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Dietary Intake of Patients Recovered from COVID-19

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Abstract: COVID-19 is a multisystemic infection with profound physiological and behavioral sequelae, including marked disruption of dietary patterns. This study comprehensively analyzed the effect of the infection on nutrient intake by comparing dietary consumption pre and post COVID-19 and estimated dietary intake against the Estimated Average Requirement (EAR, 2020). Intake data were collected using 72-hour dietary recall questionnaires and measured retrospectively for both the periods. The overall differences between pre- and post-COVID-19 periods were highly statistically significant ($p < 0.0001$, $\alpha = 0.01$) for all macronutrients-energy, protein, fat, carbohydrates, and fiber-and all micronutrients- vitamins A, C, D, E, folate and minerals such as iron, calcium, sodium, potassium, copper, zinc, magnesium, and selenium. When compared against EAR 2020, all nutrients, except vitamin D ($p = 0.62$) and iron ($p = 0.47$), demonstrated significant differences from the recommended levels at $p < 0.0001$, indicating a widespread deviation from the EAR 2020. It was also seen that the dietary intake post COVID-19 was increased for fiber, vitamins A, C, D, E, folate and minerals such as calcium, sodium, potassium, copper, zinc, magnesium, and selenium, but were reduced for energy, protein, fat, carbohydrates, and iron. Overall, the post-infection eating pattern indicated an extreme diversion from recommended standards, with overconsumption being the prevailing tendency. These results emphasize the importance of targeted nutritional interventions in post-COVID-19 rehabilitation to redress imbalances for the support of metabolic and immune rehabilitation.

Keywords: COVID-19, Nutrition, Macro nutrients, Vitamins, Minerals, Infection, EAR 2020

Introduction: The COVID-19 pandemic, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), represents one of the most devastating global health crises in modern history, with over 7 million confirmed deaths and profound multisystem morbidity (WHO, 2023). Phylogenomic analyses indicate that the SARS-CoV-2 virus originated in bats of the Rhinolophus species, with likely intermediate spillover via pangolins, as evidenced by >96% nucleotide identity with the bat coronavirus, RaTG13 and shared receptor-binding domain motifs with the pangolin-CoV (Zhou, et al., 2020; Lam, et al., 2020; Andersen, et al., 2020). This virus engages host cells via the angiotensin-converting enzyme 2 (ACE2), highly expressed in respiratory, gastrointestinal, renal, and vascular endothelia, explaining its multi-systemic effects (Hoffmann, et al., 2020). Clinically, the infection manifests across a continuum: from asymptomatic carriage to acute respiratory distress syndrome, commonly known as ARDS, multiorgan dysfunction, and post-acute sequelae, also called, Long COVID (Huang, et al., 2021). The early-pandemic variants (e.g., ancestral, Alpha) predominantly presented with upper respiratory symptoms like anosmia, rhinorrhea, sore throat, and dry cough, due to their high ACE2/TMPRSS2 co-expression in nasal epithelium (Sungnak, et al., 2020). With continued viral evolution (e.g., the Omicron lineages), the infection focus shifted

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

towards the upper airways, altering symptom profiles but not actually reducing the systemic sequelae, including gastrointestinal, neurocognitive, and autonomic dysfunction.

As the pandemic evolved, so did the symptoms and the effects of the virus itself. One of the most widely discussed facet in this whole pandemic has been immunity improvement as it has been proven again and again that one of the safest ways to avoid or mitigate the effects of the virus is to improve one's immunity levels. In order to do so, nutrition has emerged as the prime contender for the job by profoundly modulating the host immune system and thus affecting the symptom trajectory of the disease itself. Immune activation demands surge in energy substrates (glucose, amino acids, fatty acids) and biosynthetic precursors; for instance, T-cell proliferation requires nucleotides (dependent on folate, B12, zinc), protein synthesis (arginine, glutamine), and membrane phospholipids (Calder, et al., 2020). Micronutrients serve as enzymatic cofactors, like selenium for glutathione peroxidase, zinc for thymulin, etc. and also serve as direct transcriptional regulators, with vitamin D modulating the cathelicidin and defensin expression via the vitamin D receptor, while retinoic acid (a vitamin A metabolite) governs mucosal immunity and T-cell differentiation (Aranow, 2011; Huang, et al., 2021). Antioxidant defenses, critical for mitigating NADPH oxidase driven oxidative bursts during phagocytosis, rely on vitamins C and E and selenium dependent selenoproteins. Critically, malnutrition impairs both innate immunity, specifically, neutrophil chemotaxis and natural killer cells' cytotoxicity and also affects adaptive immunity by influencing T cell and B cell clonal expansions, antibody affinity maturation, etc., thus increasing susceptibility, severity, and mortality (Jayawardena, et al., 2020). Conversely, SARS-CoV-2 infection itself disrupts nutritional homeostasis, by giving rise to issues like dysgeusia and anosmia, which reduce appetite, cytokine-driven anorexia, gastrointestinal involvement, muscle catabolism and micronutrient sequestration all of which can collectively induce a negative energy and protein balance (Iddir, et al., 2020). This bidirectional nutrient-infection axis is now well documented. It has also been seen that while pre-infection nutritional deficits amplify severity, acute and post-acute infection also disturbs dietary intake, anthropometry and biochemical markers.

Accordingly, this study investigates the nutritional impact of SARS-CoV-2 infection in recovered individuals, employing a validated 72-hour dietary recall questionnaire designed to elicit information about the pre and post-infection intake of macro and micronutrients. By then benchmarking the obtained data against India's Estimated Average Requirements (EAR, 2020), the study aims to objectively characterize dietary disruption, providing evidence for targeted nutritional rehabilitation in post COVID-19 care.

Methodology:

Selection of Sample: In the present study, according to a sample size selection formula, known as the Cochran Formula (Cochran, 1977), 384 adults (males and females) were to be selected. However, looking into the all pervasive nature of this pandemic, it was decided to increase this number to 500 subjects (250 males and 250 females). A total of 511 subjects (255 males and 256 females) had all the required information and were included in the study. Along with the information provided, biochemical tests were conducted on all 511 subjects to elicit the post COVID-19 parameters. All the subjects selected had a medically confirmed diagnosis of COVID-19 and all of them were from the urban areas of Nagpur city, over the age of 21 years.

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

Sampling Methods: The sampling method used in the present study was purposive sampling.

Data Collection: The data was collected using a 72 hours' dietary recall questionnaire which was designed to elicit information about the pre and post COVID-19 consumption patterns of macronutrients (energy, proteins, fats, carbohydrates and fiber) and micronutrients (vitamins A, C, E, D, folate and minerals iron, calcium, sodium, potassium, copper, zinc, magnesium, and selenium).

Hypotheses of the Study: The following were considered as the hypotheses for the study:

H0: There is no significant difference in the nutrient intake of pre and post COVID-19 patients.

H0: There is no significant difference between the Estimated Average Intake (EAR 2020) and the nutrient intake of pre and post COVID-19 patients.

Statistical Analysis: A host of different types of statistics were used in the present study that analyzed the interrelation of each and every variable with other variables. The statistical tests applied for the nutritional parameters were Descriptive statistic, t Tests (Paired t Test & 1 Sample t Test) to compare the intake before and after COVID-19 and to compare the consumption of nutrients intake with the EAR, both before and after COVID-19.

Results and Discussions: The study showed statistically significant results for the macro and micronutrient consumption.

Table 1: Macronutrient Intake of Pre and Post COVID-19 Patients

Sr. No	**Nutrients**	**Pre COVID-19**		**Post COVID-19**		**Range**		**Mean Differences**	**t Stat**	**p Value**
		Mean	**SD**	**Mean**	**SD**	**Min**	**Max**			
1	**Energy (Kcals)**	2098.25	±221.37	1942.06	±188.91	Pre: 1663.78	Pre: 2846.78	-156.19	13.25	<0.0001
						Post: 1443.75	Post: 2459.77			
2	**Proteins (g)**	61.82	±12.60	59.18	±15	Pre: 32.03	Pre: 122.55	-2.60	3.05	<0.0001
						Post: 28.86	Post: 133.42			
3	**Fats (g)**	34.71	±10.07	31.39	±12.14	Pre: 5.67	Pre: 69.58	-3.316	4.86	<0.0001
						Post: 3.62	Post: 58.96			

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

4	**Carbohydrates (g)**	384.78	±61.41	355.68	±61.39	Pre: 231.36	Pre: 575.11	-29.10	7.90	<0.0001
						Post: 114.25	Post: 519.35			
5	**Fiber (g)**	27.75	±9.79	29.39	±6.77	Pre: 9.2	Pre: 64.58	+1.37	-3.16	<0.0001
						Post: 11.44	Post: 51.88			

t Critical two-tail: 2.58

For the macronutrients, the t statistic that showed the highest difference was in the pre and post COVID energy consumption (t=13.25, with a mean difference of 156.1 kcal) followed by carbohydrates (showing a mean difference of -29.1g and t = 7.90), fats (with a mean difference of -3.3g and t = 4.86) and the smallest difference was between the pre and post COVID proteins consumption (t=3.05 and a mean intake difference of -2.6g). The overall trend was towards a reduction of intake of the macronutrients post COVID with fiber being the exception as a mean increase of 1.37g was seen in the post COVID recall. All comparisons achieved $p < 0.0001$, far exceeding the critical t-value of 2.58 for a two-tailed test at $\alpha = 0.01$, indicating robust and clinically meaningful changes in dietary behavior post-infection. This pattern of macronutrient restriction aligns with emerging evidence that acute and post-acute SARS-CoV-2 infection induces a state of "metabolic anorexia" driven by systemic inflammation. Pro-inflammatory cytokines, including IL-1β, IL-6, and TNF-α, suppress appetite via hypothalamic signaling and reduce gastric motility, contributing to reduced caloric intake (Iddir et al., 2020). The observed 7.4% reduction in daily energy intake (from 2098 to 1942 kcal) also mirrors findings from multiple hospitalized cohorts, where energy expenditure remains elevated due to fever and respiratory effort, yet intake is suppressed, causing an imbalance that promotes catabolism and muscle wasting. This is particularly concerning given that protein intake fell by 4.2% (from 61.8 to 59.2 g/day), a level insufficient to counteract sarcopenia, especially in older adults or those with pre-existing comorbidities (Landi, et al., 2021). The decline in carbohydrate and fat intake may reflect both behavioral adaptation (e.g., loss of taste/smell altering food preferences) and metabolic reprogramming. The infection is also known to trigger insulin resistance even in non-diabetic individuals, potentially reducing carbohydrate tolerance and leading to self-imposed restriction (Montefusco et al., 2021). Simultaneously, lipid metabolism is profoundly disrupted as plasma HDL-C and ApoA1 levels drop acutely due to hepatic down-regulation and increased clearance, while triglycerides rise, due to a phenomenon linked to impaired mitochondrial β-oxidation and increased de novo lipogenesis (Nguyen et al., 2022). Reduced fat intake post-COVID may represent an unconscious attempt to mitigate this dyslipidemia, though it risks depriving the body of essential fatty acids needed for membrane repair during immune recovery. Surprisingly, fiber intake increased significantly with an increase of +1.37 g/day, suggesting a potential, compensatory shift toward plant-based or whole-food diets. This could be interpreted as a positive adaptive response, as dietary fiber

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

enhances gut microbiota diversity, promotes short-chain fatty acid (SCFA) production (notably butyrate), and modulates systemic inflammation (Valdes et al., 2018). The SCFAs produced due to higher fiber intake also support regulatory T-cell differentiation, which is critical for resolving post-viral immune hyperactivity (Arpaia et al., 2013).

The next group of nutrients analyzed were the vitamins which showed highly significant differences between the pre and post COVID intake.

Table 2: Vitamin Intake of Pre and Post COVID-19 Patients

Sr. No	Nutrients	Pre COVID-19		Post COVID-19		Range		Mean Differences	t Stat	p Value
		Mean	SD	Mean	SD	Min	Max			
1	Vitamin A (mcg)	521.88	±185.98	673.57	±172.84	Pre: 212.4	Pre: 1118.28	-151.69	-13.55	<0.0001
						Post: 97.98	Post: 1228.51			
2	Vitamin C (mg)	31.25	±11.42	54.61	±16.593	Pre: 2.21	Pre: 65.75	-23.36	-26.45	<0.0001
						Post: 8.4	Post: 108.55			
3	Vitamin D (mcg)	5.85	±3.10	10.07	±3.21	Pre: 4.5	Pre: 15.48	-4.21	-20.87	<0.0001
						Post: 1.12	Post: 20.74			
4	Vitamin E (mg)	10.34	±2.17	12.69	±2.30	Pre: 3.01	Pre: 17.25	-2.35	-16.80	<0.0001
						Post: 6.37	Post: 18.7			
5	Folate (mcg)	148.88	±33.75	185.72	±35.75	Pre: 37.66	Pre: 240.3	-36.84	-17.80	<0.0001
						Post: 72.31	Post: 283.1			

t Critical two-tail: 2.58

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

The study revealed statistically significant increases in the dietary intake of all five assessed micronutrients, vitamins A, C, D, E and folate, post COVID (all $p < 0.0001$), with t-statistics ranging from -13.55 for vitamin A, to -26.45 for vitamin C, far exceeding the critical two-tailed t-value of 2.58 at $\alpha = 0.01$. This uniform elevation suggests a consistent behavioral or physiological shift toward increased consumption of micronutrient-dense foods post-infection, potentially reflecting either conscious dietary compensation or altered taste/smell perception driving food selection. The most pronounced increase was observed for vitamin C with a 23.3mg increase in consumption and a t statistic of -26.45, followed by vitamin D with a mean intake increase of 4.2µg and a t statistic of -20.87. Vitamin E also showed an increased intake of 2.3mg and a t statistic of -16.80, while folate demonstrated an increase of 36.8µg and the t statistic of -17.80. Lastly, vitamin A, which had the smallest t statistic of 13.55, showed an increased consumption of 151.6µg. Notably, these increases occurred despite no formal nutritional intervention, implying that patients may have spontaneously adopted diets richer in foods naturally abundant in these vitamins. This pattern aligns with emerging evidence that post-COVID recovery involves active modulation of antioxidant balance, immune regulation, and tissue repair, all of which are processes that are heavily dependent on antioxidant and cofactor vitamins. Vitamin C, for instance, is not only a potent scavenger of reactive oxygen species generated during neutrophil respiratory bursts but also a cofactor for collagen synthesis and epigenetic regulation (Carr, et al., 2017). Its dramatic rise post-infection may reflect an adaptive response to counteract persistent oxidative stress, which has been documented in Long COVID patients even months after acute resolution. Similarly, the near-doubling of vitamin D intake (from 5.85 to 10.07 µg/day) is clinically significant and represents a welcome and meaningful shift toward correcting widespread pre-pandemic insufficiency. Vitamin D deficiency is a well-established risk factor for severe COVID-19, linked to impaired interferon signaling, reduced cathelicidin production and heightened pro-inflammatory cytokine release (Aranow, 2011; Grant et al., 2020). The observed increase may indicate patient awareness or subconscious dietary adjustment to bolster innate immunity as a lot of information was made available in the public domain regarding the dietary requirements useful for combating the widespread infection. Vitamin E intake also rose by 22.7% (from 10.34 to 12.69 mg/day), consistent with its role as a lipid-soluble antioxidant protecting cellular membranes from peroxidation — especially relevant in the context of post-COVID dyslipidemia and endothelial dysfunction (Traber, et al., 1999). Its elevation may also reflect increased consumption of plant oils, nuts, and seeds, all of which are foods often associated with “health-conscious” dietary patterns adopted during recovery. The intake of folate increased by 24.7% (from 148.88 to 185.72 µg/day), a change that may support nucleotide synthesis required for immune cell proliferation and DNA methylation-dependent gene silencing during inflammation resolution (Calder et al., 2020). Importantly, folate is also a methyl donor for homocysteine re-methylation and elevated homocysteine is a known risk factor for thromboembolic events in COVID-19 (Zhao et al., 2021). The 151.69 µg increase in vitamin A is particularly interesting. While excessive preformed vitamin A can be hepatotoxic, moderate increases from dietary sources enhance mucosal immunity via retinoic acid signaling, promoting IgA secretion and gut barrier integrity, all of which are critical for preventing secondary infections in post-viral states (Ivanov et al., 2006). Given that anosmia and ageusia were common early symptoms,

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

the rise in vitamin A intake may reflect improved olfactory and gustatory function enabling greater consumption of flavorful, nutrient-dense foods.

The last group of micronutrients analyzed consisted of minerals, which also showed a significant difference between the pre and post COVID intake.

Table 3: Mineral Intake of Pre and Post COVID-19 Patients

Sr. No	Nutrients	Pre COVID-19		Post COVID-19		Range		Mean Differences	t Stat	p Value
		Mean	SD	Mean	SD	Min	Max			
1	Iron (mg)	14.84	±3.84	13.11	±3.73	Pre: 7.2 Post: 7.7	Pre: 26.1 Post: 24.59	1.73	7.19	<0.0001
2	Calcium (mg)	526.61	±164.34	645.88	±152.93	Pre: 149.1 Post: 201.93	Pre: 1063.98 Post: 1312.36	-119.27	-11.72	<0.0001
3	Sodium (mg)	3690.80	±680.50	3986.77	±706.05	Pre: 1722.76 Post: 1884.78	Pre: 5620.13 Post: 6205.46	-295.96	-7.03	<0.0001
4	Potassium (mg)	1197.97	±220.29	3403.02	±682.52	Pre: 515.65 Post: 1032.58	Pre: 1883.24 Post: 5553.75	-2205.04	-69.17	<0.0001
5	Copper (mg)	1.15	±0.62	1.40	±0.39	Pre: 0.2 Post: 0.3	Pre: 2.94 Post: 2.4	-0.24	-7.53	<0.0001
6	Zinc (mg)	9.70	±2.48	10.95	±2.87	Pre: 1.49 Post: 2.35	Pre: 17.05 Post: 18.76	-1.24	-7.25	<0.0001
7	Magnesium (mg)	326.40	±62.01	347.92	±74.40	Pre: 116.03 Post: 130.13	Pre: 498.78 Post: 574.74	-21.51	-4.95	<0.0001
8	Selenium (mcg)	44.59	±7.90	50.84	±9.76	Pre: 20.63 Post: 24.33	Pre: 69.79 Post: 81.14	-6.24	-11.11	<0.0001

t Critical two-tail: 2.58

The analysis reveals statistically significant alterations in the dietary intake of all eight assessed minerals post COVID (all $p < 0.0001$), with t-statistics ranging from -4.95 for magnesium to -69.17 for potassium, far exceeding the critical two-tailed t-value of 2.58 at $\alpha = 0.01$. The iron intake decreased significantly post-COVID with a mean difference of 1.73 mg, showing a t

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

statistic of 7.19, despite the well-documented "anemia of inflammation" (where the iron levels are low and the ferritin levels are normal to high), driven by the inflammatory protein hepcidin's up-regulation during acute infection. This suggests that patients could have been consciously avoiding iron-rich foods due to altered taste and/or smell, gastrointestinal discomfort, potentially exacerbating functional iron deficiency. While ferritin levels often rise during acute COVID-19 due to inflammation, tissue iron availability remains low, impairing mitochondrial respiration and immune cell proliferation (Ganz, et al., 2015). The observed reduction in dietary iron may thus contribute to persistent fatigue and impaired recovery, particularly in women and older adults. Next, calcium intake rose substantially to119.2mg and a t statistic of -11.72, likely reflecting increased consumption of dairy products, fortified plant milk or leafy greens, foods commonly associated with "recovery diets." Similarly, magnesium intake increased modestly by 21.5mg and showed a t statistic of -4.95, consistent with its role as a cofactor for over 300 enzymatic reactions, including ATPase activity, DNA repair and inflammation regulation (Chacko et al., 2021). Given that hypo-magnesemia is linked to increased pro inflammatory IL-6 and TNF-α levels, this increase may represent an adaptive response to mitigate post-viral inflammation. Sodium intake also increased significantly by 295.9mg and a t statistic of 7.03, while potassium intake surged dramatically with a whopping 2205mg increase that had a t statistic of -69.17. The massive rise in potassium could potentially suggest a shift toward potassium-rich whole foods. Potassium is critical for maintaining membrane potential, regulating blood pressure, and modulating inflammation (Munoz-Planillo et al., 2013). Its elevation may reflect conscious or subconscious dietary compensation to counteract endothelial dysfunction and autonomic dysregulation, particularly common in Long COVID. Conversely, the rise in sodium, may stem from increased consumption of soups, broths, or electrolyte beverages used to combat dehydration or fatigue, a pragmatic but potentially risky strategy, which, if sustained, could lead to hypertension and vascular stiffness along with a host of cardiometabolic issues. With respect to the trace minerals, zinc intake increased significantly with a 1.24 mg increase and a t statistic of -7.25, aligning with its critical role as a cofactor for over 300 enzymes, including those involved in DNA synthesis, antioxidant defense and antiviral immunity (Wessels et al., 2020). Copper intake also rose by 0.24 mg and showed a t statistic of -7.53), supporting ceruloplasmin synthesis and superoxide dismutase activity, both crucial for managing oxidative stress. Notably, copper and zinc share absorption pathways and their concurrent rise suggests balanced dietary intake rather than isolated supplementation. Lastly, selenium intake increased by 14% (6.24 µg) and had a t statistic of 11.11, reinforcing its importance in the glutathione peroxidase and thioredoxin reductase activity, the key enzymes in neutralizing hydrogen peroxide and lipid hydroperoxides generated during immune activation. Selenium status has been directly linked to COVID-19 severity, with low serum selenium correlating with higher mortality, while adequate intake reducing pro-inflammatory cytokine production (Zhao et al., 2021).

Following the analyses of the differences between the pre and post COVID intake of the nutrients, the study also focused on investigating the deviations of the dietary intake, both pre and post COVID, from the recommended levels as dictated by the National Institute of Nutrition (NIN, Hyderabad) by the way of Estimated Average Requirements (EAR, 2020).

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

For the macronutrients, the consumption of energy, proteins, fats, carbohydrates and fiber was considered, both pre and post COVID.

Table 7: Nutrient Adequacy Ratios for Macronutrients

Sr. No	Nutrients	Mean	SD	EAR	NAR %	t Stat
1	Energy Pre (kcals)	2098.26	±221.37	1885	111.31	21.77
2	Energy Post (kcals)	1942.07	±188.91	1885	103.03	6.28
3	Proteins Pre (g)	61.83	±12.60	39.6	156.13	39.88
4	Proteins Post (g)	59.19	±15.01	39.6	149.46	29.5
5	Fats Pre (g)	34.71	±10.08	22.5	154.28	27.38
6	Fats Post (g)	31.40	±12.15	22.5	139.55	16.55
7	Carbohydrates Pre (g)	384.79	±61.42	100	384.79	104.81
8	Carbohydrates Post (g)	355.69	±61.39	100	355.69	94.15
9	Fiber Pre (g)	27.75	±9.80	40	69.39	-28.25
10	Fiber Post (g)	29.40	±6.78	40	73.50	-35.34

The Nutrient Adequacy Ratios (NAR) were calculated as (Mean Intake / Estimated Average Requirement [EAR] × 100) and revealed a complex and clinically significant shift in dietary adequacy, especially post COVID. While pre-infection intake exceeded EARs for energy, protein, fat, and carbohydrates, indicating relative sufficiency, post-infection values show a consistent decline across all macronutrients, with fiber remaining chronically inadequate both before and after illness. The pre COVID energy intake (2098 kcal/day) exceeded the EAR (1885 kcal/day) by 11.3% (NAR = 111.31, t = 21.77), suggesting adequate caloric support for baseline metabolic needs. Post-infection, however, energy intake fell to 1942 kcal/day, still above the EAR but only marginally so (NAR = 103.03; t = 6.28). This 7.4% reduction aligns with well documented "metabolic anorexia," both during and after acute infection, driven by pro-inflammatory cytokines like IL-1β, IL-6, TNF-α, etc., suppressing appetite and increasing catabolic drive. The protein intake analyses showed even more dramatic changes. The pre COVID NAR was 156.13% (61.8 g vs. EAR 39.6 g), reflecting a substantial surplus, likely sufficient to support immune cell proliferation and tissue repair functions. However, post COVID, while still exceeding EAR (NAR = 149.46%), the absolute intake dropped by 4.3% (to 59.2 g/day), potentially reducing the safety margin for meeting increased protein demands during recovery. Given that protein requirements rise by 20-30% during convalescence from severe illness (Singer et al., 2019), this marginalization may contribute to sarcopenia and impaired immune reconstitution, particularly in older adults or those with comorbidities (Landi, et al., 2021). Next, the fats intake pre-COVID was 154.28% of EAR (34.7 g vs. 22.5

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

g), consistent with global trends toward high-fat diets. Post-infection, however, it declined to 139.55% (31.4 g), still adequate but reduced and potentially reflecting behavioral adaptation to post-COVID dyslipidemia (Nguyen et al., 2022). While not deficient, this decline may potentially deprive the body of essential fatty acids needed for eicosanoid synthesis and membrane repair functions during immune recovery. The carbohydrate intake was massively excessive pre-COVID (NAR = 384.79%), far beyond physiological need (EAR = 100 g/day). This aligns with typical Indian dietary patterns dominated by refined grains (rice, wheat) and sugary snacks. Post-infection, carbohydrate intake decreased only slightly (NAR = 355.69%), suggesting no meaningful dietary recalibration toward balanced macronutrient distribution. Such chronic overconsumption may exacerbate insulin resistance and inflammation, both of which are implicated in Long COVID pathophysiology (Montefusco et al., 2021). The most alarming finding is the persistent inadequacy of dietary fiber. The pre-COVID NAR was only 69.39% (27.8 g vs. EAR 40 g), and post-COVID rose modestly to 73.50% (29.4 g), thus, still falling short of the recommendations. This deficiency is not unique to our cohort as national surveys consistently show <10% of Indians meet fiber targets (National Institute of Nutrition, ICMR, 2017). Fiber insufficiency impairs gut microbiota diversity, reduces SCFA production (butyrate, propionate) and weakens mucosal immunity, all of which are critical for resolving post-viral inflammation and preventing secondary infections (Valdes et al., 2018; Arpaia et al., 2013). The modest post-COVID increase (+1.65 g), while statistically significant (t = -35.34), remains clinically insufficient to correct dysbiosis or modulate systemic immunity.

The next group analyzed was that of the vitamins A, C, D, E and folate.

Table 8: Nutrient Adequacy Ratios for Vitamins

Sr. No	Nutrients	Mean	SD	EAR	NAR	t Stat
1	Vitamin A Pre (mcg)	521.88	±185.99	425	122.80	11.77
2	Vitamin A Post (mcg)	673.58	±172.84	425	158.49	32.51
3	Vitamin C Pre (mg)	31.26	±11.42	60	52.09	-56.88
4	Vitamin C Post (mg)	54.62	±16.59	60	91.03	-7.33
5	Vitamin D Pre (mcg)	5.86	±3.11	10 (400IU)	58.60	-30.09
6	Vitamin D Post (mcg)	10.07	±3.21	10 (400 IU)	100.70	0.49*
7	Vitamin E Pre (mg)	10.34	±2.18	10	103.40	3.52
8	Vitamin E Post (mg)	12.69	±2.31	10	126.95	26.32
9	Folate Pre (mcg)	148.88	±33.76	215	69.25	-44.27
10	Folate Post (mcg)	185.73	±35.75	215	86.39	-19.6

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

Even here, while pre-infection intake was inadequate for vitamins C, D and folate and marginally sufficient for vitamin E, post infection values show marked improvement across all vitamins, with vitamin A and E achieving optimal levels, vitamin D reaching adequacy, and vitamin C and folate approaching sufficiency. This pattern suggests a spontaneous, adaptive dietary recalibration toward immune-supportive nutrients though maybe not potentially sufficient to meet the optimal physiological demands during recovery. The pre-COVID vitamin A intake (521.88 µg) exceeded the EAR (425 µg) by 22.8% (NAR = 122.80; t = 11.77), indicating baseline adequacy. Post-infection, the intake rose dramatically to 673.58 µg (NAR = 158.49; t = 32.51), far surpassing the EAR for adults. Vitamin A is critical for mucosal immunity via retinoic acid signaling, promoting IgA secretion and gut barrier integrity, all of which are essential for preventing secondary infections in post-viral states (Ivanov et al., 2006). However, excessive preformed vitamin A (>3,000 µg/day) can be hepatotoxic. Our cohort's intake, however, remains within safe limits, suggesting a beneficial adaptation rather than over-supplementation. Next, pre-COVID vitamin C intake was alarmingly low (31.26 mg/day; NAR = 52.09; t = -56.88), falling far below the EAR of 60 mg/day, a level associated with impaired neutrophil function, collagen synthesis, and antioxidant capacity (Carr, et al., 2017). Post-infection, the intake increased to 54.62 mg/day (NAR = 91.03; t = -7.33), approaching adequacy but still below the recommended threshold shown to saturate plasma and leukocyte levels for optimal immune function (Hemila, et al., 2013). This near-correction may be due to the increased fruit/vegetable consumption driven by patient awareness or altered taste and/or smell perception. Nevertheless, the persistent deficit underscores the need for targeted supplementation in post-COVID populations, especially given vitamin C's role in mitigating oxidative stress and supporting T-cell proliferation. The pre-COVID vitamin D intake (5.86 µg/day; NAR = 58.60; t = -30.09) was severely deficient and consistent with widespread insufficiency in India due to limited sun exposure, dietary scarcity and genetic factors (Holick, 2004). Post-infection, the intake rose to 10.07 µg/day (NAR = 100.70; t = 0.49), meeting the EAR (10 µg/day). This marginal improvement may reflect conscious dietary changes or behavioral shifts like increased outdoor activity during recovery. Given that vitamin D deficiency is a well-established risk factor for severe COVID-19 and is linked to impaired interferon signaling and heightened pro-inflammatory cytokine release (Aranow, 2011), this partial correction is clinically meaningful but insufficient without supplementation in high-risk individuals. Vitamin E intake was adequate pre-COVID (10.34 mg/day; NAR = 103.40; t = 3.52), just above the EAR (10 mg/day). Post-infection, it rose significantly to 12.69 mg/day (NAR = 126.95; t = 26.32), aligning with its role as a lipid-soluble antioxidant protecting cellular membranes from peroxidation, especially relevant in the context of post-COVID dyslipidemia and endothelial dysfunction (Traber, et al., 1999). Its elevation may also reflect increased consumption of plant oils, nuts, and seeds, all of which are foods often associated with "health-conscious" dietary patterns adopted during recovery. The folate intake improved post-COVID (from 148.88 to 185.73 µg/day; NAR from 69.25 to 86.39; t = -19.6), but remained below the EAR (215 µg/day). This chronic deficiency is not unique to our cohort; national surveys consistently show <20% of Indians meet folate targets (National Institute of Nutrition, ICMR, 2017). Folate is essential for nucleotide synthesis required for immune cell proliferation and DNA methylation-dependent gene silencing during inflammation resolution

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

(Calder et al., 2020). Its persistent inadequacy may contribute to impaired lymphocyte clonal expansion and prolonged fatigue, especially in cases of Long COVID.

The last group of nutrients to be compared to the EAR for obtaining the NAR was that of the minerals.

Table 9: Nutrient Adequacy Ratios for Minerals

Sr. No	Nutrients	Mean	SD	EAR	NAR	t Stat
1	**Iron Pre (mg)**	14.85	±3.84	13	114.22	10.89
2	**Iron Post (mg)**	13.12	±3.74	13	100.90	0.72*
3	**Calcium Pre (mg)**	526.62	±164.35	800	65.83	-37.6
4	**Calcium Post (mg)**	645.89	±152.93	800	80.74	-22.77
5	**Magnesium Pre (mg)**	326.41	±62.01	295	110.65	11.45
6	**Magnesium Post (mg)**	347.92	±74.41	295	117.94	16.07
7	**Zinc Pre (mg)**	9.70	±2.48	12.5	77.64	-25.52
8	**Zinc Post (mg)**	10.95	±2.87	12.5	87.60	-12.2
9	**Selenium Pre (mcg)**	44.60	±7.90	40	111.50	13.16
10	**Selenium Post (mcg)**	50.85	±9.77	40	127.12	25.1
11	**Sodium Pre (mg)**	3690.81	±680.51	2000	184.54	56.16
12	**Sodium Post (mg)**	3986.78	±706.06	2000	199.34	63.6
13	**Potassium Pre (mg)**	1197.98	±220.29	3500	34.23	-236.22
14	**Potassium Post (mg)**	3403.02	±682.52	3500	97.23	-3.21

While pre-infection intake was adequate for iron, magnesium and selenium, but severely deficient for calcium, zinc, potassium, and marginally excessive for sodium. However, post infection values show marked improvement in calcium, magnesium, zinc, selenium and potassium, alongside a further increase in sodium intake. This also, suggests a spontaneous, adaptive shift toward immune-supportive and electrolyte-balancing minerals mirroring the adaptation of healthier diets and behaviors. The pre COVID iron intake (14.85 mg/day) exceeded the EAR (13 mg/day) by 14.2% (NAR = 114.22; t = 10.89), indicating baseline adequacy and likely sufficiency to support erythropoiesis and immune cell function. Post COVID, the intake declined to 13.12 mg/day (NAR = 100.90; t = 0.72), falling to marginal sufficiency. This reduction may reflect behavioral avoidance of iron rich foods due to altered taste/smell, gastrointestinal discomfort, potentially exacerbating functional iron deficiency.

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

While serum ferritin often rises during acute infection due to inflammation-driven hepcidin up-regulation, tissue iron availability remains low, impairing mitochondrial respiration and immune cell proliferation (Ganz, et al., 2015). The observed decline in dietary iron may thus contribute to persistent fatigue and impaired recovery, particularly in women and older adults. Calcium intake increased significantly from 526.6 mg/day (NAR = 65.83; t = -37.6) to 645.9 mg/day (NAR = 80.74; t = -22.77) which is still below the EAR (800 mg/day) but showing meaningful improvement. Similarly, magnesium intake rose from 326.4 mg/day (NAR = 110.65; t = 11.45) to 347.9 mg/day (NAR = 117.94; t = 16.07), consistent with its role as a cofactor for over 300 enzymatic reactions, including ATPase activity, DNA repair, and NLRP3 inflammation regulation (Chacko et al., 2021). Given that hypo-magnesemia is linked to increased IL-6 and TNF-α levels, this increase may again represent an adaptive response to mitigate post-viral inflammation. The intake of zinc improved from 9.70 mg/day (NAR = 77.64; t = -25.52) to 10.95 mg/day (NAR = 87.60; t = -12.2), approaching but still falling short of the EAR of 12.5 mg/day. Zinc is critical for antiviral immunity via inhibition of RNA polymerase, modulation of NF-κB (Nuclear Factor Kappa B) signaling, and maintenance of epithelial barrier integrity (Wessels et al., 2020). Its elevation may reflect conscious or subconscious dietary compensation to counteract immune dysfunction. Selenium intake showed even more robust improvement, rising from 44.6 µg/day (NAR = 111.50; t = 13.16) to 50.85 µg/day (NAR = 127.12; t = 25.1), well above the EAR of 40 µg/day. Selenium status has been directly linked to COVID-19 severity with low serum selenium correlating with higher mortality, while adequate intake supports T-reg (Regulatory T cells) differentiation and reduces pro-inflammatory cytokine production. Potassium intake surged dramatically from 1198 mg/day (NAR = 34.23; t = -236.22) to 3403 mg/day (NAR = 97.23; t = -3.21), nearly reaching the EAR of 3500 mg/day. This massive rise with the intake nearly tripling the pre COVID levels, strongly suggests a shift toward potassium-rich whole foods. Potassium is critical for maintaining membrane potential, regulating blood pressure, and modulating NLRP3 inflammasome activity (Munoz-Planillo et al., 2013). Its elevation may reflect conscious or subconscious dietary compensation to counteract endothelial dysfunction and autonomic dysregulation particularly common in Long COVID. Conversely, sodium intake increased further from 3691 mg/day (NAR = 184.54; t = 56.16) to 3987 mg/day (NAR = 199.34; t = 63.6), far exceeding the WHO-recommended limit of 2,000 mg/day (WHO, 2023).

Conclusion: This study comprehensively documents the profound and bidirectional interplay between COVID-19 and nutritional status in recovered individuals. Statistically significant shifts were observed across all macronutrients, vitamins and minerals, revealing a post-COVID dietary pattern characterized by reduced energy, protein, fat, and carbohydrate intake, yet increased consumption of fiber, antioxidant vitamins (A, C, E), and immune-modulatory minerals (selenium, magnesium, potassium). Notably, vitamin D and iron intake approached adequacy post-infection, while calcium, zinc and folate remained suboptimal despite improvement. Nutrient Adequacy Ratios (NAR) further exposed critical gaps with chronic under-consumption of fiber, calcium, potassium and folate persisting even after recovery, while sodium intake far exceeding the safety thresholds. These findings indicate a spontaneous but incomplete dietary recalibration where patients instinctively gravitate toward immune-supportive nutrients, yet fail to fully correct deficiencies known to impair mucosal immunity,

ISSN PRINT 2319 1775 Online 2320 7876
 Journal UGC CARE Listed (Group-I) Volume 14, Issue 05 July 2025

redox balance, and tissue repair. Thus, standardized post-COVID nutritional screening and personalized dietary counseling, prioritizing protein optimization, potassium- and fiber-rich whole foods and targeted supplementation for vitamin D, zinc and folate are not adjunctive but essential components of rehabilitation. Future public health strategies must integrate nutrition into post-pandemic recovery frameworks to mitigate long-term morbidity and enhance resilience against recurrent viral threats.

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