Inflammation and diet: Focus on mental and cognitive health

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Abstract

It has been well established that chronic low-grade inflammation is implicated in both physical and mental noncommunicable diseases. Diet, a leading risk factor for non-communicable diseases, has been repeatedly shown to be related to inflammation, as well as various health outcomes, including mental and cognitive health. In the current editorial paper, we briefly summarize the current state of evidence and discuss the potential mediating role of inflammation between diet and mental/cognitive health. We also outline our perspective on challenges and future research directions in the domain of inflammation and diet, with a specific focus on mental and cognitive health.

Key words: inflammation, diet, nutrition, mental health, cognitive health

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Systemic chronic inflammation, diet and health

Chronic low-grade inflammation is implicated in the etiology of various noncommunicable diseases, including diabetes,[1] obesity,[2] cancer,[3] and cardiovascular diseases,[4] and even increased mortality.[5,6] Additionally, inflammation is also implicated in numerous mental disorders. For instance, meta-analytic and systematic reviews that focused on observational evidence have shown that a range of blood and cerebrospinal fluid pro-inflammatory markers such as interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor alpha (TNF-α), and C-reactive protein (CRP) were increased in a range of mental illnesses including depression, anxiety, schizophrenia, and bipolar disorder.[7–11] Large cohort studies have also shown elevated CRP levels and white blood cell counts in depressive and anxiety disorders.[12–16]

In terms of cognitive health, evidence from cross-sectional and prospective studies indicate that peripheral inflammatory markers, such as IL-6 and CRP, are associated with the global cognitive decline,[17–20] and a decline in specific cognitive domains, particularly short-term memory,[21–23] processing speed,[24] verbal fluency, and executive function.[25–27] For neurodegenerative conditions, meta-analyses of prospective studies have reported that higher levels of CRP and IL-6 are associated with a higher risk of all-cause dementia.[28–30] While inflammation was not associated with an increased risk of Alzheimer's disease (AD), evidence from cross-sectional studies indicates that peripheral markers of inflammation such as CRP, IL-1β, IL-6, and IL-8 are significantly higher in AD patients compared to controls.[31–33] Elevated peripheral inflammatory markers such as monocyte chemoattractant protein-1 (MCP-1), IL-6 and IL-8 have been reported in mild cognitive impairment,[33] but this finding is not consistent,[31,34] and it may be that systemic inflammation is similar to that of healthy individuals and occurs at a later stage in the progression of AD.[35] Additionally, due to kynurenine pathway enzymes directly influencing inflammation and the immune system, kynurenic acid was shown to be lower in individuals with neurodegenerative conditions.[36,37]

The aforementioned adverse physical, mental and cognitive health outcomes are also known to be associated with poor diet quality.[38–40] Diet, a leading risk factor for noncommunicable diseases, affects disease risk via modulation of various mechanisms including, but not limited to, oxidative stress, plasticity, microbiota–gut–brain axis, and, most importantly, inflammatory responses.[41,42] Negative associations between adherence to a Mediterranean style diet (MED) and food groups that are abundant in these diets, such as fruits, vegetables, oily and non-oily fish, and inflammatory markers such as platelet and leukocyte counts, neutrophil to lymphocyte ratios (NLRs), and CRP[43–47] levels have been observed. Intervention studies have also shown a similar pattern of results. For instance, results from meta-analyses of randomized controlled trials (RCTs) have provided evidence that a MED decreases inflammation, specifically CRP, IL-6 and IL-1β.[48,49]

Based on the evidence discussed above, inflammation may mediate the association between anti-inflammatory diets, such as the MED, and health outcomes. In fact, a recent cross-sectional study found a role for various inflammatory biomarkers in the relationship between diet and sleep quality,[45] though the role of inflammation on the relationship between diet and mental and cognitive health has not yet been examined. However, there is a considerable amount of cross-sectional evidence showing that the adherence to a MED or healthy dietary patterns, including higher intakes of fruit, vegetables, fish, and wholegrains, were associated with a reduced risk of depression (but not anxiety), age-related cognitive decline, pathological neurodegeneration, and better general mental well-being.[50–55] Additionally, the consumption of various nutrients (B vitamins, vitamin D, polyphenols, n-3 fatty acids, fiber) and certain food groups (fish/seafood, vegetables, fruits) were also shown to be associated with better mental and cognitive outcomes.[51,54,56–61] Similarly, based on recent reviews, a low number of heterogeneous dietary intervention studies have shown beneficial effects of the MED on symptoms of depression (but not anxiety) and cognitive decline.[62–64] Though, to the best of our knowledge, none of these studies have tested the extent to which these benefits are due to changes in inflammation.

Challenges and future directions

The research briefly presented above, unfortunately, is not free from challenges. First and foremost, many of the studies are observational, and either cross-sectional or prospective in design, which limits our ability to infer a causal relationship between diet, inflammation, and physical, cognitive and mental health. Secondly, although various RCTs have been conducted in this area, they recruited small numbers of participants, and the heterogeneity of research methodology cannot be underestimated. Specifically, there is considerable variation in assessment of mental and cognitive health and dietary outcomes, and not all of the studies utilized consistent, validated and sensitive measures. Of specific importance, dietary intake measures are known to rely on the ability of participants to recall and report, and are prone to under-reporting.[65] Furthermore, the definition of a specific dietary pattern differs across distinct geographical and cultural contexts.[48] Third, the majority of the evidence in the area relies mainly on clinical samples and specific disorders (such as depression); however, research in subclinical samples and across a variety of psychopathological conditions is warranted. Fourth, most of the studies examined only a small subset of single measurements

of inflammation biomarkers, and time of the blood draw was not taken into consideration. Indeed, while single measurements of various inflammatory biomarkers have been shown to predict a range of health outcomes,[12,66] it is also important to note that intraindividual variability in inflammatory biomarkers has been observed before,[67] and some biomarkers show circadian rhythms.[68] Fifth, one of the biggest challenges to the field is conducting interdisciplinary research to address the interactions between peripheral and brain alterations. Therefore, future research studies would benefit from combining multiple techniques and linking inflammation to the structure, function and connectivity of the brain, as well as to other biomarkers, such as neurometabolites, amyloid, tau, and α-synuclein, that are known to be sensitive to mental and cognitive health alterations. Moreover, there is no current consensus regarding the best diet that has anti-inflammatory potential, and, to the best of our knowledge, no trials have compared the efficacy of dietary interventions on mental and cognitive health outcomes to pharmacological and/or other nonpharmacological interventions. Finally, it is important to note that interpopulation differences in dietary patterns may result in markedly different inflammatory potential, as energy, nutrient intake and density differ greatly across populations.[69]

Based on the challenges and limitations discussed above, we invite researchers to conduct longitudinal studies that aim to clarify the temporal relationships between mental/cognitive health, inflammation and nutritional domains, in order to ascertain whether immune dysregulation is a precursor or the result of mental and cognitive health outcomes, or if it is a bidirectional pathway. We would also like to encourage researchers to conduct large RCTs: 1) by using consistent, validated, sensitive (and ideally objective) diet, mental and cognitive health measures; 2) in clinical and subclinical samples, across various psychopathological conditions; 3) by using wide range of inflammation biomarkers (measured at multiple time points to control for intraindividual variations); and 4) ideally by also utilising brain imaging and cerebrospinal fluid biomarkers, to identify the best anti-inflammatory diets, test the efficacy of these on mental and cognitive health outcomes, and test the efficacy of these in comparison to pharmacological and other nonpharmacological interventions. These interventions should, of course, control for various participant (such as sex,[70] severity of health issues, body mass index (BMI), smoking, exercise, medical comorbidities,[71] genetic heterogeneity, etc.) and sample (collection/processing/storage practices, time of the sample collection, etc.) characteristics. Filling the knowledge gaps discussed in this editorial will not only advance theoretical frameworks that characterize interactions between the gut and the brain, but also move fundamental research towards translational applications that could be used for disorders where inflammation is implicated.

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