COVID-19 and Fast Foods Consumption: a Review

Jalal Bohlouli, Amir Reza Moravejolahkami, Marjan Ganjali Dashti, Zakiyeh Balouch Zehi, Mohammad Ali Hojjati Kermani, Mohammad Borzoo-Isfahani, and Nimah Bahreini-Esfahani

*Department of Nutrition, Nutrition and Food Security Research Centre, School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran; 2Department of Clinical Nutrition, School of Nutrition & Food Science, Isfahan University of Medical Sciences, Isfahan, Iran; 3Department of Biological Sciences, University of Texas at Dallas, Richardson, Texas, USA; 4Department of Nutrition, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran; 5Clinical Tuberculosis and Epidemiology Research Center, National Research Institute of Tuberculosis and Lung Diseases (NRITLD), Shahid Beheshti University of Medical Sciences, Tehran, Iran; 6Department of Community Nutrition, School of Nutrition and Food Sciences, Isfahan University of Medical Sciences, Isfahan, Iran; 7Department of Food Science and Technology, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, Iran

ABSTRACT

While all groups are affected by the COVID-19 pandemic, the aged people as well as those with underlying chronic medical conditions are at the greatest risk. The higher adherence to refined carbohydrate diets, sweets, and saturated fats contributes to the prevalence of obesity and type 2 diabetes; these disorders increase the risk for severe COVID-19 morbidity and mortality. Fast food consumption activates the intrinsic immune system and impairs adaptive immunity, leading to chronic inflammation and impaired host defence against viruses. Furthermore, inflammatory responses caused by COVID-19 may have long-term costs in survived individuals, leading to chronic disorders such as dementia and neurodegenerative disease through neuroinflammatory mechanisms that are related to an unhealthy diet. Therefore, now more than ever, wider access to healthy foods should be a main concern and individuals should be aware of healthy eating habits to reduce COVID-19 complications.

ARTICLE HISTORY

Received 10 August 2020
Revised 2 January 2021
Accepted 4 January 2021

KEYWORDS

Fast Foods; Nutrition; Inflammation; Virus Diseases; COVID-19; SARS-CoV-2

INTRODUCTION

Coronavirus Disease 2019 (COVID-19) is an acute respiratory disease caused by a novel coronavirus (Severe Acute Respiratory Syndrome Coronavirus 2, SARS-CoV-2), that has reached pandemic grade. While COVID-19 affects all groups, rigorous pathology and mortality are disproportionately highest in the elderly, underrepresented minorities, and/or in those with underlying comorbidities. Type 2 diabetes and obesity, as two prominent risk factors for severe forms of COVID-19, can explain the health difference observed in these individuals. The high occurrence of these risk factors, worldwide, but especially in Iran and other developing countries is likely driven by increased consumption of the typical fast-food diets. They are consisting of high amounts of Saturated Fatty Acids (SFAs), refined carbohydrates, and sweets, and low levels of fibres, unsaturated fats, and antioxidants. Here, we wanted to evaluate the evidence on unhealthy nutrition in viral infections, so this review mainly has focussed on fast foods.
DETERMINANTS OF COVID-19 OUTCOME: THE ROLE OF DIETARY HABITS

The fast foods can lead to chronic activation of the innate immune system and an inhibition of the adaptive immune system. Briefly, excessive SFA usage may induce a lipotoxic condition and activate the Toll-Like Receptor 4 (TLR-4) on macrophages, dendritic cells, and neutrophils. The activation of these biological stimulants produces proinflammatory mediators and other effectors of the innate immune system. In addition, the use of High–Fat Diet (HFD) in rats increased macrophage infiltration to lung tissues, specifically in the alveoli. Recently, Siegers et al. demonstrated that HFD increases influenza A virus-associated cardiovascular damage in mice.

This is particularly pertinent to COVID-19 patients agreed on the high rate of infection among lung alveolar epithelial cells and the taking part of lung tissue inflammation and alveolar damage in COVID-19 morbidity. Moreover, fast food consumption and HFD inhibits T and B lymphocyte in the adaptive immune system, followed by an increase in oxidative stress markers. Particularly, oxidative stress – induced by HFD – impairs T and B cell proliferation and maturation, and induces B cell apoptosis; it has vital implication in host protection against viruses.

In an animal intervention, HFD exacerbated lung pathology due to influenza infection and impaired the adaptive immune response. Therefore, high intake of fast foods practically impairs adaptive resistance whereas shifting into chronic inflammation and severely weaken host protection against viral morbidity.

Interleukin (IL)-1β is an important mediator in immune responses of obese individuals. Animal studies also reported the upregulation of this cytokine in the lungs as well as the activation of Nuclear Factor kappa B (NF-kB) and some proinflammatory markers. Recent investigations have shown the effects of HFD feeding on lung inflammatory response, which was mediated by proinflammatory cytokines such as IL-1β, NF-kB, and IL-6. Furthermore, researchers have found that neutralization of IL-1β at different stages of entero viral infection prevents the development of chronic viral myocarditis by reducing inflammation. Today, scientists are trying to introduce the chemical compounds that affect IL-1β signalling and reduce the inflammation in COVID-19; therefore, from the nutritional aspect of view, individuals should decrease the consumption of HFD such as fast foods to suppress the expression of IL-1β and related proinflammatory process. This effort may be beneficial in COVID-19 pandemic.

In a recent study, the population of T and B cell were considerably lowered in patients with severe COVID-19; so, unhealthy diets may act as a negative trigger in SARS-CoV-2 infection. As known before, higher consumption of fast foods increases the risk of obesity. It should be also highlighted that in patients with obesity the response to antiviral and antimicrobial drugs is poorer, and the response to the vaccine is reduced.

Fast foods typically contain high amounts of industrially produced Trans Fatty Acids (TFAs). Diet rich in TFAs is associated with higher production of proinflammatory molecules, especially in individuals with diabetes. As well, previous studies reported that higher intake of TFAs is associated with the risk of weight gain and abdominal obesity in all age groups. In addition, TFAs has been associated with increased asthma risk and lung inflammation. Therefore, TFAs can indirectly worsen COVID-19 manifestations especially respiratory complications.

From another point of view, fast foods can act as a big source of toxic heavy metals in human, especially children. Cadmium (Cd), Chromium (Cr), Nickel (Ni), and Lead (Pb) are nonessential and their bioaccumulation in tissues leads to intoxication and inflammation depending on their potential toxic effects. For example, Pb exposure initiates Mitogen-Activated Protein Kinase (MAPK)-dependent inflammation by activating oxidative stress and miRNA-155 expression in vitro and in vivo. Cd is also very toxic and its long-term exposure leads to lung damage. Furthermore, oral feeding of Cd, Pb, Ni, and mercury increased encephalomyocarditis virus-induced mortality rates in mice. There are no further researches to identify the possible relationships between heavy metals and coronaviruses infections, but the people should lower the intakes of these toxic metals through the elimination of fast foods from their dietary habits. The gut microbiota also plays an important role in disease progression. The gut microbiota composition also changes by both disease and diet. A recent animal pilot study showed that fast food diet
has a considerable impact on gut microbiome composition in as short a time frame as 4 days. Mosquera et al. also observed that chronic inflammation resulting from a change in the gut microbiome of engineered mice or antibiotic-treated mice reduces the immune response induced by polymeric nanovaccines. The inefficient immune response associates with changes in microbiota post-vaccination and can be resolved by a new immunomodulatory nanomaterial that stimulates immune cells.

On the other hand, the role of the gut microbiome in lung diseases has been well expressed. It is also known that respiratory virus infections like SARS-CoV2 cause negative changes in the gut microbiome. In a pilot study on 15 patients with COVID-19, Zuo et al. found persistent alterations in the faecal microbiome during the time of hospitalization, compared with controls; faecal microbiota alterations were associated with faecal levels of SARS-CoV-2 and COVID-19 severity. With a better understanding of the gut microbiome, it is now identified that in addition to the intestinal flora itself, its metabolites such as Short-Chain Fatty Acids (SCFAs; mainly acetic acid, propionic acid, butyric acid, and valeric acid) are also involved in regulating vital activities of the human body. SCFAs modulate the activity of T cells and, therefore, they have an important link between flora and the immune system; they involve different molecular mechanisms and also play a role in viral infections. In addition, several studies have demonstrated that SCFAs have a beneficial impact on animal allergic airway disease and human asthma mainly through their anti-Tumor Necrosis Factor-alpha (TNF-α) properties. Although the controversy still exists for the therapeutic use of SCFA. Therefore, above, fast food consumption may worsen the clinical manifestation of COVID-19 through gut microbiota dysbiosis and proinflammation (Figure 1).

**VITAMINS, MINERALS AND COVID-19**

Epidemiological data report that various vitamin deficiencies may have increased susceptibility to complications and mortality due to COVID-19 infection. From a practical point of view, fast food intake can decrease the bioavailability and serum levels of essential micronutrients. Moreover, diet quality is inversely associated with fast food intake; vitamin B1, selenium (Se), and vitamin B3 intake decrease in fast food.

**Figure 1.** A summarized negative effects of fast foods and high-fat diet (HFD) on immune function.
consumers.\textsuperscript{[43]} The impact of vitamin D has been much discussed recently because of its protective effects on acute respiratory tract infections.\textsuperscript{[42]} Lower serum 25(OH) vitamin D concentrations have also been shown to associate with exposure to SARS-CoV-2 infection\textsuperscript{[44]} and COVID-19 severity.\textsuperscript{[45,46]} Muhairi et al.\textsuperscript{[47]} reported that circulating 25(OH) D concentrations were inversely correlated with the consumption of fast food per week in the United Arab Emirates population.

Vitamin B status should also be assessed in COVID-19 patients. Vitamin B deficiency has the potential to suppress immune function (both the innate and adaptive immune responses).\textsuperscript{[48,49]} High-doses of thiamine (B1) have been recommended for COVID-19 patients.\textsuperscript{[50]} Furthermore, supplementation of niacin (B3) can help control the inflammatory process (generally caused by interleukin 6) in patients with COVID-19.\textsuperscript{[51]} Another vitamin, folic acid, can be prescribed as an adjunct treatment for COVID-19 and respiratory disease in the early stages,\textsuperscript{[52]} because tetrahydrofolic acid and 5-methyl tetrahydrofolic acid have strong and stable binding affinities against the SARS-CoV-2.\textsuperscript{[53]} A recent paper reported that methylcobalamin (vitamin B12) has the potential to reduce organ damage in COVID-19.\textsuperscript{[54,55]}

The current evidence has controversial comments regarding vitamin C supplementation in COVID-19 infection. A meta-analysis of 29 controlled trials with 11,306 participants could not detect any therapeutic effect of vitamin C (1 g/day) on upper respiratory tract infections.\textsuperscript{[56]} In contrast, the administration of ~15 g/day of IV vitamin C for 4 days decreases mortality in sepsis-related Acute Respiratory Distress Syndrome.\textsuperscript{[57]} In another study, 17 COVID-19 patients who received IV vitamin C (1 g every 8 h for 3 days) had a significant decrease in inflammatory markers, including ferritin and D-dimer, and a trend to decreasing FiO\textsubscript{2} requirements.\textsuperscript{[58]} It might also help to reduce lung inflammation and lung injury in COVID-19.\textsuperscript{[59]}

The essential trace element Se may be useful in severely diseased and Se-deficient COVID-19 patients.\textsuperscript{[60]} Recently, Zhang et al.\textsuperscript{[61]} showed an association between the reported cure rates for COVID-19 and selenium status. In summary, there is strong evidence that low Se status in both animals and humans can result in more severe forms of the disease.\textsuperscript{[62]}

In practice, we suggest individuals to take these nutrients from the diet. We recommend that people consume high amounts of fiber, whole grains, fruits, and vegetables to boost immune function, especially those who survived from SARS-CoV-2. The therapeutic doses of nutrients may be beneficial as an adjunct therapy in COVID-19 infection.

**CONCLUSION**

Notably, it is necessary to consider the effect of lifestyle habits, for example diet, on the vulnerability to COVID-19 and recovery. Moreover, the large number of peoples that will survive from COVID-19 might be exposed to chronic medical circumstances that can be extra worsened by unhealthy diets. Finally, we recommend that individuals consume foods high in whole grains, fibres, unsaturated fats, and antioxidants to modulate gut microbiome and enhance immune function.

**Acknowledgments**

We thank all the nurses, doctors, clinicians, and researchers during COVID-19 pandemic.

**Funding**

The authors declare no support from any commercial organization for the submitted study.

**ORCID**

Jalal Bohlouli https://orcid.org/0000-0003-0266-2537
Amir Reza Moravejolahkami https://orcid.org/0000-0001-9707-0352
Marjan Ganjali Dashki https://orcid.org/0000-0003-0823-3805
Conflicts of interest/Competing interests

None of the authors had a conflict of interest.

Availability of data and material (data transparency)

Not applicable

Code availability (software application or custom code)

Not applicable

Authors’ contributions

CRediT author statement

Amir Reza Moravejolahkami, Nimah Bahreini-Esfahani: Conceptualization, Writing-Original Draft, Supervision.
Jalal Bohlouli, Marjan Ganjali Dashti, Zakiyeh Balouch Zehi, Mohammad Ali Hojjati Kermani, Mohammad Borzoo-Isfahani: Writing-Review & Editing, Resources.

References

[1] Suvarna, V. R.; Vitamin, M. V. D and Its Role in Coronavirus Disease 2019 (COVID-19). J. Diabetol 2020, 11(2), 71.
[2] Richardson, S.; Hirsch, J. S.; Narasimhan, M.; Crawford, J. M.; McGinn, T.; Davidson, K. W.; et al. Presenting Characteristics, Comorbidities, and Outcomes among 5700 Patients Hospitalized with COVID-19 in the New York City Area. Jama. 2020, 323(20), 2052.
[3] Forse, R. A.; Betancourt-Garcia, M. M.; Kisse, M. C. Epidemiology and Discrimination in Obesity. The ASMBS Textbook of Bariatric Surgery; Springer International Publishing, 2020, pp 3–14.
[4] Moravejolahkami, A. R.; Paknahad, Z.; Chitsaz, A. Association of Dietary Patterns with Systemic Inflammation, Quality of Life, Disease Severity, Relapse Rate, Severity of Fatigue and Anthropometric Measurements in MS Patients. Nutr. Neurosci. 2019, 1–11.
[5] Christ, A.; Lauterbach, M.; Latz, E. Western Diet and the Immune System: An Inflammatory Connection. Immunity. 2019, 51(5), 794–811. DOI: 10.1016/j.immuni.2019.09.020.
[6] Eguchi, K.; Manabe, I. Toll-like Receptor, Lipotoxicity and Chronic Inflammation: The Pathological Link between Obesity and Cardiometabolic Disease. J. Atheroscler. Thrombosis. 2014, 21(7), 22533. DOI: 10.5551/ jat.22533.
[7] Rogero, M. M.; Calder, P. C. Obesity, Inflammation, Toll-like Receptor 4 and Fatty Acids. Nutrients. 2018, 10(4), 432. DOI: 10.3390/nu10040432.
[8] Tashiro, H.; Takahashi, K.; Sadamatsu, H.; Kato, G.; Kurata, K.; Kimura, S.; et al. Saturated Fatty Acid Increases Lung Macrophages and Augments House Dust Mite-induced Airway Inflammation in Mice Fed with High-fat Diet. Inflammation. 2017, 40(3), 1072–1086.
[9] Siegers, J. Y.; Novakovic, B.; Hulme, K. D.; Marshall, R.; Bloxham, C. J.; Thomas, W. G.; et al. A High Fat Diet Increases Influenza A Virus-associated Cardiovascular Damage. J. Infect. Dis. 2020, 222(5), 820–831.
[10] Xu, Z.; Shi, L.; Wang, Y.; Zhang, J.; Huang, L.; Zhang, C.; et al. Pathological Findings of COVID-19 Associated with Acute Respiratory Distress Syndrome. Lancet Respir. Med. 2020, 8(4), 420–422.
[11] Myles, I. A.; Fast Food Fever: Reviewing the Impacts of the Western Diet on Immunity. Nutr. J. 2014, 13(1), 1–17.
[12] Tao, W.; Sun, W.; Liu, L.; Wang, G.; Xiao, Z.; Pei, X.; et al. Chitosan Oligosaccharide Attenuates Nonalcoholic Fatty Liver Disease Induced by High Fat Diet through Reducing Lipid Accumulation, Inflammation and Oxidative Stress in C57BL/6 Mice. Mar. Drugs. 2019, 17(11), 645.
[13] Green, W. D.; Beck, M. A. Obesity Impairs the Adaptive Immune Response to Influenza Virus. Ann. Am. Thoracic Soc. 2017, 14(Supplement 5), S406–S9. DOI: 10.1513/AnnalsATS.201706-447AW.
[14] Mirea, A.-M.; Stienstra, R.; Kanneganti, T.-D.; Tack, C. J.; Chavakis, T.; Toonen, E. J.; et al. Mice Deficient in the IL-1β Activation Genes Prtn3, Elane, and Casp1 are Protected against the Development of Obesity-Induced NAFLD. Inflammation. 2020, 43(3), 1054–1064.
[39] Halnes, I.; Baines, K. J.; Berthon, B. S.; MacDonald-Wicks, L. K.; Gibson, P. G.; Wood, L. G. Soluble Fibre Meal Challenge Reduces Airway Inflammation and Expression of GPR43 and GPR41 in Asthma. *Nutrients*. 2017, 9(1), 57. DOI: 10.3390/nu9010057.

[40] Parada Venegas, D.; De la Fuente, M. K.; Landskron, G.; González, M. J.; Quera, R.; Dijkstra, G.; et al. Short Chain Fatty Acids (Scfas)-mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases. *Front. Immunol.* 2019, 10, 277. DOI: 10.3389/fimmu.2019.00277.

[41] Duntas, L. H.; Dumont, J. Bicentennial of the Discovery of Selenium Commemorated at the Museum of Natural History in Athens. *Hormones*. 2020, 19(1), 1–2. DOI: 10.2478/horm-2020-00172-3.

[42] Martinou, A. R.; Jolliffe, D. A.; Hooper, R. L.; Greenberg, L.; Aloia, J. F.; Bergman, P.; Dubnov-Raz, G.; Esposito, S.; Ganmaa, D.; Ginde, A. A.; et al. Vitamin D Supplementation to Prevent Acute Respiratory Tract Infections: Systematic Review and Meta-analysis of Individual Participant Data. *BMJ*. 2017, 356, 356. DOI: 10.1136/bmj.j6583.

[43] Rouhani, M. H.; Misrsefinezhad, M.; Omrani, N.; Esmailzadeh, A.; Azadbakht, L. Fast Food Consumption, Quality of Diet, and Obesity among Isfahanian Adolescent Girls. *J. Obes.* 2012, 2012, 2012. DOI: 10.1155/2012/597924.

[44] D’Avolio, A.; Avataneo, V.; Manca, A.; Cusato, J.; De Nicolò, A.; Lucchini, R.; et al. 25-hydroxyvitamin D Concentrations are Lower in Patients with Positive PCR for SARS-CoV-2. *Nutrients*. 2020, 12(5), 1359.

[45] Panagiotou, G.; Tee, S. A.; Ihsan, Y.; Athar, W.; Marchitelli, G.; Kelly, D.; et al. Low Serum 25-hydroxyvitamin D (25 [OH] D) Levels in Patients Hospitalized with COVID-19 are Associated with Greater Disease Severity. *Clin. Endocrinol.* 2020, 93(4), 508–511.

[46] Martinou, A. R.; Forouhi, N. G. Vitamin D for COVID-19: A Case to Answer? *Lancet Diab. Endocrinol.* 2020, 8(9), 735–736. DOI: 10.1016/S2213-8578(20)30268-0.

[47] Muhairi, S. J.; Mehairi, A. E.; Khouri, A. A.; Naibli, M. M.; Maskari, F. A.; Al Kaabbi, J.; et al. Vitamin D Deficiency Among Healthy Adolescents in Al Ain, United Arab Emirates. *BMJ Public Health*. 2013, 13(1), 1–7.

[48] Mikkelsen, K.; Vitamin, A. V. B1, B2, B3, B5, and B6 and the Immune System. *Nutrition and Immunity*; Springer, 2019; pp 115–125.

[49] Michele, C. A.; Angel, B.; Valeria, L.; Teresa, M.; Giuseppe, C.; Giovanni, M.; et al. Vitamin Supplements in the Era of SARS-CoV2 Pandemic. *GSC. Biol. Pharm Sci.* 2020, 11(2), 007–19. DOI: 10.30574/gscbps.2020.11.2.0114

[50] Shakoor, H.; Feehan, J.; Mikkelsen, K.; Al Dhaheri, A. S.; Ali, H. I.; Platat, C.; et al. Be Well: A Potential Role for Vitamin B in COVID-19. *Maturitas*. 2020, 144, 108–111.

[51] Liu, B.; Li, M.; Zhou, Z.; Guan, X.; Xiang, Y. Can We Use Interleukin-6 (IL-6) Blockade for Coronavirus Disease 2019 (Covid-19)-induced Cytokine Release Syndrome (CRS)? *J. Autoimmun.* 2020, 111, 102452. DOI: 10.1016/j.jaut.2020.102452.

[52] Sheybani, Z.; Dokhoohaki, M. H.; Negahdari-pour, M.; Dehdashti, M.; Zolghadr, H.; Moghadami, M.; et al. The Role of Folic Acid in the Management of Respiratory Disease Caused by COVID-19. ChemRxiv. Preprint. 2020. https://doi.org/10.26434/chemrxiv.12034980.v1

[53] Kumar, V.; Jena, M. In Silico Virtual Screening-based Study of Nutraceuticals Predicts the Therapeutic Potentials of Folic Acid and Its Derivatives against COVID-19. 2020.

[54] Dos Santos, L. M. I.; Can Vitamin B12 Be an Adjuvant to COVID-19 Treatment? *GSC. Biol. Pharm Sci.* 2020, 11(3), 001–5. DOI: 10.30574/gscbps.2020.11.3.0155.

[55] Tan, C. W.; Ho, L. P.; Kalimuddin, S.; Cherrng, B. P. Z.; Teh, Y. E.; Thien, S. Y.; et al. A Cohort Study to Evaluate the Effect of Combination Vitamin D, Magnesium and Vitamin B12 (DMB) on Progression to Severe Outcome in Older COVID-19 Patients. *medRxiv*. 2020.

[56] Hemila, H.; Chalker, E. Vitamin C for Preventing and Treating the Common Cold. *Cochrane Database Syst. Rev.* 2013, 1. doi: 10.1002/14651858.CD000980.pub4

[57] Truwit, J. D.; Hite, R. D.; Morris, P. E.; DeWilde, C.; Priday, A.; Fisher, B.; et al. Effect of Vitamin C Infusion on Organ Failure and Biomarkers of Inflammation and Vascular Injury in Patients with Sepsis and Severe Acute Respiratory Failure: The CITRIS-ALI Randomized Clinical Trial. *Jama*. 2019, 322(13), 1261–1270.

[58] Hiedra, R.; Lo, K. B.; Elbashabsheh, M.; Gul, F.; Wright, R. M.; Albano, J.; et al. The Use of IV Vitamin C for Patients with COVID-19: A Case Series. *Expert Rev. Anti-Infect. Ther.* 2020, 18(12), 1259–1261.

[59] Hernández, A.; Papadakos, P.; Torres, A.; González, D.; Vives, M.; Ferrando, C.; et al. Two Known Therapies Could Be Useful as Adjuvant Therapy in Critical Patients Infected by COVID-19. Revista Española de Anestesiología y Reanimación (English Edition). 2020, 67(5), 245–252. DOI: 10.1016/j.redare.2020.05.002

[60] Moghaddam, A.; Heller, R. A.; Sun, Q.; Seelig, J.; Cherkezov, A.; Selbert, L.; et al. Selenium Deficiency Is Associated with Mortality Risk from COVID-19. *Nutrients*. 2020, 12(7), 2098.

[61] Zhang, J.; Taylor, E. W.; Bennett, K.; Saad, R.; Rayman, M. P. Association between Regional Selenium Status and Reported Outcome of COVID-19 Cases in China. *Am. J. Clin. Nutr.* 2020, 111(6), 1297–1299. DOI: 10.1093/ajcn/nqaa095.

[62] Bermano, G.; Méplan, C.; DK, M.; JE, H. Selenium and Viral Infection: Are There Lessons for COVID-19? *Br. J. Nutr.* 2020, 1–10.