral disorders (ASD) along with depression pathogenesis. There is lot of proof that aberrant escalation of n-6:n3 ratio has a main part among dietary lipids, disturbed GM community along with chances of NPD. Further an interesting correlation is there among n3 PUFAS amounts ,GM diversity along with short chain fatty acids( SCFA) synthesis [4].

Greater circulating amounts of DHA was seen to positively associate with greater micro biome diversity along with greater amounts of Lachnospiraceae bacterial family ,unrelated to the dietary fiber consumption or not. Knowing that Lachnospiraceae family is a significant generator of SCFA, this study pointed to a an extra mode beneath the Association of n3 PUFAS amounts,

GM health in addition to decreased chances of NPD. Further depression-like behaviors evoked in mice via social isolation were observed to switch the GM composition but further reduce the SCFA synthesizing bacteria (like Allobacterium)that was sensitive to DHA dietary supplementation[5].

A lot of proof for many associations among unbalanced intake of selected dietary FA's to the higher chances of NPD's .More information of mode of connection among dietary lipids ,aberrant GM and alterations in neuron active compounds mainly dopamine(DA) along with serotonin ,more knowledge on how we can design innovative strategies for NPD pathogenesis in the form of probiotics containing say Bifidobacterium longus that might generate more butyrate from dietary fibres which help by escalating butyrate synthesis which through epigenetic changes like acting as histone deacetylase (HDAC) Inhibitors help in decreasing dose or might be omitting gradually the antidepressants like serotonin noradrenaline reuptake inhibitors inhibitors( SSRI's) ,monoamine oxidase (MAO) inhibitors .With activation of gene transcription by prevention of histone acetylation. Further HDAC Inhibitors extend their action on NF (BDNF up regulation) in hippocampus and prefrontal cortex(PFC) ,thought to cause neuroplasticity and aiding in antidepressant action of imipramine also. Avoidance of Western Diet rich in SFA, refined grains, corn generated fructose ,proteins from red meat changes the GM, like decreased F.prausnistzi and increased invasive bacteria like E.Coli and dramatic fall in SCFA(main BA),besides associated with enhanced intestinal permeability [6-7] .

Currently emphasis made on inflammation resolution along with n3 PUFAs obtained lipids known as ‘‘ specialized proresolving mediators’’ (SPM) made of lipoxins, resolves, protectins and maresins [rev in 3 and unpublished observations by us]. Deficiency of n3 PUFAs has been constantly documented in SCZ patients, bipolar disorder pts along with depression, although no evidence that eicosapentaenoic acid( EPA) along with docosa hexaenoic acid(DHA) supplementation in ASD cases

helped. Thus simple dietary changes might go a long way in even preventing microglial activation in new borns through neuro inflammation as shown by maternal immune activation [8-10]. Further evaluation is needed in these lines with lot of patients presenting with IBD, depression like symptoms who have had EEG, endoscopy for this common basis of micro biota-brain communication.

## References

1. Collins SM, Surette M, Bercik P. (2012) The interplay between the intestinal microbiota and the brain. *Nat Rev Microbiol* 10(11): 735-742.
2. Kulvinder KK, Allahbadia GN, Mandeep S. (2019) Have Probiotics and Synbiotics passed the test of time to be implemented in management of obesity and related metabolic disorders-a comprehensive review. *Adv Obes Weight Manag Control* 9(1):21–28.
3. Marrone MC, Coccorello R. (2020) Dietary Fatty Acids and Microbiota – Brain Communication in Neuropsychiatric diseases. *Biomolecules* 10(1):1-12.
4. Menni C, Zierer J, Pallister T, Jackson MA, Long T, Mohny RP, et al. (2017) Omega -3- fatty acids correlate with gut Microbiome diversity and production of N-carbonyl glutamate in middle aged and elderly women. *Sci Rep* 7(1):11079.
5. Davis DJ, Hecht PM, Jasarevic E, Bevders DQ, Will MJ, et al. (2017) Sex specific effects of docosahexaenoic acid(DHA,) on the Microbiome and behaviour of socially isolated mice . *Brain Behav Immunol* 59(2): 38-48.
6. Bercik P, Pibark AJ, Sinclair D, Khoshdel A, Huang X, et al. (2011) The anxiolytic effect of Bifidobacterium longum NCC3001 involves vagal pathways for gut-brain communication . *Neurogastroenterol Motil* 23(12):1132-1139.
7. Wu X, Chen PS, Dallas S, Wilson B, Block MI, et al. (2008) Histone deacetylase inhibitors upregulate astrocyte GDNF and BDNF gene transcription and protect dopaminergic neurons. *Int J Neuropsychopharmacol* 11(8):1123-1134.
8. Bozzatello P, Brignolo E, DeGrandi E, Bellino S. (2016) Supplementation with omega -3 fatty acids in Psychiatric Disorders:A review of literature Data. *J Clin Med* 5(8):67.
9. Chriett S, Dabek A, Wojtala M, Vidal H, Balcerczyk A, et al. (2019) Prominent action of butyrate over  $\beta$ -hydroxy butyrate as Histone deacetylase inhibitor, transcriptional modulator and anti-inflammatory molecule. *Sci Rep* 9(1):742.
10. Bilbo SD, Block CL, Bolton JL, Hanamsagar R, Tran PK. (2018) Beyond infection-Maternal immune activation by environmental factors , microglial development, and relevance for autism spectrum disorders. *Exp Neurol* 299:241-51.