Original Article

The Association between Dietary Inflammatory Index with Risk of Coronavirus Infection and Severity: A Case-Control Study

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Abstract

Background: Recently, several have evaluated the association between the components of the dietary inflammatory index (DII) score with the risk and severity of Coronavirus Disease 2019 (COVID-19). For the first time, we examined the association between DII® with risk of coronavirus infection and symptom severity through a case-control study in Iran. **Methods:** The present case-control study was conducted on COVID-19 cases (n = 100) and healthy control (n = 100) volunteer, aged from 18 to 65 years. Dietary intake, DII, body mass index, COVID-19 infection, and the severity of its symptoms were assessed for each participant. A multivariable logistic regression analysis test was used to estimate the odds ratio and 95% confidence interval. **Results:** Our results demonstrated that COVID-19–infected patients were significantly older and had longer history of diabetes as compared to the healthy control group (P <.05). Furthermore, the participants with COVID-19 had a significantly greater intake of total fat (P =0.259), saturated fat (P =0.005), and dietary fiber (P = .004). In contrast, individuals in the healthy control group had a higher intake of carbohydrate (P = .005), sodium (P <.001), and iron (P <.001). However, there was no significant difference in DII score between COVID-19 and healthy controls (P =.259). In addition, we did not detect any specific association between DII score and risk of COVID-19 infection (odds ratio = 1.08, 95% confidence interval: 0.92 to 1.27; P =.294) and the severity of its symptoms (P >.05). **Conclusions:** There appears to be no specific association between DII score and risk of COVID-19 infection and the severity of its symptoms. More prospective cohort studies are necessary to confirm the veracity of our results.

Keywords: *Coronavirus, COVID-19, dietary inflammatory index, symptoms*

Introduction

The newly universal health challenge, the novel coronavirus SARS-CoV-2,[1] is a respiratory infectious disease, known as Coronavirus Disease 2019 (COVID-19), that has imposed a huge burden of morbidity and mortality around the world.[1,2] Patients infected with COVID-19 present with various symptoms and complications in primary stages, in particular, headache, fatigue, fever, diarrhea, dry cough, and weakness.[3,4] In severe cases, the disease may even lead to acute respiratory distress syndrome, multiple organ failure, and ultimately death.[5,6] Recent studies have suggested that some risk factors, including elderly age (>60 years), being male, comorbidities, poor nutritional status, decreased lymphocytes, reduced hemoglobin, and virus-induced inflammatory storm are associated with disease severity in COVID-19 patients.[7,8]

The regulatory control of virus-induced inflammatory cytokine storm may help COVID-19 patients to reduce the risk of the disease progresses and consequently death.[8,9] Medications are considered as the main mediator of body inflammation control in COVID-19 patients.[10] However, some effective lifestyle modifications in these patients, such as diet modification and exercise training, can decrease inflammatory cytokines and reduce the risk of disease and the severity of symptoms.[11,12] Numerous studies have documented that dietary inflammatory index (DII) acts a role in improving body inflammation status,[13-16] whereas several have evaluated the association between the components of the DII score with the risk and severity of COVID-19.[17-22] Ye k *et al.*[17] reported that higher rates of vitamin D deficiency were observed among COVID-19 patients, especially for the severe stages of the disease, and Ilie *et al.*[18] suggested that the vitamin D concentration negatively associated with the number of

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COVID-19 patients and the death caused by COVID-19. Omega 3 also represents one of the factors affecting on DII score that have an anti-inflammatory function and can ameliorate COVID-19 and related complications.[19,22] Other components of the DII with antioxidant properties have been proposed to decrease inflammation risk among COVID-19, namely vitamin C, vitamin E, phytochemicals, zinc, and selenium.[20,21,23] Furthermore, Conte *et al*.[11] demonstrated that dietary fibers intake may lead to decline inflammatory cytokine levels and enhance the immune system activity in COVID-19 patients.

Recently, the relationship between dietary intakes of macronutrients and micronutrients and COVID-19 disease has been conducted.[11,17-23] However, no study has yet examined the association between DII, as an important dietary index related to inflammation, and the risk of the severity of COVID-19 symptoms. Therefore, for the first time in the literature, we sought to examine the association between DIIwith risk of coronavirus infection and the severity of its symptoms through a case-control study.

Methods and Martials

Participants

The present case-control work was carried out on COVID-19 cases (n = 100) and healthy control (n = 100) participants, aged from 18 to 65 years, from Emam Reza Hospital, Tehran, Iran (May 2020 to January 2021). This work was approved by the local ethics committee of AJA University of Medical Sciences (Ethic ID: IR.AJAUMS.REC.1399.229) and was performed based on the Declaration of Helsinki. The participants were selected in accordance with the inclusion criteria including aged more than 18 and less than 65 years and a positive polymerase chain reaction for COVID-19 or influenza in the abovementioned periods. The control group inclusion criteria were healthy adults aged more than 18 and less than 65 years and without any disease history.

Exclusion criteria if they were aged less than 18 and more than 65 years, the questionnaires were not completed and/or did not respond to more than 40 food items available in the food frequency questionnaire, dementia, low level of consciousness, previous history of smell and/or taste disorders or a drug-induced, neurological, rhinological, or systemic disease-related smell and/or taste disorder, and (for controls) lack of response to the telephone call. All participants signed the informed consent form prior to taking part in the research.

Variables evaluations

The demographic variables, including age, gender, height, body mass index (BMI), and history of disease were gathered through a general questionnaire. The diagnosis of COVID-19 was conducted as per results of the polymerase chain reaction SARS-CoV-2 test for all cases. Furthermore, the visual analogue scale was used to assess COVID-19 complications. This scale was ranged from 0 (worst imaginable health) to 100 (best imaginable health).

Dietary intake was evaluated with the use of a valid and reliable,[24] 168-item, semi-quantitative food frequency questionnaire to assess the usual dietary intake during the year preceding the evaluation. Individuals reported the amount and frequency of consumption of each food item during the past year on a daily, weekly, or monthly basis. Household measures were used to convert all the portion sizes of consumed foods to grams.[25] Nutrient analysis of diets was done using Nutritionist IV software with its database modified for Iranian foods.

The Shivappa *et al*.[26] method was used to evaluate the DII score. For this, first global mean intake of each dietary item was subtracted from dietary intake of it and then this value was divided by world standard deviation intake of that factor as derived from the datasets of 11 countries (i.e., to obtain the z-score).[27] To minimise the effect of 'right skewing', dietary factor-specific z-scores were converted to a proportion (i.e., with values from 0 to 1). Next, these values were multiplied by 2 and then 1 was subtracted to achieve a symmetrical distribution with a range of -1 to +1 and centred on 0. To obtain the factor-specific DII score of each food, the amount for each dietary factor was multiplied by the overall dietary factor-specific inflammatory effect score.[27] Ultimately, all dietary factor-specific DII scores were summed to calculate the overall DII score for each participants. Data on 32 of 45 possible dietary factors that could be used to calculate the DII score were available in this case-control study. A total of 32 items included; energy, carbohydrate, protein, fibre, total fat, cholesterol, n-3 fatty acids, n-6 fatty acids, saturated fatty acid, tranc fatty acid, mono-unsaturated fatty acid, poly-unsaturated fatty acid, vitamin B6, vitamin B_1, vitamin B_2, vitamin B_3, vitamin B_9, vitamin B12, vitamin A, β-Carotene, vitamin C, vitamin D, vitamin E, Garlic, Iron, Magnesium, Selenium, Zinc, onion, pepper, Caffeine, and Tea were used to DII score calculation. Other components of DII® were not collected as part of the food frequency questionnaire.

Sample size calculation

The sample size was determined to provide detecting differences in smell loss or taste loss prevalence between COVID-19 positive and negative populations. Based on previous research,[28,29] we applied conservative estimates of 60% prevalence in the positive population and 35% prevalence in the negative population. Power was estimated by Monte-Carlo simulations, repeatedly (b = 1000) resampling from 2 binomial distributions corresponding to the positive population and the negative population. Assuming 100 individuals are assigned to each group and a 2-sided *t*-test is used, the probability of detection (power) is 92%.

Statistical analysis

Descriptive statistics, including the mean (standard deviation) and frequency summary statistics, were used to describe the study population charatractics. Comparisons between groups for data with normal distribution were made using an independent *t*-test for continuous variables and the Chi-squared test to investigate categorical variables. Multiple logistic regression analyses were then used to calculate adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for risk of coronavirus infection and the severity of its symptoms in relation to DII in three separate models. Model 1 represents the crude model without adjusment, and in model 2, the effects of age and gender were adjusted for as potential confounders. In the third model, we adjusted model 2 for diabetes disease, pulmonary disease, and BMI. All reported *P* values were two-sided and a *P* value of. 05 was, *a priori*, considered statistically significant. All statistical analysis was conducted using SPSS version 22.0 (SPSS, Chicago, IL, USA).

Results

Participants features

Demographic features and disease history of both COVID-19 and healthy groups are illustrated in Table 1. Our results showed that subjects with COVID-19 were significantly older, in comparison with the healthy group (*P* =0.031). Furthermore, we observed that subjects with COVID-19 had significantly longer history of diabetes disease than healthy participants (*P* = 0.019). The COVID-19 patients also, marginally, had a greater prevalence of history of pulmonary disease (*P* =. 054) or hypertension (*P* = .077) than the healthy group. However, the healthy participants had, marginally, greater incidence of a history of hypothyroidism disease than the COVID-19 group (*P* = .050). We did not detect significant differences between two groups for BMI (*P* =.381), gender (*P* =.444), height (*P* =.145), weight (*P* =.242), fatty liver disease (*P* =.529), or hyperlipidemia (*P* =.525).

Table 1: Background characteristics of participants by health status

Variable	Subjects with COVID-19 (*n*=100)	Healthy Controls (*n*=100)	*P*
Age (y)	40.92±14.01	36.84±12.53	0.031[a]
Male	51.0%	49.0%	0.444[b]
Height (cm)	170.37±9.92	167.90±13.63	0.145[a]
Weight (kg)	74.83±14.51	72.30±15.94	0.242[a]
BMI (kg/m^2)	25.71±4.15	25.32±4.32	0.381[a]
Disease History			
Diabetes	11.7%	3.0%	0.019[b]
Hypertension	17.0%	9.1%	0.077[b]
Fatty Liver	3.2%	4.0%	0.529[b]
Hypelipidemia	8.5%	9.1%	0.545[b]
Pulmonary	4.3%	0.0%	0.054[b]
Hypothyroidism	3.2%	10.1%	0.050[b]

Note: Variables are expressed as mean±SD. [a]*P* Value for independent *t* test. [b]*P* Value for Chi-Square tests

Dietary intake and DII score

The between groups differences for macronutrient or micronutrient intake and DII score are reported in Table 2. As per our outcomes, there was not any significant difference in DII score between COVID-19 and healthy groups (*P* =0.259). Our results also indicated that participants with COVID-19 had significantly more intake of total fat (*P* =0.003), saturated fat (*P* =.023), omega 3 fatty acids (*P* =.001), dietary fiber (*P* <.001), and maltose (*P* <.001) than the healthy control group. In contrast, individuals in the healthy control group had a higher intake of carbohydrate (*P* =.004), sodium (*P* <.001), iron (*P* <.001), vitamin K (*P* <.001), manganese (*P* =.004), lutein (*P* =.003), and sucrose (*P* <.001) in comparison with the COVID-19 group. However, we did not find significant differences between two groups for other dietary items.

DII score and risk of coronavirus infection and the severity of its symptoms

The ORs and 95% CIs for the DII score with risk of COVID-19 infection and the severity of its symptoms are shown in Table 3. Overall, we did not find any specific relationship, after adjustment for confounding factors, between DII score and risk of COVID-19 infection (OR = 1.08, 95% CI: 0.92 to 1.27; *P* =0.294). We also did not detect any specific association, after adjustment for confounding factors, between DII score and risk of the severity of COVID-19 symptoms, including headache (OR = 0.89, 95% CI: 0.66 to 1.18; *P* =0.423), weakness (OR = 0.97, 95% CI: 0.75 to 1.24; *P* =.828), cough (OR = 0.83, 95% CI: 0.66 to 1.05; *P* =.133), fever (OR = 1.01, 95% CI: 0.73 to 1.40; *P* =.916), and diarrhea (OR = 1.60, 95% CI: 0.60 to 4.27; *P* =.343).

Discussion

For the first time in the available literature, we evaluated the relationship between DII with the risk of coronavirus infection and the severity of its symptoms through a case-control study. Our results demonstrated that COVID-19–infected patients were significantly older in comparison with the healthy group. Furthermore, we observed that diabetes was significantly more prevalent in COVID-19 patients compared to healthy controls. Our results also indicated that participants with COVID-19 had significantly more intake of total fat, saturated fat, dietary fiber, and maltose than the healthy control group. In contrast, individuals in the healthy control group had a higher intake of carbohydrate, sodium, iron, vitamin K, manganese, lutein, and sucrose in comparison with the COVID-19 group. However, there was not any significant difference noted in DII score between COVID-19 and healthy groups. Finally, we did not discern any specific

Table 2: Dietary intake and DII score of the study participants

Variables	Subjects with COVID-19 (*n*=100)	Healthy Controls (*n*=100)	*P*[a]
Total DII score	0.07±2.0	-0.23±1.76	0.259
Energy (kcal/day)	3203.12±1368.02	3481.21±1748.38	0.212
Carbohydrate (g/day)	133.76±17.32	141.79±21.54	0.004
Protein (g/day)	40.13±7.87	39.40±8.98	0.542
Fat (g/day)	36.06±6.55	33.06±7.60	0.003
Cholesterol (mg/day)	146.68±75.62	129.44±48.99	0.057
MUFAs (g/day)	12.30±2.96	11.83±3.25	0.281
Trance fat (g/day)	0.0011±0.00400	0.0007±0.00172	0.305
Saturated fat (g/day)	11.84±3.18	10.82±3.11	0.023
Omega 3 fatty-acids (mg/day)	0.49±0.19	0.40±0.15	0.001
Omega 6 fatty-acids (mg/day)	0.03±0.043	0.03±0.07	0.671
PUFAs (g/day)	7.18±2.43	6.46±2.33	0.033
Dietary fibre (g/day)	16.69±6.98	13.69±4.00	< 0.001
Sodium (mg/day)	2377.21±1463.55	4108.27±3359.04	< 0.001
Potassium (mg/day)	1787.65±451.85	1886.02±441.55	0.121
Vitamin A (mg/day)	277.10±176.41	299.21±144.19	0.333
Vitamin C (mg/day)	64.92±40.99	72.70±35.20	0.152
Vitamin E (mg/day)	5.68±3.04	5.89±3.21	0.633
Vitamin K (mg/day)	88.60±59.85	139.94±89.58	< 0.001
Vitamin D (µg/day)	0.72±0.65	0.69±0.65	0.684
B1 (mg/day)	0.76±0.15	0.7323±0.16	0.140
B2 (mg/day)	0.89±0.25	0.90±0.27	0.735
B3 (mg/day)	10.78±2.56	10.52±2.84	0.506
B6 (mg/day)	0.89±0.20	0.89±0.18	0.318
B9 (µg/day)	224.94±45.70	217.88±50.22	0.300
B12 (µg/day)	2.19±0.85	2.22±0.94	0.814
Biotin (mg/day)	13.77±6.38	13.22±4.71	0.489
Pantothenic (mg/day)	2.46±0.62	2.54±0.63	0.363
Calcium (mg/day)	506.74±168.08	539.64±204.53	0.215
Phosphorus (mg/day)	663.41±142.87	661.62±161.93	0.934
Iron (mg/day)	10.41±3.91	13.62±5.88	< 0.001
Magnesium (mg/day)	165.58±28.19	172.46±33.47	0.118
Zinc (mg/day)	5.54±1.24	5.77±1.26	0.201
Selenium (mg/day)	43.96±10.86	45.12±10.92	0.453
Copper (mg/day)	0.67±0.11	0.66±0.15	0.620
Manganese (mg/day)	2.46±0.71	2.81±0.96	0.004
Beta-Carotene (µg/day)	1734.66±1675.88	2054.17±1325.17	0.136
Caffeine (mg/day)	56.52±51.11	45.49±41.67	0.096
Alpha-caroten (µg/day)	436.12±643.88	495.19±486.37	0.465
Lutein (µg/day)	837.43±528.53	1134.21±850.25	0.003
Beta-cryptoxanthin (µg/day)	128.18±100.19	128.28±87.73	0.994
Lycopene (mg/day)	23.50.65±1471.31	2293.40±1382.93	0.840
Alpha-tocopherol (mg/day)	4.05±2.04	4.40±2.16	0.239
Fluorine (mg/day)	980.26±837.68	786.03±667.19	0.071
Chromium (mg/day)	0.01±0.01	0.02±0.02	0.131
Soluble fiber (g/day)	0.25±0.20	0.24±0.14	0.603
Insoluble fiber (g/day)	1.33±1.01	1.24±0.65	0.461
Crude fiber (g/day)	5.53±3.69	6.28±2.72	0.106
Glucose (g/day)	7.44±3.25	7.66±3.23	0.636
Galactose (g/day)	1.53±1.32	1.48±1.30	0.774
Fructose (g/day)	8.88±4.59	9.51±5.33	0.373
Sucrose (g/day)	15.27±9.08	21.34±17.30	0.002
Lactose (g/day)	6.60±4.61	6.50±5.05	0.890
Maltose (g/day)	0.59±0.39	0.39±0.17	< 0.001

DII: Dietary inflammatory index score, MUFAs: Monounsaturated fatty acids , PUFAs: Polyunsaturated fatty acids. Note: Variables are expressed as mean±SD. [a]Obtained from independent t test, adjusted for total energy intake

Table 3: Odds ratios (ORs) and 95% confidence intervals (CIs) for the dietary inflammatory index with risk of COVID-19 infection and the severity of its symptoms

Variable	Model 1	*P*	Model 2	*P*	Model 3	*P*
COVID-19	1.08 (0.93 to 1.26)	0.260	1.11 (0.95 to 1.30)	0.163	1.08 (0.92 to 1.27)	0.294
Headache	0.93 (0.71 to 1.22)	0.623	0.90 (0.68 to 1.19)	0.481	0.89 (0.66 to 1.18)	0.423
Weakness	0.91 (0.73 to 1.13)	0.424	0.96 (0.77 to 1.20)	0.750	0.97 (0.75 to 1.24)	0.828
Cough	0.84 (0.66 to 1.04)	0.114	0.83 (0.66 to 1.05)	0.125	0.83 (0.66 to 1.05)	0.133
Fever	1.03 (0.78 to 1.36)	0.791	0.96 (0.72 to 1.28)	0.811	1.01 (0.73 to 1.40)	0.916
Diarrhea	1.60 (0.17 to 3.78)	0.282	1.59 (0.67 to 3.79)	0.291	1.60 (0.60 to 4.27)	0.343

Model 1: Crude model. Model 2 : Adjusted for age and sex. Model 3 : Adjusted as in Model 2, diabetes disease, pulmonary disease, and BMI

relationship, after adjustment for confounding factors, between DII score and risk of COVID-19 infection and the severity of its complications including headache, weakness, cough, fever, and diarrhea.

We observed that COVID-19–infected patients were older and had higher prevalence of a diabetes disease history in comparison with the healthy control group. Previous research has reported that individuals with a history of diabetes disease or older ages were more susceptible to COVID-19 infection and severity of its complications such as pneumonia, acute respiratory distress syndrome, multiple organ failure, and mortality.[30-33] Several potential mechanisms of action have been suggested in this regard, including mitochondrial dysfunction, dysregulation, defective immune function, chronic inflammation, cytokine storms, and coagulation dysfunction including mitochondrial dysfunction, dysregulation or defective immune function, chronic inflammation, cytokine storms, and coagulation dysfunction.[30-33] Therefore, differences between the two groups in the demographic parameters, which show older age and higher history of diabetes disease prevalence in the COVID-19–infected group are concordant with the extant literature.

Another finding of the present study revealed that COVID-19 patients had a significantly greater intake of total fat, saturated fat, and dietary fiber. In contrast, healthy participants had a higher intake of carbohydrates, sodium, and iron. These differences between the two groups may be due to the nutritional recommendations implemented for COVID-19 patients during their illness. Indeed, in the preceding year, several studies have suggested nutritional recommendations for COVID-19 patients.[11,34] Kossoff, *et al*.,[35] Paoli, *et al*.,[36] and Paoli, *et al*.[34] have suggested that high fat and low carbohydrate diets, like the Ketogenic diet, may help COVID-19 patients by increasing serum ketone bodies, decrease circulating glucose, promoting immunity response, declining virus-induced proinflammatory and inflammation cytokines, and delaying respiratory cell apoptosis. Moreover, Khoramipour, *et al*.[34] and Conte, *et al*.[11] have suggested that dietary fiber via probiotic features, produce short-chain fatty acids, decrease blood glucose concentration, and enhances the level of adiponectin as anti-inflammatory adipocytokine with insulin-sensitive characteristics may reduce inflammation and enhance the immune system activities in COVID-19 patients. Furthermore, our observations revealed that healthy participants had a higher intake of sodium and iron vs. COVID-19 patients. It seems that better dietary intake of vitamins and minerals, especially sodium and iron, in healthy individuals led to a boost in immune function and reduced the risk of COVID-19 infection.[37-39] However, although some dietary components of the DII score were different between the two groups, these were not significant.

In addition, we did not observe any specific association between DII score and risk of COVID-19 infection and the severity of its complications. The DII score has been used in studying the association between dietary intake and inflammation,[40] where this score can be used to reduce the risk of inflammation-related complications in patients or healthy individuals by selecting foods with lower inflammatory potential or higher anti-inflammatory properties.[40,41] However, no studies, to our knowledge, have examined the association between DII and the risk of the severity of COVID-19 symptoms so far. Some previous studies have focused on the association between DII lung function and inflammatory cytokine in lung-related disease[16,42]; for instance, Wood *et al*.[16] demonstrated that DII score was significantly related to developed inflammation and diminished lung forced expiratory volume. Moreover, the higher DII score in Wood *et al*.[16] was associated to more serious asthma status. Han *et al*.[42] also indicated that the higher DII score was related to wheezing in adults, whereas the higher DII quartiles were linked to lower lung function parameters, such as forced expiratory volume and forced vital capacity, among adults. The difference between our results and these aforementioned studies may be due to differences in participants' obesity status. Indeed, the Wood *et al*.[16] and Han *et al*.[42] studies were conducted on overweight (BMI ≈ 28) or obese (BMI ≈ 30) cases, respectively. Obesity is an important factor in causing inflammation and immune responses[43] and can affect the association between DII score and risk of coronavirus infection and the severity of its symptoms. Therefore, it is advisable that this relationship be studied in future studies, in more detail, among overweight and/or obese individuals.

Strength and limitations

To our knowledge, the present study represents the first work to assess the relationship between DII with the risk of coronavirus infection and the severity of its symptoms. Further strengths of present study include the homogeneous case and control individuals of adults. Despite these strengths, our work possesses some limitations, which should be acknowledged in the interpretation of our outcomes. Indeed, we were not able to evaluate the effect of other possible confounding factors, such as clinical parameters and genetic differences, which could yield additional insights. Another limitation of the current work was the relatively small number of participants in the both COVID-19 and healthy control groups.

Conclusions

In summary, we assessed the relationship between DII with the risk of coronavirus infection and the severity of its symptoms. Our results demonstrated that COVID-19–infected patients were significantly older and diabetes disease history prevalence was greater, in comparison with the healthy group. Furthermore, the participants with COVID-19 had a significantly greater intake of total fat, saturated fat, and dietary fiber vs. healthy control group. In contrast, individuals in the healthy control group had a higher intake of carbohydrates, sodium, and iron. However, there was no significant difference in DII score between COVID-19 and healthy groups. Finally, we did not detect any specific association between DII score and risk of COVID-19 infection and the severity of its symptoms. More prospective cohort studies, with larger sample sizes and extended follow-up periods, are necessary to confirm the veracity of our results.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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References

1. Velavan TP, Meyer CG. The COVID-19 epidemic. Trop Med Int Health 2020;25:278-80.
2. Fauci AS, Lane HC, Redfield RR. Covid-19-navigating the uncharted. Mass Medical Soc; 2020;382:1268-9.
3. Day M. Covid-19: Ibuprofen should not be used for managing symptoms, say doctors and scientists. BMJ 2020;368:m1086.
4. Rothan HA, Byrareddy SN. The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. J Autoimmun 2020;109:102433.
5. Latimer G, Corriveau C, DeBiasi RL, Jantausch B, Delaney M, Jacquot C, *et al*. Cardiac dysfunction and thrombocytopenia-associated multiple organ failure inflammation phenotype in a severe paediatric case of COVID-19. Lancet Child Adolesc Health 2020;4:552-4.
6. Fan E, Beitler JR, Brochard L, Calfee CS, Ferguson ND, Slutsky AS, *et al*. COVID-19-associated acute respiratory distress syndrome: Is a different approach to management warranted? Lancet Respir Med 2020;8:816-21.
7. Zhang J, Wang X, Jia X, Li J, Hu K, Chen G, *et al.* Risk factors for disease severity, unimprovement, and mortality in COVID-19 patients in Wuhan, China. Clinical Microbiology and Infection. 2020;26:767-72.
8. Ye Q, Wang B, Mao J. The pathogenesis and treatment of the Cytokine Storm'in COVID-19. J infect 2020;80:607-13.
9. Ye Q, Wang B, Mao J. Cytokine storm in COVID-19 and treatment. Journal of Infection. 2020;80:607-13.
10. Zhang W, Zhao Y, Zhang F, Wang Q, Li T, Liu Z, *et al*. The use of anti-inflammatory drugs in the treatment of people with severe coronavirus disease 2019 (COVID-19): The perspectives of clinical immunologists from China. Clin Immunol 2020;214:108393.
11. Conte L, Toraldo DM. Targeting the gut–lung microbiota axis by means of a high-fibre diet and probiotics may have anti-inflammatory effects in COVID-19 infection. Ther Adv Respir Dis 2020;14:1753466620937170.
12. Leandro CG, Ferreira E Silva WT, Lima-Silva AE. Covid-19 and exercise-induced immunomodulation. Neuroimmunomodulation 2020;27:75-8.
13. Shivappa N, Hébert JR, Rietzschel ER, De Buyzere ML, Langlois M, Debruyne E, *et al*. Associations between dietary inflammatory index and inflammatory markers in the Asklepios Study. Br J Nutr 2015;113:665-71.
14. Shivappa N, Hebert JR, Marcos A, Diaz LE, Gomez S, Nova E, *et al*. Association between dietary inflammatory index and inflammatory markers in the HELENA study. Mol Nutr Food Res 2017;61:10.1002/mnfr.201600707.
15. van Woudenbergh GJ, Theofylaktopoulou D, Kuijsten A, Ferreira I, van Greevenbroek MM, van der Kallen CJ, *et al*. Adapted dietary inflammatory index and its association with a summary score for low-grade inflammation and markers of glucose metabolism: The Cohort study on Diabetes and Atherosclerosis Maastricht (CODAM) and the Hoorn study. Am J Clin Nutr 2013;98:1533-42.
16. Wood LG, Shivappa N, Berthon BS, Gibson PG, Hebert JR. Dietary inflammatory index is related to asthma risk, lung function and systemic inflammation in asthma. Clin Exp Allergy 2015;45:177-83.
17. Ye K, Tang F, Liao X, Shaw BA, Deng M, Huang G, *et al*. Does serum vitamin d level affect covid-19 infection and its severity?-A case-control study. J Am Coll Nutr 2020;40:724-31.
18. Ilie PC, Stefanescu S, Smith L. The role of vitamin D in the prevention of coronavirus disease 2019 infection and mortality. Aging Clin Exp Res 2020;32:1195-8.
19. Hathaway D III, Pandav K, Patel M, Riva-Moscoso A, Singh BM, Patel A, *et al*. Omega 3 fatty acids and COVID-19:

A comprehensive review. Infect Chemother 2020;52:478-95.
20. Kieliszek M, Lipinski B. Selenium supplementation in the prevention of coronavirus infections (COVID-19). Med Hypotheses 2020;143:109878.
21. Skalny AV, Rink L, Ajsuvakova OP, Aschner M, Gritsenko VA, Alekseenko SI, *et al*. Zinc and respiratory tract infections: Perspectives for COVID-19. Int J Mol Med 2020;46:17-26.
22. Weill P, Plissonneau C, Legrand P, Rioux V, Thibault R. May omega-3 fatty acid dietary supplementation help reduce severe complications in Covid-19 patients? Biochimie 2020;179:275-80.
23. Iddir M, Brito A, Dingeo G, Fernandez Del Campo SS, Samouda H, La Frano MR, *et al*. Strengthening the immune system and reducing inflammation and oxidative stress through diet and nutrition: Considerations during the COVID-19 crisis. Nutrients 2020;12:1562.
24. Shrestha A, Koju RP, Beresford SA, Chan KCG, Connell FA, Karmacharya BM, *et al*. Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for Nepalese diet. International journal of food sciences and nutrition. 2017;68:605-12.
25. Ghaffarpour M, Houshiar-Rad A, Kianfar H. The manual for household measures, cooking yields factors and edible portion of foods. Tehran: Nashre Olume Keshavarzy. 1999;7:42-58.
26. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr 2014;17:1689-96.
27. Aslani Z, Qorbani M, Hébert JR, Shivappa N, Motlagh ME, Asayesh H, *et al*. Association of Dietary Inflammatory Index with anthropometric indices in children and adolescents: The weight disorder survey of the Childhood and Adolescence Surveillance and Prevention of Adult Non-communicable Disease (CASPIAN)-IV study. Br J Nutr 2019;121:340-50.
28. Ayala DJM-F, Navas P, López-Lluch G. Age-related mitochondrial dysfunction as a key factor in COVID-19 disease. Experimental Gerontology. 2020:111147.
29. Michelozzi P, de'Donato F, Scortichini M, De Sario M, Noccioli F, Rossi P, *et al.* Mortality impacts of the coronavirus disease (COVID-19) outbreak by sex and age: rapid mortality surveillance system, Italy, 1 February to 18 April 2020. Eurosurveillance. 2020;25:2000620.
30. Pinto LC, Bertoluci MC. Type 2 diabetes as a major risk factor for COVID-19 severity: a meta-analysis. Archives of endocrinology and metabolism. 2020;64:199-200.
31. Varikasuvu SR, Varshney S, Dutt N. Markers of coagulation dysfunction and inflammation in diabetic and non-diabetic COVID-19. Journal of Thrombosis and Thrombolysis. 2020:1-6.
32. Khoramipour K, Basereh A, Hekmatikar AA, Castell L, Ruhee RT, Suzuki K. Physical activity and nutrition guidelines to help with the fight against COVID-19. Journal of Sports Sciences. 2021;39:101-7.
33. Kossoff EH, Turner Z, Adams J, Bessone SK, Avallone J, McDonald TJ, *et al.* Ketogenic diet therapy provision in the COVID-19 pandemic: dual-center experience and recommendations. Epilepsy & Behavior. 2020;111:107181.
34. Paoli A, Gorini S, Caprio M. The dark side of the spoon-glucose, ketones and COVID-19: a possible role for ketogenic diet? Journal of Translational Medicine. 2020;18:1-9.
35. Naja F, Hamadeh R. Nutrition amid the COVID-19 pandemic: a multi-level framework for action. European journal of clinical nutrition. 2020;74:1117-21.
36. Aman F, Masood S. How Nutrition can help to fight against COVID-19 Pandemic. Pak J Med Sci. 2020;36(COVID19-S4):S121-S3. PubMed PMID: 32582329.
37. Post A, Dullaart RP, Bakker SJ. Is low sodium intake a risk factor for severe and fatal COVID-19 infection? European journal of internal medicine. 2020.
38. Wirth MD, Hébert JR, Shivappa N, Hand GA, Hurley TG, Drenowatz C, *et al*. Anti-inflammatory Dietary Inflammatory Index scores are associated with healthier scores on other dietary indices. Nutrition research. 2016;36:214-9.
39. Morais AHdA, Aquino JdS, da Silva-Maia JK, Vale SHdL, Maciel BLL, Passos TS. Nutritional status, diet and viral respiratory infections: perspectives for severe acute respiratory syndrome coronavirus 2. The British journal of nutrition. 2020:1-12.
40. Wood LG, Shivappa N, Berthon BS, Gibson PG, Hebert JR. Dietary inflammatory index is related to asthma risk, lung function and systemic inflammation in asthma. Clin Exp Allergy. 2015;45:177-83. PubMed PMID: 24708388.
41. Han Y-Y, Forno E, Shivappa N, Wirth MD, Hébert JR, Celedón JC. The Dietary Inflammatory Index and Current Wheeze Among Children and Adults in the United States. J Allergy Clin Immunol Pract. 2018;6:834-41.e2. PubMed PMID: 29426751. Epub 02/14. eng.
42. Stolarczyk E. Adipose tissue inflammation in obesity: a metabolic or immune response? Current opinion in pharmacology. 2017;37:35-40.
43. Valdearcos M, Douglass JD, Robblee MM, Dorfman MD, Stifler DR, Bennett ML, *et al.* Microglial inflammatory signaling orchestrates the hypothalamic immune response to dietary excess and mediates obesity susceptibility. Cell metabolism. 2017;26:185-97.