COVID

Article

Long-Haul COVID: Investigating the Effects Within the Mauritian Context

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Abstract: Background: COVID-19 infection can have a protracted course in many survivors, with varied sociodemographic and medical characteristics, exhibiting a plethora of symptoms that have consequential impacts on their quality of life. This study sought to gather pertinent data about the prevalence of Long-Haul COVID (LC), the predisposing factors to this condition and the burden on the quality of life of Mauritian survivors. **Research Setting:** A cross-sectional study was performed using an adapted online questionnaire, using two definitions of Long COVID, namely the WHO and NICE, SIGN and RCGP definitions. Associations between LC and categorical variables were employed to explore relationships between LC and ratio (FAS, FSS, PCS-12, MCS-12) variables. Simple and multivariable logistic regression models were used to assess the predictors and outcomes associated with LC. **Findings:** Of 285 Mauritians with a confirmed history of COVID-19 infection, 64.2% developed Long COVID (WHO LC-38.9%, NICE, SIGN and RCGP LC-55.8%). The most prevalent symptoms were fatigue or muscle weakness (88.0%), cough (57.4%), difficulty concentrating (55.2%), trouble remembering or memorising (49.7%), insomnia or sleep disturbance (43.7%), amongst others. Statistically significant associations were determined between LC and age, gender, vaccination status, severity of acute illness, reinfections, self-perception of disease and having more than five acute symptoms. Long COVID positively correlated with fatigue. Both Long COVID and severe fatigue ($F = 73.266$, $p < 0.001$) negatively impacted PCS-12. Fatigue had no significant impact on MCS-12. **Conclusions:** This study demonstrated the presence of Long COVID in the Mauritian population. Long COVID manifests as a complex and long-lasting affliction that affects even young adults with disabling outcomes, owing to multiple lingering symptoms but, most importantly, fatigue. The latter brings about distressing declines in physical and overall quality of life that thump both individual and societal health and productivity.

Keywords: Long COVID (LC); HRQOL; post-acute sequelae of SARS-CoV-2 infection (PASC); fatigue; Long COVID symptoms; post-acute COVID-19 syndrome (PACS)

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Academic Editor: Luigi Vimercati

Received: 18 November 2024
Revised: 27 December 2024
Accepted: 29 December 2024
Published: 2 January 2025

Citation: Soreefan, A.B.; Putteeraj, M.; Somanah, J. Long-Haul COVID: Investigating the Effects Within the Mauritian Context. *COVID* **2025**, *5*, 6. https://doi.org/10.3390/covid5010006

1. Introduction

The novel coronavirus, first detected in Wuhan, China, in 2019, led to rapidly expanding cases of pneumonia [1]. It soon became apparent that this infection caused by the SARS-CoV-2, termed COVID-19, has a more severe and protracted course than the 2003 outbreak of severe acute respiratory syndrome (SARS) and the Middle East Respiratory Syndrome (MERS) [1], and is associated with a myriad of short- and long-term complications [2]. The COVID pandemic brought forward an alarming rate of severe cases that progressed to acute respiratory distress syndrome (ARDS) and multiorgan failure [3].

With time, the survivors were found to experience incomplete recovery from COVID-19 or new unexplained symptoms, resulting in the emergence of the perplexing Long-Haul COVID [4]. The latter is detected in both hospitalised and non-hospitalised patients and often leads to poor quality of life with psychosocial impacts. However, this syndrome is often missed or correlated with other pathologies, given the lack of clear clinical definition and diagnostic criteria [5,6], further impeding its detection.

It is estimated that several million people are afflicted with this illness, ranging from 10 to 70% of COVID survivors worldwide [7]. Long-Haul COVID (or Long COVID) is known under several other acronyms. Likewise, the symptoms seen in Long COVID (LC) are multisystemic and differ from patient to patient [8].

1.1. Defining Long COVID

Different medical bodies have tried to define the term Long COVID, yet there is no full consensus on the definition [9]. NICE, SIGN and RCGP (2022) [10] have teamed up to paint a comprehensive picture of LC. Their definition, though being to the point, did not take into consideration the substantial number of patients reporting the appearance of new, unexplained symptoms following an acute phase of COVID-19 infection. On the other hand, WHO (2021) [11] included this aspect in its definition by having recourse to Delphi methodology. Accordingly, as defined by NICE, SIGN, and RCGP (2022) [10], LC involves the continuation of acute COVID-19 symptoms for a duration starting from 4 weeks and potentially extending beyond 12 weeks. WHO, on the other hand, defines Post COVID-19 condition as occurring in people "with a history of probable or confirmed SARS-CoV-2 infection", with symptoms manifesting 3 months after disease onset and persisting for 2 months or more, and that cannot be justified by any other diagnosis [12]. The symptoms can be a continuity of or occur as new onset following remission from the acute phase. They can also have a relapsing–remitting pattern [11].

1.2. Prevalence and Spectrum of Long COVID Symptoms

The prevalence of symptoms persisting beyond 28 days varies widely, ranging from 10% to 70% [7]. Research by Hossain et al. (2021) [13] demonstrated a prevalence of LC in 16.1% of the 2198 participants in Bangladesh, with symptoms persisting for an average duration of 21.8 $\pm$ 5.2 weeks. A UK-based study showed that 2.5% of the entire population had experienced at least one symptom of Long COVID [2], complementing a retrospective study, which identified that 57% out of 273,618 COVID survivors in the UK experienced at least one LC symptom lasting for 6 months, and 36.55% experienced such symptoms for a duration between 3 and 6 months [14]. A prospective study conducted on 4182 COVID-19 survivors in the UK provided the following insights into LC: 13.3% exhibited symptoms lasting $\geq$28 days, 4.5% experienced symptoms for $\geq$8 weeks, and 2.3% had symptoms for $\geq$12 weeks [15]. Muzyka et al. (2023) [16] established a prevalence rate of 23% for Long COVID within a Ukrainian cohort. Across continents, in Morocco, it was found that 47.4% of individuals exhibited the presence of at least one LC symptom [17]. Moreover, the symptoms of LC were found to be typically characterised by psychosocial aspects [18] and have multisystemic manifestations [19].

LC englobes a variety of symptoms, namely fatigue, chest pain, cough, hair loss, etc. The number of symptoms and the specific symptoms experienced in the first week of the disease are risk factors for Long COVID. Manifestation of more than five symptoms or presence of fatigue, headache, dyspnoea, hoarse voice or myalgia in the first week showed greater odds for Long COVID [15]. The findings by Ayegbusi et al. (2021) [20] showed similar relationships between symptom load, presence of dyspnoea and Long COVID incidence. The most frequently occurring ones are fatigue, dyspnoea and neuropsychiatric

symptoms in descending order. Muzika et al. (2023) [16] showed that fatigue was manifested in 90% of COVID long-haulers, followed by "muscular pain (85%), anosmia (70%), hair loss (70%), sleep disorders (70%), dyspnea (30%), and lastly brain fog (25%)" [16]. A meta-analysis revealed the predominance of persistent fatigue (one-third of cases) and cognitive impairment (one-fifth of cases) $\geq$ 12 weeks after COVID-19 onset [21]. Similarly, the prevailing incidences of asthenia, sleep disturbance and anxiety or depression were noted 6 months after the COVID-19 diagnosis [22]. Brain fog, headache, anosmia, ageusia, anxiety, depression [23] and insomnia [8] form part of the neuropsychiatric afflictions.

Fatigue appears to be a key manifestation of LC and has been extensively researched. Most studies ascertained its predominance in PASC and described it as having physical and cognitive influences [24]. Interestingly, one study revealed a mean score of 21.2 on the fatigue assessment scale (*FAS*) [25]. Chronic fatigue syndrome (CFS) is characterised by fatigue on exertion, disturbed sleep, cognitive dysfunction and chronic pain, and has been linked to viral infections. According to NICE, the prevalence of the aforementioned symptoms for more than 4 months following COVID-19 infection would fit the diagnosis of CFS. Thus, it can be extrapolated that CFS is yet another incapacitating affliction experienced by COVID survivors. Similar to CFS, the risk of developing LC is dependent on demographic factors, including gender and age [26].

Interestingly, El Otmani et al. (2022) [17] established that even the asymptomatic patients exhibited Long COVID symptoms, namely asthenia, myalgia and anorexia, after 4 weeks of infection.

1.3. Associating Demographics, Comorbidities and Vaccination Status with Long-Haul COVID

Female preponderance has been established in many studies [20,27]. In comparison to their male counterparts (9.5%), women below 70 years of age are more prone (14.9%) to develop LC [15]. Besides displaying predominance (72%) in the female gender, PASC afflicted them extensively with fatigue (43.3%), exceeding the fatigue assessment score in the male gender [25]. Women are also at higher risk of exhibiting dyspnoea [28] and brain fog [29]. Ganesh et al. (2021) [7] similarly denoted the dominance of persisting COVID symptoms in women. Age-wise, the prevalence of LC increases from 9.9% (18–19 years) to 21.9% ($\geq$70 years) [15], complementing findings from Jones et al. (2021) [27] who showed that those above 40 years display higher odds of developing LC.

Socioeconomic status has certain implications as far as PASC is concerned [30]. In a recent study, it was found that individuals with low SES were 50% more likely to suffer from LC [31], including a high likelihood of experiencing fatigue [24]. Those in rural areas were predicted to be more prone to developing LC [13]. Lower income was shown to be a predictor of the severity of LC afflictions [32]. Contrastingly, using the Index of Multiple Deprivation (IMD) demonstrated that LC occurs irrespective of SES [15]. Similar findings were revealed in the UCLA COVID Ambulatory Program [33], where the Social Vulnerability Index was employed to qualify for SES.

Pre-existing health conditions are predisposing factors for developing Long COVID [8] and were linked with the incidence of post-COVID fatigue and dyspnoea [28]. Bronchial asthma strongly influenced Long COVID prevalence [20], with an odds ratio (OR) of 2.14 (95% CI 1.55–2.96) [15]. Besides asthma, diabetes [34], obesity, hypersensitivities, and obstructive lung pathologies also similarly influenced Long COVID incidence [35].

There is increasing evidence that COVID-19 vaccination has protective effects against acute and post-acute illness alike [36]. Unvaccinated versus vaccinated individuals were more prone to COVID sequelae [30]. In an Israeli cohort, 64% of fully vaccinated patients were found to be less likely to develop LC fatigue in comparison to their unvaccinated counterparts [2]. Double-vaccinated individuals were less likely to experience LC than

unvaccinated ones, 9.5% vs. 14.6% [37], while successive doses and boosters attenuated the risks of LC prevalence from 41.8% in unvaccinated participants against 30.0% in a single dose to 16.0% with triple doses [35].

1.4. Quality of Life and Long COVID: Consequential Effects

The impacts of PASC on quality of life (QOL) have been broadly researched. Disruption of social, professional and, subsequently, financial aspects of life have resulted from PASC. Using the PROMIS® scales, dysfunction was established in COVID survivors, with a significant 43.2% affection of social roles, followed by disabling effects of pain (17.8%) and fatigue (16.2%) [7]. Persistence of such incapacitating symptoms has occasioned protracted recovery, resulting in delays in resuming work and studies, even in young individuals without comorbidities [38].

Prevailing PASC symptoms, mainly dyspnoea and fatigue, impair the aptitude to perform physical exercises and activities of daily living (ADLs) and the ability to resume work. These contribute to the deterioration of QOL [39]. A distressing 45% of individuals reported impairments with ADLs [28], ensuing negative outcomes on work and daily functioning [18]. Sandler et al. (2021) [4] showed the extent of PASC-related disability's influence on the inability to get back to work: 40%, 31%, and 9–15% at 2, 3, and 4 months post-disease onset, respectively. Similarly, in a cohort of hospitalised COVID survivors with established LC, 18% out of the 68% full-time workers could not resume work, 19% had to change their work routine owing to the disabling symptoms, 46% experienced decreased physical faculties, and 20% suffered a novel disability [40].

Ziauddeen et al. (2022) [32] noted that 64.4% of COVID survivors displayed an inability to execute their usual tasks, with 37.0% cases of loss of revenue, affecting a large number of individuals. According to Vaes et al. (2021) [41], 83% of participants in their research "*reported moderate-to-poor self-reported health*", and 49% suffered from "*moderate-to-severe functional limitations*". Besides physical and employment issues, psychological and mental health consequences were equally observed [42].

LC fatigue initiates frustration [24], emotional disequilibrium as well as psychological and mental disorders [20]. Dyspnoea mediated by LC also triggers similar responses along with the surge of new "*psycho-neurological symptoms*" that can last up to one-year post-infection [42]. There is evidence of about 25% of cases of depression, 12% of post-traumatic stress disorder (PTSD) and 17% of cognitive dysfunction in COVID survivors [40]. Cognitive impairments trigger deteriorations in the QOL of many survivors and are a threat to the autonomy, health and independence of individuals [43]. Mainous et al. (2021) [44] demonstrated the presence of mortality risk within the framework of post-acute sequelae of SARS-CoV-2 infection. Notably, LC has been connected to escalated risks of both near-term and persistent cardiovascular disease (CVD) as well as heightened mortality rates [45]. The CDC (2022) [46] reported over 3500 direct LC-related deaths, with the highest rates observed among individuals aged 64 and above and a higher prevalence in males (51.5%).

Locally, there is a critical need to fully grasp the prevalence, underlying factors, symptomatology, and immense burden of LC. This cross-sectional study endeavours to evaluate the presence and profound impacts of LC within the Mauritian population and examine the intricate relationship between LC and a wide array of crucial factors, including sociodemographic variables, disease severity, vaccination status and baseline comorbidities. By incorporating both the WHO (Delphi consensus) and NICE, SIGN and RCGP case definitions of Long-Haul COVID, this research aims to shed light on the relentless repercussions of this lingering condition.

2. Materials and Methods

2.1. Study Setting and Participant Characteristics

A cross-sectional study using a self-reporting questionnaire was undertaken. Random stratified sampling was used to gather responses from the strata of interest. Recruitment was carried out across the community through voluntary participation using an online questionnaire set on Google Forms and disseminated using mediums such as WhatsApp and email. Inclusivity was taken into consideration by adopting an assisted approach towards groups who are less likely to participate in the study, including the elderly and those with low education levels, low income, who are unemployed and who do not have access to a computer or internet. The sample size was calculated to be 384 with a margin error of 5%. The population earmarked was based on the number of infected people across the Mauritian population. The number of infected people in the Mauritian population as of 4 November 2022, i.e., 40,718 [47].

Inclusion Criteria

i. Mauritian citizens, irrespective of socioeconomic background.
ii. History of COVID-19 infection at least one month prior to participating in this study.
iii. Aged 18 years or more.

Exclusion Criteria

Those who contracted COVID-19 less than one month prior to participating in this study and aged under 18 years.

2.2. Research Instrument and Design

The questionnaire was designed using inputs stemming from a comprehensive literature search with similar objectives and pre-set scales to establish the prevalence of LC and items relevant to symptomatic examinations. The questionnaire was divided into five parts and consisted of 40 questions (Table 1). The items included in this instrument were mainly in the form of dichotomous, Likert and multi-responses to ensure adaptability of each item to the nature of the query.

Table 1. Instrument structure and variables.

Section	Variables
Demographic	Age, gender, marital status, education level, familial entourage occupation, income Loss of or change in job, financial dependence of family members on participant
COVID-19 History	Details of the infection: frequency, dates, symptoms and treatment, severity of the disease hospitalisation history; perception of the acute COVID illness; persistence of symptoms; appearance of new, unexplained symptoms
Medico-Social History	Pre-COVID comorbidities and treatment; COVID-19 vaccination details
Fatigue Scales	The Fatigue Assessment Scale (FAS Score); The Fatigue Severity Scoring (FSS Score)
Quality Of Life	The Short Form 12 (*SF12*) scale—12 questions that qualify and quantify physical and mental impacts on QOL: PCS and MCS scores. PCS 12 score less than 50 is suggestive of declining/poor physical health whereas MCS 12 lower than 42 is indicative of mental health decline and depression [48,49].

2.3. Instrument Reliability and Validity

Prior to analysis, data were tested for reliability, construct validity and sample adequacy in SPSS, even though a sample size of 285 may be considered statistically large. Construct validity testing was conducted to verify whether "research constructs were unidimensional" [50], while the aim of reliability was to test the measuring instruments for internal consistency [51].

2.4. Data Analysis

All data collected were extracted to Microsoft Excel. The Statistical Package for Social Sciences (SPSS) software (V21-IBM Corp., Armonk, NY, USA) was used for data analysis. Descriptive statistics was applied to determine the prevalence of LC and the predominant symptoms reported in terms of frequency and/or percentage. Inferential analysis was carried out to find out whether WHO LC and NICE/SIGN/RCPG LC were associated with respondents' demographic characteristics (age, gender), socioeconomic status (education, income, occupation) and health-related variables (self-perception of illness, severity of COVID-19, symptoms in the first week, history of diseases, vaccination status, type of vaccine, fourth dose status). The following analyses were used as a guiding thread.

Dependent Variables:

1- *WHO Long COVID*
2- *NICE/SIGN/RCGP Long COVID*

Independent Variables:

1. Age
2. Gender
3. Socioeconomic status: occupation, income and education level
4. Job status (job loss/job change)
5. Financial dependence (family members dependent financially on participant or not)
6. COVID-19 vaccination status (non-vaccinated/partially vaccinated/fully vaccinated)
7. Fourth dose of vaccine
8. Severity of the COVID infection (non-severe * vs. severe/critical **)
9. Symptomatology (number of symptoms in the first week of disease)
10. Pre-existing health conditions

* Non-severe: not hospitalised or hospitalised because of protocol; ** Severe/Critical: hospitalised because severely ill +/− ICU admission +/− Oxygen/artificial ventilation (due to lack of data such as oxygen saturation and pulmonary vitals, severe and critical have been combined as one variable).

Correlations between categorical variables (*WHO Long COVID* and NICE/SIGN/RCPG Long COVID) and ratio variables (*FAS*, *FSS*, *PCS-12* and *MCS-12*) were determined by Spearman's Rho, whereas those between two ratio variables were obtained by Pearson's product–moment correlation. Multiple regression analysis was conducted to determine the impacts of *WHO Long COVID* and *NICE/SIGN/RCGP Long COVID* on (i) Fatigue Assessment Score (*FAS*), (ii) Fatigue Severity Score (*FSS*), (iii) the Physical Component Summary (*PCS-12*) of the Short Form (*SF-12*) Health Survey and (iv) the Mental Component Summary (*MCS-12*) of the Short Form (*SF-12*) Health Survey.

2.5. Ethical Consideration

Ethical clearance with the following approval reference, MHC/CT/NETH/2022/AV5 (10 January 2023), was obtained from the Ethics Committee of the Ministry of Health and Wellness of Mauritius prior to proceeding with the research work. Anonymity of

respondents has been maintained throughout, and all data has been treated with utmost confidentiality. Participants were allowed to withdraw from the study at any given point and were not under any obligation to enrol in the study. Agreeing to participate in the study was considered as a formal consent to collect the data through the instrument administered.

3. Results

3.1. Demographics and Anamnesis Vitae

A total of 285 individuals were included in this study. The majority were in the age group 18–39 years (63.9%), female (61.4%), employed (56.5%) and from urban areas (60.0%) (Supplementary Data S1). From the sample, 64.9% of the participants did not have any history of disease. The most prevalent comorbidities were non-communicable diseases, accounting for 37.3% of total respondents, with a predominance of hypertension and diabetes mellitus, 31% and 25% (of the participants with medical pathologies), respectively (Table 2). Out of the respondents, only 10 had not been vaccinated. The majority of respondents had received their first vaccine doses before contracting COVID-19, while a small portion (n = 31) received their initial vaccine dose after being infected with the virus.

Table 2. Comorbidity profile of participants.

Disease	Frequency	Percentage
Cardiovascular diseases	12	4.2%
Diabetes mellitus	25	8.8%
Hypertension	31	10.9%
Renal impairment or failure	2	0.7%
Stroke	5	1.8%
Dyslipidaemia	15	5.3%
Rheumatoid arthritis	8	2.8%
SLE	2	0.7%
Bronchial asthma	22	7.7%
Lung problems	3	1.1%
Obesity	16	5.6%
Psoriasis	11	3.9%
Thyroid gland pathologies	6	2.1%
Vitamin D deficiency	21	7.4%
Total participants with comorbidities	100	35.1%
No history of diseases	185	64.9%

3.2. Anamnesis Morbi: COVID-19 Infection

Among the respondents, 72.6% reported being infected with COVID-19 once, while 20.4% experienced two infections. A smaller proportion, 4.9%, had been infected three times, and only 2.1% had more than three infections. In terms of the timing of their most recent infection, 2.8% had been infected up to four weeks ago, 5.6% between 4 and 12 weeks ago, 20.0% between 3 and 6 months ago, 34.7% between 7 and 12 months ago, and the majority, 36.8%, had been infected more than 12 months ago. Regarding diagnosis, 44.2% used rapid antigen tests (RAT) at home, while 30.5% were tested by RAT at medical institutions. A smaller percentage, 6.3%, were diagnosed by RT-PCR test only, while for 18.9%, the diagnosis was confirmed by a combination of both RAT and RT-PCR. When

describing their COVID-19 infection, 4.2% reported having no symptoms, 29.8% likened it to the common cold, 39.6% considered it worse than the flu but not the worst infection they had encountered, 21.8% regarded it as the worst infection in their life, and 4.6% feared they would die from COVID-19.

Given the lack of clinical data, severity was classified as non-severe (not hospitalised or hospitalised because of the sanitary protocol) and severe/critical (hospitalised because severely ill +/− ICU admission +/− oxygen/artificial ventilation). The distribution of participants according to severity was as follows: severe/critical 7 (2.5%) and non-severe 278 (97.5%).

The majority of respondents (45.8%) experienced COVID-19 symptoms for 3–5 days, followed by 33.4% with symptoms lasting 6–10 days. A smaller proportion reported symptoms persisting for 11–15 days (11.2%), 16 days to 1 month (6.0%), and a minority endured symptoms for over 1 month (3.6%). The most prevalent symptoms were sore throat, fever or chills, weakness or fatigue and muscle aches or body aches, followed by cough (Supplementary Data S2). Whilst five respondents (1.8%) were asymptomatic, the number of symptoms ranged from 1 to 12, with a mean of 5.30 ± 2.54. The symptoms were categorised into three groups: no symptoms (n = 5, 1.8%), 1–5 symptoms (n = 150, 52.6%) and >5 symptoms (n = 130, 45.6%).

3.3. Long Haul COVID: Prevalence and Symptomatology

Both definitions, WHO and NICE/SIGN/RCGP, were used to determine the presence of LC. While 35.8% of respondents did not exhibit LC, a staggering 64.2% of the sampled participants exhibited LC symptoms. However, the long-haulers proportion was higher under the NICE, SIGN and RCGP definitions in comparison with the WHO one (Table 3). It is also worth noting that 30.5% of participants related to both definitions of LC.

Table 3. Prevalence of Long COVID.

Descriptors	Frequency	Percentage
WHO Long COVID	111	38.9%
NICE, SIGN and RCGP Long COVID	159	55.8%
Long COVID characterised according to both definitions	87	30.5%
Overall Long COVID Prevalence	183	64.2%
No Long COVID	102	35.8%

Data presented as percentage of total number of respondents.

The data showed that a significant proportion of participants experienced fatigue or muscle weakness (88.0%), followed by cough (57.4%). Neuropsychiatric symptoms were also substantially present: difficulty to concentrate (55.2%), trouble to remember or memorise (49.7%), insomnia or sleep disturbance (43.7%), headache (41.0%), anxiety or depression (26.8%) and chemosensory impairments such as loss of taste (27.9%) and loss of smell (21.9%). Other frequently reported symptoms included shortness of breath (31.1%), sore throat (30.1%) and hair loss (21.3%). Chest pain, fever, diarrhoea and other symptoms (skin rashes, body aches, chills, restlessness) were also experienced in a minority of cases. The symptoms had varied appearance timelines and durations. All the symptoms, except the non-specific ones (others), were experienced by the majority during the first 3 months following COVID-19 infection. On average, the manifestations of LC symptoms decreased with time from the acute phase (Table 4).

Table 4. Timelines of Long COVID symptoms manifestation.

	4–12 Weeks After COVID	4–6 Months After COVID	7–12 Months After COVID	>12 Months After COVID
Symptoms	**Frequency *n*, (%)**			
Cough	84 (80.0)	10 (9.5)	7 (6.7)	4 (3.8)
Shortness of breath	28 (49.1)	19 (33.3)	6 (10.5)	4 (7.0)
Sore throat	38 (69.1)	11 (20.0)	6 (10.9)	0 (0.0)
Fever	25 (80.6)	3 (9.7)	0 (0.0)	3 (9.7)
Fatigue or muscle weakness	82 (50.9)	31 (19.3)	31 (19.3)	17 (10.6)
Loss of taste	38 (74.5)	7 (13.7)	6 (11.8)	0 (0.0)
Loss of smell	32 (80)	4 (10)	3 (7.5)	1 (2.5)
Headache	49 (65.3)	14 (18.7)	9 (12.0)	3 (4.0)
Insomnia or sleep disturbance	33 (41.25)	19 (23.75)	17 (21.25)	11 (13.75)
Difficulty to concentrate	45 (44.6)	24 (23.8)	16 (15.8)	16 (15.8)
Trouble to remember or memorise	33 (36.26)	19 (20.88)	24 (26.37)	15 (16.48)
Difficulty to understand information	20 (45.5)	11 (25.0)	6 (13.6)	7 (15.9)
Anxiety or depression	18 (36.73)	11 (22.45)	9 (18.37)	11 (22.45)
Diarrhoea	10 (58.82)	3 (17.65)	3 (17.65)	1 (5.88)
Chest pain	12 (42.9)	5 (17.9)	5 (17.9)	6 (21.4)
Hair loss	13 (33.3)	7 (17.9)	10 (25.6)	9 (23.1)
Others	8 (26.7)	11 (36.7)	5 (16.7)	6 (20.0)

Data presented as percentage of total number of respondents.

3.4. Examining Fatigue and Participant's Quality of Life

The *FAS* score had a mean of 26.43 ± 8.131. Only 28% of participants had normal levels of fatigue. The *FSS* total score had a mean of 34.59 ± 15.73, and the *FSS* mean score had a mean of 3.85 ± 1.74. Based on the *FSS* scale, 46.0% of respondents experienced severe levels of fatigue (Supplementary Data S3).

The *SF-12* scale was administered to determine any decline in the physical and mental dimension of the participants' quality of life. A larger portion of respondents exhibited poor QOL, with the physical component (68.4%) being more affected (Table 5). Whilst the mean for *MCS 12* was higher than the standard mean of 42, that of *PCS 12* was lower than its standard mean of 50. This implies a more important physical deterioration in the QOL of the participants. More females versus males (*PCS 12*: 70.9% vs. 64.2% and *MCS 12:* 48.6% vs. 36.7%) displayed scores equivalent to poor QoL.

Table 5. Gender-based QoL scores.

PCS 12	***Frequency n, (%)***		
	Total	***Male***	***Female***
Poor Physical Quality of Life	195 (68.4)	71 (64.5)	124 (70.9)
Good Physical Quality of Life	90 (31.6)	39 (35.5)	51 (29.1)
Mean		45.42	
MCS 12	***Frequency, n (%)***		
	Total	***Male***	***Female***
Poor Mental Quality of Life	125 (43.9)	41 (37.3)	85 (48.6)
Good Physical Quality of Life	160 (56.1)	69 (62.7)	90 (51.4)
Mean		42.96	

3.5. Examining the Relationship Between Sociodemographic Factors, COVID-19 Manifestations and Perception of COVID-19 Versus Long COVID

Polarising differences were identified with respect to the self-perception of their illness due to COVID-19. With respect to WHO Long COVID, 39.1% of respondents stated that it was like the common flu and showed no further signs of COVID-19, whereas 36% of those who said it was the worst infection they ever experienced, displayed Long COVID symptoms as per the association between perceived severity of illness and LC experience [χ^2 (4) = 44.124, $p < 0.01$]. Identical patterns in the percentages for NICE/SIGN/RCPG Long COVID were noted, whereby 42.9% of respondents who referred to symptoms of common flu showed no further manifestations, whereas 28.3% acknowledging the seriousness of their infection experienced persisting symptoms [χ^2 (4) = 36.139, $p < 0.01$].

Age group was not significantly associated with the LC based on both definitions, i.e., WHO LC or NICE/SIGN/RCPG LC. However, gender showed significant associations with both WHO LC [χ^2 (1) = 4.868, $p < 0.05$] and NICE/SIGN/RCPG LC [χ^2 (1) = 9.183, $p < 0.01$]. For both LC variables, it was observed that females were significantly more affected than males (69.4% for WHO and 69.2% for NICE/SIGN/RCGP).

With regards to SES variables, there was no association between both LC measures and income [WHO: χ^2 (3) = 2.026, $p > 0.05$–NICE, SIGN and RCGP: χ^2 (3) = 0.071, $p > 0.05$], occupation [WHO: χ^2 (5) = 1.492, $p > 0.05$–NICE, SIGN and RCGP: χ^2 (5) = 4.496, $p > 0.05$] or lost job status [WHO: χ^2 (1) = 2.924, $p > 0.05$–NICE, SIGN and RCGP: χ^2 (1) = 0.183, $p > 0.05$]. Nonetheless, WHO LC was significantly associated with education [χ^2 (1) = 12.166, $p < 0.05$] and changed job status [χ^2 (1) = 6.322, $p < 0.05$]. A total of 52.3% of respondents who studied up to the secondary level and 97.1% of those who did not change jobs did not show any signs or symptoms of LC. In contrast, no association was found between the two LC variables and history of diseases, type of vaccine and the fourth dose status, the latter of which was encouraged in the local setting.

Interestingly, the present data suggests a statistically significant model between WHO LC and the frequency of COVID-19 infections [χ^2 (3) = 16.090, $p < 0.01$] with the number of infections accounting for 23.8% variability of WHO LC. The data also indicates that a higher percentage of participants with WHO LC have experienced multiple COVID-19 infections (29.7% infected twice, 7.2% infected three times, and 3.6% infected more than three times) compared to those without WHO LC (14.4% infected twice, 3.4% infected three times, and 1.1% infected more than three times).

Within those lines, a strong significant association was noted between the number of symptoms in the first week of COVID-19 infection and the occurrence of both WHO LC [χ^2 (2) = 21.665, $p < 0.001$] and NICE/SIGN/RCPG LC [χ^2 (2) = 27.835, $p < 0.001$]. Participants exhibiting up to five symptoms were less likely to develop LC based on their reports of LC characteristics according to WHO (62.1%) and NICE/SIGN/RCPG (66.7%). On the other hand, the trend was reversed when the number of symptoms exceeded five in the first week of acute COVID infection: 62.2% and 58.5% exhibited signs of *WHO Long COVID* and *NICE/SIGN/RCPG Long COVID*, respectively. Additionally, respondents who did not develop *WHO Long COVID* did not experience severe COVID-19 infection [χ^2 (1) = 11.249, $p < 0.01$], while those who showed symptoms of COVID-19 in the first week displayed signs of *NICE/SIGN/RCPG Long COVID* [χ^2 (1) = 6.422, $p < 0.05$]. Interestingly, 81.7% of respondents who were fully vaccinated showed no signs of *NICE/SIGN/RCPG Long COVID* [χ^2 (2) = 8.174, $p < 0.05$].

3.6. Predicting the Effects of Long COVID on Fatigue and Quality of Life

The first regression model exploring the impact on *FAS*, showed that both *WHO Long COVID* (β = 0.382) and *NICE/SIGN/RCGP Long COVID* (β = 0.160) had significant positive

effects on fatigue, with the former exerting a stronger impact, hence, indicating higher levels of fatigue on the *FAS* scale. The model (χ^2 (2) = 38.82, $p < 0.001$) explained 21.6% of the variability in *FAS* scores, highlighting the importance of Long COVID as a contributing factor to fatigue.

Similar patterns emerged in a second model examining the impact on *FSS*. *WHO Long COVID* and *NICE/SIGN/RCGP Long COVID* had significant positive effects on *FSS* scores (χ^2 (2) = 24.89, $p < 0.001$), with WHO Long COVID exerting a stronger impact on fatigue levels ($\beta = 0.318$). The results of the multiple regression analyses reveal a strong propensity to fatigue in Long COVID, as measured by the *FAS* and *FSS* scales.

The simple regression model demonstrated that *FSS* had a significant negative impact on *PCS-12*, explaining 20.6% of the variability in *PCS-12* (F (1282) = 73.27, $p < 0.001$). To sum up, both *WHO Long COVID* and *NICE/SIGN/RCGP Long COVID* had significant impacts on *FAS*, *FSS* and *PCS-12* at the 5% level. However, they did not significantly affect *MCS-12*. Similarly, fatigue severity significantly impacted on *PCS-12* but not on *MCS-12*.

4. Discussion

4.1. Prevalence of Long COVID

Differing terms and definitions from organisations like WHO, NICE and CDC, ranging from broad to stringent, have created variability in how Long COVID symptoms are identified and characterised, leading to heterogeneity in Long COVID phenotyping [52]. In order to maximise homogeneity, this study utilised both the WHO and NICE/SIGN/RCGP definitions to determine the presence of LC in the cohort. A higher proportion of individuals met the criteria for LC under the NICE/SIGN/RCGP definition compared to the WHO definition (55.8% versus 38.9%, respectively), while 30.5% of the sampled participants demonstrated Long-Haul COVID symptoms that overlapped and could be characterised by both definitions. Studies have mostly used only one definition of Long COVID, besides the research by Talhari et al. (2023) [53], which englobed both continuation of acute COVID symptoms and new onset, unexplained symptoms and revealed consequential LC presence (83.2%). Limiting the definition to only persisting acute symptoms could lead to the exclusion of LC cases and left without appropriate care. Therefore, unless there is a unification of the definition and characteristics, the LC phenotypes will keep growing, thereby creating further confusion among concerned stakeholders and impeding the development of effective interventions [52].

A total of 64.2% of the participants in the current study experienced LC symptoms, while 35.8% did not exhibit such symptoms. Comparatively, across the globe, it is evident that the burden of this condition is substantial. Studies by Carfi et al. (2020) [54] and Talhari et al. (2023) [53] revealed soaring LC rates of at least one symptom up to 12 weeks after acute infection, as reflected by 87.4% and 83.2% participants in an Italian and Brazilian cohort, respectively. Likewise, LC symptoms were detected in 76% of patients in a Wuhan hospital [22]. Other research demonstrated varying yet lower rates of LC prevalence, and some had analogous results to this academic work. Studies conducted in the UK have reported a prevalence ranging from 2.5% to 57% of individuals [2,14], while in Bangladesh, the prevalence was found to be 16.1% in a cohort of 2198 participants [13]. Moreover, other countries such as Morocco and Ukraine have reported prevalence rates of 47.4% and 23% in their communities, respectively [16,17]. These data point towards differing characteristics in different populations, as suggested by Michelen et al. (2021) [55], in the likes of generation variations as proposed by Proal and VanElzakker et al. (2021) [56].

4.2. Spectrum of Long COVID Symptoms

The findings from this Mauritian cohort highlight a diverse range of symptoms associated with Long-Haul COVID, with fatigue or muscle weakness being the most prevalent symptom. This aligns with research by Muzyka et al. (2023) [16] and Sudre et al. (2021) [15], which found fatigue to be manifested in 90% and 97.7% of COVID long-haulers, respectively. Likewise, it was shown that fatigue could persist up to 12 months post-COVID in 60% of cases [57]. Comparing the findings from this Mauritian sample with the existing literature, fatigue consistently emerges as a major symptom in both datasets, indicating its significance as a hallmark of Long-Haul COVID. Neuropsychiatric symptoms, such as cognitive difficulties, sleep disturbances, and emotional challenges, are also prevalent in both datasets, highlighting their importance in understanding the impact of Long-Haul COVID on patients' well-being.

The experience of fatigue in Long COVID is underpinned by a range of interrelated mechanisms. Davis et al. (2023) [58] showed that reactivation of Epstein–Barr virus (EBV) contributes to this persistent fatigue, in addition to a disrupted fatty acid metabolism coupled with dysfunctional mitochondrion-dependent lipid catabolism that is suggestive of mitochondrial dysfunction and exercise intolerance. This intricate web of factors is exacerbated by COVID-19-induced inflammation, which may impair GABAergic signalling, potentially acting as a foundation for the prevailing fatigue [59]. Additionally, the relentlessly elevated interferon levels, demonstrated by Phetsouphanh et al. (2022) [60], up to 8 months after the acute phase of infection, suggest sustained fatigue in long-haulers. Since both EBV reactivation and GABAergic impairments impact neurocognitive function [58,59], the interplay between the latter and fatigue are plausible. In fact, the important predictors of fatigue are "*neurocognitive and psychiatric symptoms*" [61].

Participants in this study reported multiple cognitive, psychological and sleep irregularities, which either occurred individually or concomitantly. These symptoms are consistent with existing findings where neuropsychiatric afflictions, such as brain fog, cognitive impairment, anxiety, and depression, have been documented in Long-Haul COVID patients from different countries [58,62]. Neurocognitive and psychiatric manifestations have been attributed to a plethora of mechanistic actions such as neuroinflammation, decreased haemoglobin, micro-coagulopathy, impaired cerebral blood flow, metabolic disorders, and dysfunctional neurological signalling among others [56,58,63–66]. Given the high levels of fatigue in this sample, the prevalence of neuropsychiatric and cognitive impairments is relatable. Nevertheless, the presence of baseline psychiatric, neurocognitive, sleep disorders and stress were not ascertained in the sample, which could also account for the symptoms.

The current study also isolated LC-associated chemosensory impairments, such as loss of taste (27.9%) and smell (21.9%). These symptoms have been reported in other studies as well, with varying prevalence rates depending on the geographical location and the timing of data collection. Sudre et al. (2021) [15], in their multi-country study, found that loss of smell was reported by 72% of participants, while El Otmani et al. (2022) [17] reported a prevalence of 9.6% for anosmia/hyposmia. Two theories were posited for smell dysfunction by Zeng et al. (2021) [67], namely, the obstruction of olfactory receptors due to nasal congestion and the swelling of the olfactory clefts without any congestion. Given the presence of "runny nose or nasal congestion" in more than half of the participants (53%), the first theory seems more applicable in the Mauritian context. As for taste changes, they were attributed to cytokines, which have the capability to disrupt the usual taste transduction process and interfere with the normal turnover of cells within taste buds [68].

Additionally, cough was reported by 57.4% of the Mauritian participants, differing from studies in Spain and Bangladesh by Fernández-de-las-Peñas et al. (2021) [38] and Hos-

sain et al. (2021) [13], respectively, where cough prevalence ranged from 2% to 8.7% after 4–12 weeks post-COVID onset. This implies greater lung inflammation and fibrosis [69] and baseline allergic bronchitis [70] associated with LC cough as a potential mechanism in Mauritians. Similarly, chest pain was observed among the LC symptoms in this sample, which could potentially indicate an onset or exacerbation of pre-existing cardiac conditions. Research demonstrated elevated troponin levels, a 32% incidence of cardiac damage and cardiac inflammation 3 months after acute COVID-19 [71]. In fact, prolonged cardiac inflammation in 78% and 60% of recovered patients was found, irrespective of initial severity or time since diagnosis [72]. Cardiac impairment was revealed in 20% of patients at 6 months, persisting in over 50% of patients at 12 months via cardiac magnetic resonance [73]. The present findings advocate for cardiac investigations in patients with Long COVID reporting chest pain.

4.3. Onset and Chronicity of LC

The present study revealed that most symptoms were experienced during the first three months following COVID-19 infection, and over time, the manifestations of LC symptoms decreased from the acute phase. Symptoms like cough, shortness of breath, sore throat, fever, fatigue or muscle weakness, loss of taste, and loss of smell were more prevalent within the first three months post-infection, with only cough and shortness of breath showing some amelioration as the timeline extended to 4–6 months after COVID-19, and certain symptomatic features exhibited varying frequencies over the timeline of 4–12 months post-infection. Similarly, in most studies, at least one symptom persisted for 3 months or beyond [13,15]. This research also demonstrated that some symptoms, such as difficulty in remembering or memorising and difficulty understanding information, persisted at relatively high frequencies even beyond 12 months post-infection. A comparable scenario was uncovered in Wuhan, China, where symptoms of LC persisted for more than 6 months in 76% of patients [22]. In yet another study, persisting symptoms beyond 6 months and beyond 1 year of acute COVID-19 were reported at 8.9% and 56.1%, respectively [74].

The present data portray the depth of chronicity of Long COVID symptoms in numerous survivors and may be explained by three theories, namely, hypercoagulability and micro-clots, lingering viral RNA in different organs and an immune system in overdrive [58,63]. Similarly, Phetsouphanh et al. (2022) [60] established the persistence of alarming levels of interferon 8 months after the acute phase of infection, a study which also underscored the specificity of this immune marker in Long COVID patients, implying the long-lasting inflammatory effects of SARS-CoV-2. Other studies demonstrated that 60% of Long COVID patients had circulating SARS-CoV-2 spike antigen up to 12 months post-diagnosis, suggestive of a potential active virus reservoir or its components, which may contribute to persistent symptoms [66]. Thus, the appearance, persistence and disappearance of the different symptoms could be related to genetics, the duration the virus lingers in the different organs and the individual immune responses.

4.4. Relationship Between Demographics and Long-Haul COVID

The cross-sectional analysis reveals significant associations of gender with both WHO Long COVID and NICE/SIGN/RCPG Long COVID. The data suggests that females are considerably more affected than males, with 69.4% experiencing WHO LC and 69.2% experiencing NICE/SIGN/RCPG LC. This is consistent with earlier studies like Jones et al. (2021) [27] and Aiyegbusi et al. (2021) [20], purporting a female preponderance in Long-Haul COVID. Similarly, the meta-analytical findings by Tsampasian et al. (2023) [75] revealed the predominance of female gender association with LC manifestation. Several

theories have been proposed for this disparity, including sex-based variations in antibody production, with women showing a lower likelihood of seroconversion, a higher likelihood of seroreversion, and overall reduced antibody levels. These differences even impact "*antibody waning after vaccination*" [58]. Furthermore, X chromosomes carry multiple immune-coding genes, implying stronger expression of immune responses [76]. However, women have stronger IgG production during early disease phases, which could lead to more positive acute outcomes while enabling chronic manifestations [77]. This strong immune and inflammatory response is influenced by both genetic and hormonal factors being more pronounced in the female gender [68,78]. Similarly, Petrella et al. (2022) [79] demonstrated elevated levels of the cytokine TGF-β, a biomarker for oxidative stress and inflammation, in 19-year-old girls with LC. Given that this study had a majority of young participants, it could explain the predominance of LC amongst women. Another inflammatory biomarker, the ANG-1 angiogenic biomarker, associated with the female gender, has been isolated in LC patients [80]. The intense immune response in women, despite being protective in acute infection, tends to increase their vulnerability to protracted courses of autoimmune pathologies [81,82]. Furthermore, it has been hypothesised that LC can be mediated by oestrogen, supporting its higher prevalence in the younger female population [78].

The findings from this study indicate that there is a decreasing trend in the prevalence of Long COVID with increasing age, as seen in both definitions, WHO Long COVID and NICE/SIGN/RCPG Long COVID; however, no statistical significance was achieved in associating age and Long COVID. Contrastingly, Sudre et al. (2021) [15] reported an increasing prevalence of Long COVID with age, with prevalence rates ranging from 9.9% in the age group of 18–19 years to 21.9% in individuals aged $\geq$ 70 years, similar to findings from Jones et al. (2021) [27], Chelly et al. (2023) [62]; and Crook et al. (2021) [57]. On the other hand, this research has similar findings to the studies by Taquet et al. (2021) [14], Ganesh et al. (2021) [7], and Yong (2021) [5], which established a predominance of Long COVID in young adults. Analogous findings of higher LC rates in the age groups under 70 years align with the present data (Crook et al., 2021) [57]. Based on all these data and supported by the research by Asadi-Pooya et al. (2022) [83], whereby an incidence of 44.8% of Long COVID in children and adolescents was noted, the complexity of age-related patterns in Long COVID can be established. However, the low prevalence in the older age group might be explained by the low level of participants in this sample.

This study acknowledged the role of socioeconomic status in Long-Haul COVID, though with different characteristics, revealing a significant association between WHO LC and education, lowering the ceiling of educational level as opposed to earlier findings where individuals with low SES were 50% more likely to suffer from Long COVID [31] and those with graduate level education have lower likelihood of developing LC [74,84]. Moreover, this research did not establish a significant association between income, occupation, and Long-Haul COVID, in contrast to several studies that suggested the raised probability of LC in lower socioeconomic status [13,31,85,86]. This finding might be explained by the predominance of well-educated participants in this Mauritian sample (76.5% with at least secondary school level education) and, hence, better awareness [87] and concern about health. Additionally, public healthcare in Mauritius is free at the point of use for all citizens [88] and health inequity is low, as denoted by the remarkable distribution of services benefiting lower-income strata due to increased government investment [89]. This widespread equity, linked to better COVID-19 outcomes [87], could be another reason behind the lack of association between SES and Long COVID in Mauritius.

4.5. Effect of COVID-19 Vaccination on Long-Haul COVID

The cross-sectional analysis failed to uncover any clear link between the specific type of vaccine used or the administration of a fourth vaccine dose and the incidence of Long COVID. However, several other studies have consistently demonstrated that COVID-19 vaccination offers protective effects against Long COVID and reduces the risks of post-acute illness. For instance, Kuodi et al. (2022) [36], Peluso and Deeks (2022) [30], and Azzolini et al. (2022) [35] all reported that vaccinated individuals were less prone to developing Long COVID compared to unvaccinated individuals. Additionally, Ayoubkhani et al. (2022) [37] found that double-vaccinated individuals had a lower risk of experiencing Long COVID compared to unvaccinated individuals. On the other hand, this study found that 81.7% of fully vaccinated respondents showed no signs of NICE/SIGN/RCPG Long COVID, consistent with the general trend observed in other research, where vaccination has been associated with protective effects (Chelly et al., 2023) [62] and reduced risks of long COVID [90]. Nonetheless, it is also important to acknowledge the variability in findings across different studies. Szabo et al. (2023) [2] found that 25% of fully vaccinated individuals still developed some form of long COVID. The discrepancies in results could be due to various factors, including differences in study populations, methodologies, and the specific definitions of Long COVID used in each study. The duration of follow-up and the presence of different variants of the virus may also contribute to variations in the observed outcomes. Additionally, the lack of association of LC prevalence with the type of vaccine and fourth dose could be a failure to impute other factors like COVID-19 variant, reinfections and natural immunity. Given that fully vaccinated individuals in this sample did not develop NICE/SIGN/RCGP LC, this further accentuates that using different definitions of Long COVID yields varying results. As such, the need for a standard case definition is primordial for improved diagnosis and bringing uniformity in research.

4.6. Associating COVID-19 Disease Severity/Symptomatology and Long COVID

The current study's finding that the respondents who did not develop WHO-defined Long COVID were those with non-severe COVID-19 infection is consistent with several pieces of literature. Taquet et al. (2021) [14] reported that a severe course of acute COVID-19 was linked to an increased likelihood of experiencing Long COVID. Furthermore, the persistence of LC symptoms was observed to be higher in hospitalised patients compared to non-hospitalised ones [91] and in those admitted to the ICU [75]. Halpin et al. (2021) [92] noted higher trends of LC manifestations in patients who were admitted. This aligns with the current study's finding that severe cases of COVID-19 were more likely to result in Long COVID. Additionally, the literature indicates that ICU admission and artificial ventilation were associated with an elevated risk of experiencing specific long-term effects. Asadi-Pooya et al. (2021) [29] observed a connection between ICU admission and brain fog, while Herrera et al. (2021) [24] identified a link between ICU admission and post-intensive care syndrome (PICS) and showed that the use of long-term ventilator support was associated with the development of PASC.

On the other end of the spectrum, the study by Osikomaiya et al. (2021) [93] in Nigeria reported that Long COVID was more prevalent in cases with moderate COVID-19 disease severity. Interestingly, several studies determined that Long COVID can still manifest even in cases of mild COVID-19 illness and may not necessarily be dependent on acute COVID-19 severity or hospitalisation status [5,18,40]. This contrasts with the current study's finding that all respondents who did not develop WHO-defined Long COVID were those who did not experience severe COVID-19 infection, but it does corroborate with the fact that there was no association between disease severity and NICE/SIGN/RCGP Long COVID. Nevertheless, it should be emphasised that the present study had a disproportionate

number of participants with severe cases (2.5%) of COVID-19 compared to non-severe ones (97.5%), and due to lack of clinical details, proper severity classification (mild, moderate, severe, critical) could not be applied in this sample.

The results of this study also point towards a lower incidence of LC in asymptomatic COVID cases, findings which align with previous research reported by Sudre et al. (2021) [15] and Ayegbusi et al. (2021) [20]. It was observed that a large proportion of cases did not develop Long COVID (62.1% according to WHO LC criteria and 66.7% according to NICE/SIGN/RCPG LC criteria) when experiencing $\leq$ five symptoms in the acute COVID phase. This observation supports the literature's notion that Long COVID may be less likely to occur in cases with fewer initial symptoms.

As expected, when the number of symptoms exceeded five during the first week of acute COVID-19 infection, 62.2% of cases exhibited signs of *WHO Long COVID* ($p < 0.01$), and 58.5% showed signs of NICE/SIGN/RCPG Long COVID ($p < 0.01$). This indicates that experiencing more than five symptoms during the initial phase of COVID-19 infection could predict the development of Long COVID, like the findings of Cazé et al. (2023) [94]. Additionally, Fernández-de-las-Peñas et al. (2021) [28] reported a link between the frequency of symptoms at hospitalisation and the presence of fatigue and dyspnoea in post-COVID stages, further supporting the importance of early symptomatology in predicting Long COVID outcomes. According to Sadat Larijani et al. (2022) [95], exhibiting multiple symptoms in the initial phase of infection points to an underlying hyper-immune reaction with a resulting cytokine cascade, which could rationalise the development of LC in such individuals. This raises the possibility that reducing acute symptoms through treatment might modify the risk of developing Long COVID [74].

This study also determined that those with SARS-CoV2 reinfections had a higher propensity to suffer from Long COVID, data in agreement with the research work by Bowe, Xie and Al-Aly (2022) [96] that pinpointed the elevated risk of post-acute COVID sequelae with repeated COVID infections. This could potentially be explained by the fact that, although natural immunity is provided by an initial infection, it fades over time, and this waning is found to be accelerated in infection by the Omicron variant [97]. Therefore, the weakened immune system from the previous infection, which also predisposes to higher reinfection risks [58], and the waning natural immunity could support the findings of this study. Thus, curbing infection spread in the first place would substantially decrease the burden of Long COVID.

4.7. Linking Baseline Comorbidities and Long-Haul COVID

This study found no significant association between the two long COVID variables *WHO long COVID* and NICE/SIGN/RCPG long COVID) and the respondents' history of diseases. Conversely, various research works have asserted the association between comorbidities and the risk of developing long COVID. Tsampasian et al. (2023) [75] reported an odds ratio (OR) of 2.48 (95% CI, 1.97–3.13), indicating a higher risk of Long COVID in individuals with comorbidities. Similarly, Mechi et al. (2021) [34] reported that diabetes, obesity, hypersensitivities, and obstructive lung pathologies influenced Long COVID incidence, while others showed that severe courses of Long COVID were predominant in individuals with two or more comorbidities at baseline [24,40,98]. The current study did not delve into specific individual diseases but focused on the overall history of diseases, that is, comorbidity present vs. comorbidity absent. Moreover, the subgroup without any history of diseases was larger (64.9% vs. 35.1%), and multimorbidity was not investigated, which may be the reason why an association with the incidence of Long COVID could not be established in this study.

Nevertheless, the findings reported significant differences with respect to self-perception of illness, such that the way individuals perceived their acute infection played a role in the manifestation of Long COVID symptoms. A total of 39.1% and 49.2% of respondents who perceived their acute illness to be like a common cold displayed no symptoms of *WHO Long COVID* and *NICE/SIGN/RCGP Long COVID*, respectively. A France-based study also uncovered the effects of self-perception on LC manifestation, where individuals with a positive belief in having had COVID-19, despite negative serology results, showed higher odds of experiencing persistent symptoms. Besides hearing impairment, joint pain, and sleep problems, all other persisting symptoms were more prominent in those with a self-perception of having COVID-19 infection, suggesting that persistent symptoms may not be exclusive to SARS-CoV-2 [99] or may be psychosomatic expressions related to the fear of being sick with COVID-19 [100]. This further highlights the long-lasting psychological burden of the COVID pandemic.

4.8. Quality of Life and Long COVID: Consequential Effects

In the current study, both fatigue and Long COVID were found to have negative influences on the physical QOL of the participants, with females exhibiting lower scores. This could be explained by the differences in functional ageing. Men are found to be less frail than women due to higher hormone levels, such as testosterone and growth hormone, which promote muscle cell regeneration and, hence, improved muscle mass [101]. Hence indicating better functional resilience in males. Furthermore, fatigue severity itself was revealed to be strongly impactful on *PCS-12*. Given the prominence of severe fatigue (46.0% in *FSS*), the substantial presence (68.4%) of poor physical QOL in this study can be explained. This aligns with previous studies which established a direct link between Long COVID fatigue and functional decline and, consequently, worsening of health-related quality of life [102,103].

In fact, in a study by Montes-Ibarra et al. (2022) [104], a considerable number of patients (64%) experienced functional impairment, fatigue (69%), and reduced quality of life (72%) during Long COVID. The underlying mechanisms were found to be multifactorial, explaining the substantial degree of effects on different populations [105]. Furthermore, Lemhöfer et al. (2023) [106] identified the intensity of acute phase symptoms and the level of personal resilience as statistically significant predictors of the PCS score. Given that all the participants who did not develop Long COVID were those with non-severe COVID-19 infection, the degree of LC fatigue and poor physical QOL in this study sample is coherent.

Other studies that have been pertinent about the direct and indirect afflictions of Long COVID on health-related quality of life (HRQOL) emphasised how the pervasive and deep-seated pathophysiology of Long COVID fatigue and their tenacious ramifications on different pathways for physical functions precipitate prolonged decline in the physical and overall quality of life of COVID long-haulers. The findings accentuate the imperative for targeted interventions and comprehensive care for COVID long-haulers in Mauritius and around the globe to improve their physical and overall well-being.

Presently, neither Long-COVID (both WHO and NICE/SIGN/RCGP) nor the fatigue scores (both *FAS* and *FSS*) demonstrated any significant impact on the mental score of *SF-12*, but a statistically significant impact of COVID severity on *MCS-12* score was found, which could be related to the stress around long ICU stay and fear of giving way to the fatality of the disease. The presence of baseline mental burden as a confounding variable should not be downplayed as well, a variable which was unfortunately not examined. Additional research is required to gain a deeper understanding of how patients readjust their psychological states and their mechanisms of resilience.

Overall, this study denotes a higher impact of *WHO Long COVID* on fatigue in comparison to *NICE/SIGN/RCGP Long COVID*, further suggesting that certain aspects of Long COVID may be particularly influential in driving fatigue experiences and, subsequently, alterations in quality of life. These results emphasise the importance of addressing fatigue symptoms in individuals with Long COVID, with particular attention to females, and highlight the need for targeted interventions to manage and alleviate this distressing and prevalent symptom.

4.9. Limitations and Future Directions

The co-administration of different brands of vaccine, baseline fatigue and functional/cognitive status, undiagnosed anaemia or hypothyroidism, and dietary regimen could be confounding variables that were not accounted for. Also, specific pathologies and multimorbidity were not investigated as independent variables against Long COVID, but comorbidity was treated as a nominal variable. The study relied on self-report of symptoms rather than objective physiological or cognitive measures. As such, these results must be seen as complementing, rather than replacing, analyses using patients' medical records. An important element of this research was to identify the cognitive and overt neurological impacts of Long COVID, features which may also be under the effect of pre-existing conditions that were not assessed. Hence, future works within those lines may include recruiting both hospitalised and non-hospitalised patients, with control groups and a standardised long-term follow-up after the acute COVID-19 stage. Regular consultation of their medical records, clinical examinations and paraclinical investigations could enhance the objectivity of the study, while clinical cognitive assessments using the Mini-Mental State Examination (MMSE) and fatigue scoring using the Fatigue Assessment Scale must be carried out during the first days of infection, to exclude baseline cognitive impairments and fatigue, hence leaning towards prospective and clinically objective studies to confirm the results uncovered in the current research.

The evident variability in outcomes arising from different definitions of Long COVID accentuates the imperative for a standardised case definition. This standardisation is pivotal for enhancing diagnostic accuracy and achieving uniformity in research efforts. This study revealed a noteworthy observation in that individuals with SARS-CoV-2 reinfections exhibited a significantly higher susceptibility to Long COVID. Thus, a primary focus on preventing COVID-19 transmission holds immense potential for substantially alleviating the burden of Long COVID, efforts which seem to be deterring as COVID-19 is being normalised. A profound comprehension of how patients readjust their psychological states and their mechanisms of resilience warrants deeper exploration through additional research efforts.

In light of these findings, it is fundamental for healthcare providers, policymakers, and researchers to channel their efforts towards targeted interventions and comprehensive care for COVID long-haulers, not just in Mauritius but on a global scale, with the primary goal of enhancing their physical well-being and overall quality of life.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/covid5010006/s1, Data S1: Demographics profile of the participants; Data S2: Symptoms in 1st Week of COVID-19 infection; Data S3: Scores of FAS and FSS Scales.

Author Contributions: Conceptualization, A.B.S. and M.P.; methodology, A.B.S. and M.P.; software, A.B.S.; validation, A.B.S., M.P. and J.S.; formal analysis, A.B.S.; investigation, A.B.S.; resources, A.B.S., M.P. and J.S.; data curation, A.B.S. and M.P.; writing—original draft preparation, A.B.S.; writing—review and editing, A.B.S., M.P. and J.S.; visualization, A.B.S.; supervision, M.P.; project

administration, A.B.S.; funding acquisition, M.P. and J.S. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the National Ethics Committee (NEC), Ministry of Health and Wellness (MHC/CT/NETH/2022/AV5; 10 January 2023).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Ciotti, M.; Ciccozzi, M.; Terrinoni, A.; Jiang, W.; Wang, C.; Bernardini, S. The COVID-19 pandemic. *Crit. Rev. Clin. Lab. Sci.* **2020**, *57*, 365–388. [CrossRef] [PubMed]
2. Szabo, S.; Zayachkivska, O.; Hussain, A.; Muller, V. What is Really 'Long COVID'? *Inflammopharmacology* **2023**, *31*, 551–557. [CrossRef]
3. Yang, L.; Liu, S.; Liu, J.; Zhang, Z.; Wan, X.; Huang, B.; Chen, Y.; Zhang, Y. COVID-19: Immunopathogenesis and Immunotherapeutics. *Signal Transduct. Target. Ther.* **2020**, *5*, 128. [CrossRef]
4. Sandler, C.; Wyller, V.; Moss-Morris, R.; Buchwald, D.; Crawley, E.; Hautvast, J.; Katz, B.; Knoop, H.; Little, P.; Taylor, R.; et al. Long COVID and Post-Infective Fatigue Syndrome: A Review. *Open Forum Infect. Dis.* **2021**, *8*, ofab440. [CrossRef]
5. Yong, S. Long COVID or Post-COVID-19 Syndrome: Putative Pathophysiology, Risk Factors, and Treatments. *Infect. Dis.* **2021**, *53*, 737–754. [CrossRef] [PubMed]
6. Raveendran, A.V. Long COVID-19: Challenges in the Diagnosis and Proposed Diagnostic Criteria. *Diabetes Metab. Syndr.* **2021**, *15*, 145–146. [CrossRef] [PubMed]
7. Ganesh, R.; Ghosh, A.; Nyman, M.; Croghan, I.; Grach, S.; Anstine, C.; Salonen, B.; Hurt, R. PROMIS Scales for Assessment of Persistent Post-COVID Symptoms: A Cross Sectional Study. *J. Prim. Care Amp. Community Health* **2021**, *12*, 215013272110304. [CrossRef] [PubMed]
8. Kayaaslan, B.; Eser, F.; Kalem, A.; Kaya, G.; Kaplan, B.; Kacar, D.; Hasanoglu, I.; Coskun, B.; Guner, R. Post-COVID syndrome: A single-center questionnaire study on 1007 participants recovered from COVID-19. *J. Med. Virol.* **2021**, *93*, 6566–6574. [CrossRef] [PubMed]
9. Faghy, M.A.; Owen, R.; Thomas, C.; Yates, J.; Ferraro, F.V.; Skipper, L.; Barley-McMullen, S.; Brown, D.A.; Ashton, R.E.M. Is Long COVID the next global health crisis? *J. Glob. Health* **2022**, *12*, 03047. [CrossRef]
10. NICE; SIGN; RCGP. *Overview: COVID-19 Rapid Guideline: Managing the Long-Term Effects of COVID-19*; Guidance NICE: London, UK, 2022. Available online: https://www.nice.org.uk/guidance/NG188 (accessed on 28 October 2022).
11. WHO. *A Clinical Case Definition of Post COVID-19 Condition by a Delphi Consensus, 6 October 2021*; WHO: Geneva, Switzerland, 2021. Available online: https://www.who.int/publications-detail-redirect/WHO-2019-nCoV-Post_COVID-19_condition-Clinical_case_definition-2021.1 (accessed on 28 October 2022).
12. Soriano, J.; Murthy, S.; Marshall, J.; Relan, P.; Diaz, J. A Clinical Case Definition of Post-COVID-19 Condition by a Delphi Consensus. *Lancet Infect. Dis.* **2022**, *22*, e102–e107. [CrossRef] [PubMed]
13. Hossain, M.; Hossain, K.; Saunders, K.; Uddin, Z.; Walton, L.; Raigangar, V.; Sakel, M.; Shafin, R.; Hossain, M.; Kabir, M.; et al. Prevalence of Long COVID symptoms in Bangladesh: A prospective Inception Cohort Study of COVID-19 survivors. *BMJ Glob. Health* **2021**, *6*, e006838. [CrossRef]
14. Taquet, M.; Dercon, Q.; Luciano, S.; Geddes, J.; Husain, M.; Harrison, P. Incidence, Co-occurrence, and Evolution of Long COVID Features: A 6-Month Retrospective Cohort Study of 273,618 Survivors of COVID-19. *PLoS Med.* **2021**, *18*, e1003773. [CrossRef] [PubMed]
15. Sudre, C.; Murray, B.; Varsavsky, T.; Graham, M.; Penfold, R.; Bowyer, R.; Pujol, J.; Klaser, K.; Antonelli, M.; Canas, L.; et al. Attributes and Predictors of Long COVID. *Nat. Med.* **2021**, *27*, 626–631. [CrossRef] [PubMed]
16. Muzyka, I.; Yakhnytska, M.; Savytska, M.; Zayachkivska, O. Long COVID prevalence and physiology-centered risks: Population-based study in Ukraine. *Inflammopharmacology* **2023**, *31*, 597–602. [CrossRef] [PubMed]
17. El Otmani, H.; Nabili, S.; Berrada, M.; Bellakhdar, S.; El Moutawakil, B.; Abdoh Rafai, M. Prevalence, characteristics and risk factors in a Moroccan cohort of Long COVID-19. *Neurol. Sci.* **2022**, *43*, 5175–5180. [CrossRef]

18. van Kessel, S.; Olde Hartman, T.; Lucassen, P.; van Jaarsveld, C. Post-Acute and Long COVID-19 Symptoms in Patients with Mild Diseases: A Systematic Review. *Fam. Pract.* **2021**, *39*, 159–167. [CrossRef] [PubMed]
19. Banerjee, I.; Robinson, J.; Leclézio, A.; Sathian, B.; Banerjee, I. Post COVID syndrome: A novel challenge and threat to international health. *Nepal J. Epidemiol.* **2022**, *12*, 1215–1219. [CrossRef] [PubMed]
20. Aiyegbusi, O.L.; Hughes, S.E.; Turner, G.; Rivera, S.C.; McMullan, C.; Chandan, J.S.; Haroon, S.; Price, G.; Davies, E.H.; Nirantharakumar, K.; et al. Symptoms, complications and management of long COVID: A review. *J. R. Soc. Med.* **2021**, *114*, 428–442. [CrossRef] [PubMed]
21. Ceban, F.; Ling, S.; Lui, L.; Lee, Y.; Gill, H.; Teopiz, K.; Rodrigues, N.; Subramaniapillai, M.; Di Vincenzo, J.; Cao, B.; et al. Fatigue and cognitive impairment in Post-COVID-19 Syndrome: A systematic review and meta-analysis. *Brain Behav. Immun.* **2022**, *101*, 93–135. [CrossRef]
22. Huang, C.; Huang, L.; Wang, Y.; Li, X.; Ren, L.; Gu, X.; Kang, L.; Guo, L.; Liu, M.; Zhou, X.; et al. 6-month consequences of COVID-19 in patients discharged from hospital: A cohort study. *Lancet* **2021**, *397*, 220–232. [CrossRef]
23. Chippa, V.; Aleem, A.; Anjum, F. *Post Acute Coronavirus (COVID-19) Syndrome*; StatPearl: Treasure Island, FL, USA, 2022. Available online: https://www.ncbi.nlm.nih.gov/books/NBK570608/ (accessed on 16 November 2022).
24. Herrera, J.; Niehaus, W.; Whiteson, J.; Azola, A.; Baratta, J.; Fleming, T.; Kim, S.; Naqvi, H.; Sampsel, S.; Silver, J.; et al. Multidisciplinary collaborative consensus guidance statement on the assessment and treatment of fatigue in postacute sequelae of SARS-CoV-2 infection (PASC) patients. *PM&R* **2021**, *13*, 1027–1043.
25. Khatib, S.; Sabobeh, T.; Habib, A.; John, S.; Gomez, R.; Sivasankar, S.; Masoud, A. Post-COVID-19 fatigue as a major health problem: A cross-sectional study from Missouri, USA. *Ir. J. Med. Sci.* **2022**, *192*, 699–705. [CrossRef] [PubMed]
26. Sykes, D.; Holdsworth, L.; Jawad, N.; Gunasekera, P.; Morice, A.; Crooks, M. Post-COVID-19 Symptom Burden: What Is Long COVID and How Should We Manage It? *Lung* **2021**, *199*, 113–119. [CrossRef] [PubMed]
27. Jones, R.; Davis, A.; Stanley, B.; Julious, S.; Ryan, D.; Jackson, D.; Halpin, D.; Hickman, K.; Pinnock, H.; Quint, J.; et al. Risk Predictors and Symptom Features of Long COVID Within a Broad Primary Care Patient Population Including Both Tested and Untested Patients. *Pragmatic Obs. Res.* **2021**, *12*, 93–104. [CrossRef] [PubMed]
28. Fernández-de-las-Peñas, C.; Palacios-Ceña, D.; Gómez-Mayordomo, V.; Palacios-Ceña, M.; Rodríguez-Jiménez, J.; de-la-Llave-Rincón, A.; Velasco-Arribas, M.; Fuensalida-Novo, S.; Ambite-Quesada, S.; Guijarro, C.; et al. Fatigue and Dyspnoea as Main Persistent Post-COVID-19 Symptoms in Previously Hospitalized Patients: Related Functional Limitations and Disability. *Respiration* **2021**, *101*, 132–141. [CrossRef] [PubMed]
29. Asadi-Pooya, A.; Akbari, A.; Emami, A.; Lotfi, M.; Rostamihosseinkhani, M.; Nemati, H.; Barzegar, Z.; Kabiri, M.; Zeraatpisheh, Z.; Farjoud-Kouhanjani, M.; et al. Long COVID syndrome-associated brain fog. *J. Med. Virol.* **2021**, *94*, 979–984. [CrossRef]
30. Peluso, M.; Deeks, S. Early Clues Regarding the Pathogenesis of Long COVID. *Trends Immunol.* **2022**, *43*, 268–270. [CrossRef]
31. Paul, E.; Fancourt, D. Does Pre-Infection Stress Increase the Risk of Long COVID? Longitudinal Associations between Adversity Worries and Experiences in the Month Prior to COVID-19 Infection and the Development of Long COVID and Specific Long COVID Symptoms. *medRxiv* **2022**. [CrossRef]
32. Ziauddeen, N.; Gurdasani, D.; O'Hara, M.; Hastie, C.; Roderick, P.; Yao, G.; Alwan, N. Characteristics and Impact of Long COVID: Findings from an Online Survey. *PLoS ONE* **2022**, *17*, e0264331. [CrossRef] [PubMed]
33. Yoo, S.; Liu, T.; Motwani, Y.; Sim, M.; Viswanathan, N.; Samras, N.; Hsu, F.; Wenger, N. Factors Associated with Post-Acute Sequelae of SARS-CoV-2 (PASC) After Diagnosis of Symptomatic COVID-19 in the Inpatient and Outpatient Setting in a Diverse Cohort. *J. Gen. Intern. Med.* **2022**, *37*, 1988–1995. [CrossRef] [PubMed]
34. Mechi, A.; Al-Khalidi, A.; AL-Darraji, R.; Al-Dujaili, M.; Al-Buthabhak, K.; Alareedh, M.; Shaghee, F.; Nafakhi, H. Long-term persistent symptoms of COVID-19 infection in patients with diabetes mellitus. *Int. J. Diabetes Dev. Ctries.* **2021**, *42*, 49–52. [CrossRef] [PubMed]
35. Azzolini, E.; Levi, R.; Sarti, R.; Pozzi, C.; Mollura, M.; Mantovani, A.; Rescigno, M. Association Between BNT162b2 Vaccination and Long COVID After Infections Not Requiring Hospitalization in Health Care Workers. *JAMA* **2022**, *328*, 676–678. [CrossRef] [PubMed]
36. Kuodi, P.; Gorelik, Y.; Zayyad, H.; Wertheim, O.; Wiegler, K.; Jabal, K.; Dror, A.; Nazzal, S.; Glikman, D.; Edelstein, M. Association between vaccination status and reported incidence of post-acute COVID-19 symptoms in Israel: A cross-sectional study of patients tested between March 2020 and November 2021. *NPJ Vaccines* **2022**, *7*, 101. [CrossRef] [PubMed]
37. Ayoubkhani, D.; Bosworth, M.; King, S.; Pouwels, K.; Glickman, M.; Nafilyan, V.; Zaccardi, F.; Khunti, K.; Alwan, N.; Walker, A. Risk of Long COVID in people infected with SARS-CoV-2 after two doses of a COVID-19 vaccine: Community-based, matched cohort study. *medRxiv* **2022**. [CrossRef]
38. Tenforde, M.; Kim, S.; Lindsell, C.; Billig Rose, E.; Shapiro, N.; Files, D.; Gibbs, K.; Erickson, H.; Steingrub, J.; Smithline, H.; et al. Symptom Duration and Risk Factors for Delayed Return to Usual Health Among Outpatients with COVID-19 in a Multistate Health Care Systems Network—United States, March–June 2020. *Morb. Mortal. Wkly. Rep.* **2020**, *69*, 993–998. [CrossRef]

39. O'Brien, K.; Townsend, L.; Dowds, J.; Bannan, C.; Nadarajan, P.; Kent, B.; Murphy, N.; Sheill, G.; Martin-Loeches, I.; Guinan, E. 1-Year Quality of Life and Health Outcomes in Patients Hospitalised with COVID-19: A Longitudinal Cohort Study. *Respir. Res.* **2022**, *23*, 115. [CrossRef] [PubMed]
40. Torjesen, I. COVID-19: Middle Aged Women Face Greater Risk of Debilitating Long Term Symptoms. *Br. Med. J.* **2021**, *372*, n829. [CrossRef] [PubMed]
41. Vaes, A.; Goërtz, Y.; Van Herck, M.; Machado, F.; Meys, R.; Delbressine, J.; Houben-Wilke, S.; Gaffron, S.; Maier, D.; Burtin, C.; et al. Recovery from COVID-19: A Sprint or Marathon? 6-Month Follow-Up Data from Online Long COVID-19 Support Group Members. *ERJ Open Res.* **2021**, *7*, 00141–2021. [CrossRef] [PubMed]
42. Noujaim, P.; Jolly, D.; Coutureau, C.; Kanagaratnam, L. Fatigue and quality of life in the year following SARS-CoV-2 infection. *BMC Infect. Dis.* **2022**, *22*, 541. [CrossRef] [PubMed]
43. Macpherson, K.; Cooper, K.; Harbour, J.; Mahal, D.; Miller, C.; Nairn, M. Experiences of living with Long COVID and of accessing healthcare services: A qualitative systematic review. *BMJ Open* **2022**, *12*, e050979. [CrossRef] [PubMed]
44. Mainous, A.G., III; Rooks, B.J.; Wu, V.; Orlando, F.A. COVID-19 Post-acute Sequelae Among Adults: 12 Month Mortality Risk. *Front. Med.* **2021**, *8*, 778434. [CrossRef]
45. Wan, E.Y.F.; Mathur, S.; Zhang, R.; Yang, V.K.; Lai, F.T.T.; Chui, C.S.L.; Li, X.; Wong, C.K.H.; Chan, E.W.Y.; You, K.H.; et al. Association of COVID-19 with Short- and Long-Term Risk of Cardiovascular Disease and Mortality: A Prospective Cohort in UK Biobank. *Cardiovasc. Res.* **2023**, *119*, 1718–1727. [CrossRef] [PubMed]
46. CDC. *More Than 3500 Americans Have Died from Long COVID-Related Illness in the First 30 Months of the Pandemic*; National Centre for Health Statistics, CDC, US Department of Health and Human Sciences: Maryland, DC, USA, 2022. Available online: https://www.cdc.gov/nchs/pressroom/nchs_press_releases/2022/20221214.htm (accessed on 10 February 2023).
47. GIS. *Communique COVID-19. Friday, 4th November 2022*; Government Information Service: Port Louis, Mauritius, 2022. Available online: https://gis.govmu.org/Pages/Newsroom/Covid-19.aspx (accessed on 4 November 2022).
48. Quinlan, N.D.; Hegde, V.; Bracey, D.N.; Pollet, A.; Johnson, R.M.; Dennis, D.A.; Jennings, J.M. Prevalence of Depressive Symptoms in Aseptic Revision Total Knee Arthroplasty Based on the Mode of Failure. *Arthroplast. Today* **2024**, *25*, 101298. [CrossRef]
49. Soh, S.-E.; Morello, R.; Ayton, D.; Ahern, S.; Scarborough, R.; Zammit, C.; Brand, M.; Stirling, R.; Zalcberg, J. Measurement Properties of the 12-Item Short Form Health Survey Version 2 in Australians with Lung Cancer: A Rasch Analysis. *Health Qual. Life Outcomes* **2021**, *19*, 157. [CrossRef] [PubMed]
50. Ahmad, N.S.; Sabri, A. Assessing the unidimensionality, reliability, validity and fitness of influential factors of 8th grades student's Mathematics achievement in Malaysia. *Int. J. Adv. Res.* **2013**, *1*, 1–7.
51. Weiner, B.J.; Lewis, C.C.; Stanick, C.; Powell, B.J.; Dorsey, C.N.; Clary, A.S.; Boynton, M.H.; Halko, H. Psychometric assessment of three newly developed implementation outcome measures. *Implement. Sci.* **2017**, *12*, 108. [CrossRef]
52. Munblit, D.; O'Hara, M.E.; Akrami, A.; Perego, E.; Olliaro, P.; Needham, D.M. Long COVID: Aiming for a consensus. *Lancet. Respir. Med.* **2022**, *10*, 632–634. [CrossRef] [PubMed]
53. Talhari, C.; Criado, P.R.; Castro, C.C.S.; Ianhez, M.; Ramos, P.M.; Miot, H.A. Prevalence of and Risk Factors for Post-COVID: Results from a Survey of 6958 Patients from Brazil. *An. Acad. Bras. Ciências* **2023**, *95*, e20220143. [CrossRef]
54. Carfì, A.; Bernabei, R.; Landi, F.; for the Gemelli Against COVID-19 Post-Acute Care Study Group. Persistent Symptoms in Patients After Acute COVID-19. *JAMA* **2020**, *324*, 603–605. [CrossRef]
55. Michelen, M.; Manoharan, L.; Elkheir, N.; Cheng, V.; Dagens, A.; Hastie, C.; O'Hara, M.; Suett, J.; Dahmash, D.; Bugaeva, P.; et al. Characterising long COVID: A living systematic review. *BMJ Glob. Health* **2021**, *6*, e005427. [CrossRef] [PubMed]
56. Proal, A.; VanElzakker, M. Long COVID or Post-Acute Sequelae of COVID-19 (PASC): An Overview of Biological Factors That May Contribute to Persistent Symptoms. *Front. Microbiol.* **2021**, *12*, 698169. [CrossRef]
57. Crook, H.; Raza, S.; Nowell, J.; Young, M.; Edison, P. Long COVID—Mechanisms, risk factors, and management. *BMJ* **2021**, *374*, n1648. [CrossRef]
58. Davis, H.E.; McCorkell, L.; Vogel, J.M.; Topol, E.J. Long COVID: Major findings, mechanisms and recommendations. *Nat. Rev. Microbiol.* **2023**, *21*, 133–146. [CrossRef]
59. Ortelli, P.; Ferrazzoli, D.; Sebastianelli, L.; Engl, M.; Romanello, R.; Nardone, R.; Bonini, I.; Koch, G.; Saltuari, L.; Quartarone, A.; et al. Neuropsychological and Neurophysiological Correlates of Fatigue in Post-Acute Patients with Neurological Manifestations of COVID-19: Insights into a Challenging Symptom. *J. Neurol. Sci.* **2021**, *420*, 117271. [CrossRef]
60. Phetsouphanh, C.; Darley, D.R.; Wilson, D.B.; Howe, A.; Munier, C.M.L.; Patel, S.K.; Juno, J.A.; Burrell, L.M.; Kent, S.J.; Dore, G.J.; et al. Immunological Dysfunction Persists for 8 Months Following Initial Mild-to-Moderate SARS-CoV-2 Infection. *Nat. Immunol.* **2022**, *23*, 210–216. [CrossRef] [PubMed]
61. Calabria, M.; García-Sánchez, C.; Grunden, N.; Pons, C.; Arroyo, J.A.; Gómez-Anson, B.; Estévez García, M.D.C.; Belvís, R.; Morollón, N.; Vera Igual, J.; et al. Post-COVID-19 fatigue: The contribution of cognitive and neuropsychiatric symptoms. *J. Neurol.* **2022**, *269*, 3990–3999. [CrossRef]

62. Chelly, S.; Rouis, S.; Ezzi, O.; Ammar, A.; Fitouri, S.; Soua, A.; Fathallah, I.; Njah, M.; Mahjoub, M. Symptoms and risk factors for Long COVID in Tunisian population. *BMC Health Serv. Res.* **2023**, *23*, 487. [CrossRef] [PubMed]
63. Couzin-Frankel, J. Clues to Long COVID Scientists strive to unravel what is driving disabling symptoms. *Science* **2020**, *376*, 1261–1265. [CrossRef]
64. Lai, Y.-J.; Liu, S.-H.; Manachevakul, S.; Lee, T.-A.; Kuo, C.-T.; Bello, D. Biomarkers in Long COVID-19: A systematic review. *Front. Med.* **2023**, *10*, 1085988. [CrossRef]
65. Spudich, S.; Nath, A. Nervous System Consequences of COVID-19. *Science* **2022**, *375*, 267–269. [CrossRef] [PubMed]
66. Swank, Z.; Senussi, Y.; Manickas-Hill, Z.; Yu, X.G.; Li, J.Z.; Alter, G.; Walt, D.R. Persistent Circulating Severe Acute Respiratory Syndrome Coronavirus 2 Spike Is Associated with Post-Acute Coronavirus Disease 2019 Sequelae. *Clin. Infect. Dis.* **2023**, *76*, 487–490. [CrossRef] [PubMed]
67. Zeng, M.; Wang, D.Y.; Mullol, J.; Liu, Z. Chemosensory Dysfunction in Patients with COVID-19: What Do We Learn from the Global Outbreak? *Curr. Allergy Asthma Rep.* **2021**, *21*, 6. [CrossRef] [PubMed]
68. Zheng, Z.S.; Simonian, N.; Wang, J.; Rosario, E. Transcutaneous Vagus Nerve Stimulation Improves Long COVID Symptoms in a Female Cohort: A Pilot Study. *Front. Neurol.* **2024**, *15*, 1393371. [CrossRef]
69. Castanares-Zapatero, D.; Chalon, P.; Kohn, L.; Dauvrin, M.; Detollenaere, J.; Maertens de Noordhout, C.; Primus-de Jong, C.; Cleemput, I.; Van den Heede, K. Pathophysiology and mechanism of long COVID: A comprehensive review. *Ann. Med.* **2022**, *54*, 1473–1487. [CrossRef]
70. Rai, D.K.; Sharma, P.; Karmakar, S.; Thakur, S.; Ameet, H.; Yadav, R.; Gupta, V.B. Approach to Post-COVID-19 Persistent Cough: A Narrative Review. *Lung India Off. Organ Indian Chest Soc.* **2023**, *40*, 149–154. [CrossRef]
71. Koc, H.C.; Xiao, J.; Liu, W.; Li, Y.; Chen, G. Long COVID and its Management. *Int. J. Biol. Sci.* **2022**, *18*, 4768–4780. [CrossRef]
72. Puntmann, V.O.; Carerj, M.L.; Wieters, I.; Fahim, M.; Arendt, C.; Hoffman, J.; Shchendrygina, A.; Escher, F.; Vasa-Nicotera, M.; Zeiher, A.M.; et al. Outcomes of Cardiovascular Magnetic Resonance Imaging in Patients Recently Recovered from Coronavirus Disease 2019 (COVID-19). *JAMA Cardiol.* **2020**, *5*, 1265–1273. [CrossRef]
73. Roca-Fernandez, A.; Wamil, M.; Carapella, V.; Borlotti, A.; Monteiro, D.; Thamoadeis-Brears, H.; Dennis, A.; Banerjee, R.; Robson, M.D.; Lip, G.Y.H.; et al. Cardiac Impairment in Long COVID 1-Year Post SARS-CoV-2 Infection. *Eur. Heart J.* **2022**, *43*, ehac544.219. [CrossRef]
74. Durstenfeld, M.S.; Peluso, M.J.; Peyser, N.D.; Lin, F.; Knight, S.J.; Djibo, A.; Khatib, R.; Kitzman, H.; O'Brien, E.; Williams, N.; et al. Factors Associated with Long COVID Symptoms in an Online Cohort Study. *Open Forum Infect. Dis.* **2023**, *10*, ofad047. [CrossRef] [PubMed]
75. Tsampasian, V.; Elghazaly, H.; Chattopadhyay, R.; Debski, M.; Naing, T.K.P.; Garg, P.; Clark, A.; Ntatsaki, E.; Vassiliou, V.S. Risk Factors Associated with Post−COVID-19 Condition: A Systematic Review and Meta-Analysis. *JAMA Intern. Med.* **2023**, *183*, 566–580. [CrossRef]
76. Brodin, P. Immune determinants of COVID-19 disease presentation and severity. *Nat. Med.* **2021**, *27*, 28–33. [CrossRef]
77. Bai, F.; Tomasoni, D.; Falcinella, C.; Barbanotti, D.; Castoldi, R.; Mulè, G.; Augello, M.; Mondatore, D.; Allegrini, M.; Cona, A.; et al. Female gender is associated with Long COVID syndrome: A prospective cohort study. *Clin. Microbiol. Infect. Off. Publ. Eur. Soc. Clin. Microbiol. Infect. Dis.* **2022**, *28*, 611.e9–611.e16. [CrossRef] [PubMed]
78. Conti, V.; Corbi, G.; Sabbatino, F.; De Pascale, D.; Sellitto, C.; Stefanelli, B.; Bertini, N.; De Simone, M.; Liguori, L.; Di Paola, I.; et al. Long COVID: Clinical Framing, Biomarkers, and Therapeutic Approaches. *J. Pers. Med.* **2023**, *13*, 334. [CrossRef] [PubMed]
79. Petrella, C.; Nenna, R.; Petrarca, L.; Tarani, F.; Paparella, R.; Mancino, E.; di Mattia, G.; Conti, M.G.; Matera, L.; Bonci, E.; et al. Serum NGF and BDNF in Long-COVID-19 Adolescents: A Pilot Study. *Diagnostics* **2022**, *12*, 1162. [CrossRef] [PubMed]
80. Patel, U.K.; Mehta, N.; Patel, A.; Patel, N.; Ortiz, J.F.; Khurana, M.; Urhoghide, E.; Parulekar, A.; Bhriguvanshi, A.; Patel, N.; et al. Long-Term Neurological Sequelae Among Severe COVID-19 Patients: A Systematic Review and Meta-Analysis. *Cureus* **2022**, *14*, e29694. [CrossRef] [PubMed]
81. Sylvester, S.V.; Rusu, R.; Chan, B.; Bellows, M.; O'Keefe, C.; Nicholson, S. Sex Differences in Sequelae from COVID-19 Infection and in Long COVID Syndrome: A Review. *Curr. Med. Res. Opin.* **2022**, *38*, 1391–1399. [CrossRef] [PubMed]
82. Takahashi, T.; Ellingson, M.K.; Wong, P.; Israelow, B.; Lucas, C.; Klein, J.; Silva, J.; Mao, T.; Oh, J.E.; Tokuyama, M.; et al. Sex Differences in Immune Responses that Underlie COVID-19 Disease Outcomes. *Nature* **2020**, *588*, 315–320. [CrossRef] [PubMed]
83. Asadi-Pooya, A.; Nemati, H.; Shahisavandi, M.; Akbari, A.; Emami, A.; Lotfi, M.; Rostamihosseinkhani, M.; Barzegar, Z.; Kabiri, M.; Zeraatpisheh, Z.; et al. Correction to: Long COVID in children and adolescents. *World J. Pediatr.* **2022**, *17*, 495–499. [CrossRef]
84. Perlis, R.H.; Santillana, M.; Ognyanova, K.; Safarpour, A.; Lunz Trujillo, K.; Simonson, M.D.; Green, J.; Quintana, A.; Druckman, J.; Baum, M.A.; et al. Prevalence and Correlates of Long COVID Symptoms Among US Adults. *JAMA Netw. Open* **2022**, *5*, e2238804. [CrossRef]

85. Heller, O.; Chun, Y.; Shapira, S.; Troen, A.; Shlomo, Y.; Acri, M.; Marotta, P.; Kulkarni, S.; Kinnison, B.; Grinstein-Weiss, M. Prevalence of Long-COVID Among Low-Income and Marginalized Groups: Evidence from Israel. *Int. J. Public Health* **2022**, *67*, 1605086. [CrossRef] [PubMed]
86. Shabnam, S.; Razieh, C.; Dambha-Miller, H.; Yates, T.; Gillies, C.; Chudasama, Y.V.; Pareek, M.; Banerjee, A.; Kawachi, I.; Lacey, B.; et al. Socioeconomic Inequalities of Long COVID: A Retrospective Population-Based Cohort Study in the United Kingdom. *J. R. Soc. Med.* **2023**, *116*, 263–273. [CrossRef]
87. Lukkahatai, N.; Rodney, T.; Ling, C.; Daniel, B.; Han, H.-R. Long COVID in the context of social determinants of health. *Front. Public Health* **2023**, *11*, 1098443. [CrossRef] [PubMed]
88. Jeetoo, J.; Jaunky, V.C. Willingness to Pay to Improve Quality of Public Healthcare Services in Mauritius. *Healthcare* **2021**, *10*, 43. [CrossRef] [PubMed]
89. Nundoochan, A. Improving equity in the distribution and financing of health services in Mauritius, a small island state with deeply rooted welfare state standards. *BMJ Glob. Health* **2021**, *6*, e006757. [CrossRef] [PubMed]
90. Notarte, K.I.; Catahay, J.; Velasco, J.; Pastrana, A.; Ver, A.; Pangilinan, F.; Peligro, P.; Casimiro, M.; Guerrero, J.; Gellaco, M.; et al. Impact of COVID-19 vaccination on the risk of developing Long-COVID and on existing long-COVID symptoms: A systematic review. *eClinicalMedicine* **2022**, *53*, 101624. [CrossRef]
91. Castanares-Zapatero, D.; Kohn, L.; Dauvrin, M.; Detollenaere, J.; Maertens de Noorhout, C.; Primus-de Jong, C.; Rondia, K.; Chalon, P.; Cleemput, I.; Van den Heede, K. *Long COVID: Pathophysiology—Epidemiology and Patient Needs*; Belgian Health Care Knowledge Centre: Brussels, Belgium, 2021. Available online: https://www.kce.fgov.be/sites/default/files/2021-11/KCE_344_Long_Covid_scientific_report_1.pdf (accessed on 18 December 2022).
92. Halpin, S.J.; McIvor, C.; Whyatt, G.; Adams, A.; Harvey, O.; McLean, L.; Walshaw, C.; Kemp, S.; Corrado, J.; Singh, R.; et al. Postdischarge symptoms and rehabilitation needs in survivors of COVID-19 infection: A cross-sectional evaluation. *J. Med. Virol.* **2021**, *93*, 1013–1022. [CrossRef] [PubMed]
93. Osikomaiya, B.; Erinoso, O.; Wright, K.; Odusola, A.; Thomas, B.; Adeyemi, O.; Bowale, A.; Adejumo, O.; Falana, A.; Abdus-salam, I.; et al. 'Long COVID': Persistent COVID-19 Symptoms in Survivors Managed in Lagos State, Nigeria. *BMC Infect. Dis.* **2021**, *21*, 304. [CrossRef]
94. Cazé, A.B.; Cerqueira-Silva, T.; Bomfim, A.P.; de Souza, G.L.; Azevedo, A.C.; Brasil, M.Q.; Santos, N.R.; Khouri, R.; Dan, J.; Bandeira, A.C.; et al. Prevalence and risk factors for Long COVID after mild disease: A cohort study with a symptomatic control group. *J. Glob. Health* **2023**, *13*, 06015. [CrossRef] [PubMed]
95. Sadat Larijani, M.; Ashrafian, F.; Bagheri Amiri, F.; Banifazl, M.; Bavand, A.; Karami, A.; Asgari Shokooh, F.; Ramezani, A. Characterization of Long COVID-19 Manifestations and Its Associated Factors: A Prospective Cohort Study from Iran. *Microb. Pathog.* **2022**, *169*, 105618. [CrossRef]
96. Bowe, B.; Xie, Y.; Al-Aly, Z. Acute and postacute sequelae associated with SARS-CoV-2 reinfection. *Nat. Med.* **2022**, *28*, 2398–2405. [CrossRef] [PubMed]
97. Chemaitelly, H.; Nagelkerke, N.; Ayoud, H.H.; Coyle, P.; Tang, P.; Yassine, H.M.; Al-Khatib, H.A.; Smatti, M.K.; Hasan, M.R.; Al-Kanaani, Z.; et al. Duration of immune protection of SARS-CoV-2 natural infection against reinfection. *J. Travel Med.* **2022**, *29*, 109. [CrossRef]
98. Subramanian, A.; Nirantharakumar, K.; Hughes, S.; Myles, P.; Williams, T.; Gokhale, K.M.; Taverner, T.; Chandan, J.S.; Brown, K.; Simms-Williams, N.; et al. Symptoms and Risk Factors for Long COVID in Non-Hospitalized Adults. *Nat. Med.* **2022**, *28*, 1706–1714. [CrossRef]
99. Matta, J.; Wiernik, E.; Robineau, O.; Carrat, F.; Touvier, M.; Severi, G.; de Lamballerie, X.; Blanché, H.; Deleuze, J.-F.; Gouraud, C.; et al. Association of Self-reported COVID-19 Infection and SARS-CoV-2 Serology Test Results with Persistent Physical Symptoms Among French Adults During the COVID-19 Pandemic. *JAMA Intern. Med.* **2022**, *182*, 19–25. [CrossRef] [PubMed]
100. Zhong, Y.; Huang, J.; Zhang, W.; Li, S.; Gao, Y. Addressing Psychosomatic Issues After Lifting the COVID-19 Policy in China: A Wake-Up Call. *Asian J. Psychiatry* **2023**, *82*, 103517. [CrossRef] [PubMed]
101. Hägg, S.; Jylhävä, J. Sex differences in biological aging with a focus on human studies. *eLife* **2021**, *10*, e63425. [CrossRef]
102. de Oliveira Almeida, K.; Nogueira Alves, I.G.; de Queiroz, R.S.; de Castro, M.R.; Gomes, V.A.; Santos Fontoura, F.C.; Brites, C.; Neto, M.G. A systematic review on physical function, activities of daily living and health-related quality of life in COVID-19 survivors. *Chronic Illn.* **2023**, *19*, 279–303. [CrossRef] [PubMed]
103. Tabacof, L.; Tosto-Mancuso, J.; Wood, J.; Cortes, M.; Kontorovich, A.; McCarthy, D.; Rizk, D.; Rozanski, G.; Breyman, E.; Nasr, L.; et al. Post-Acute COVID-19 Syndrome Negatively Impacts Physical Function, Cognitive Function, Health-Related Quality of Life, and Participation. *Am. J. Phys. Med. Rehabil.* **2022**, *101*, 48–52. [CrossRef]
104. Montes-Ibarra, M.; Oliveira, C.L.P.; Orsso, C.E.; Landi, F.; Marzetti, E.; Prado, C.M. The Impact of Long COVID-19 on Muscle Health. *Clin. Geriatr. Med.* **2022**, *38*, 545–557. [CrossRef]

105. Fernández-Lázaro, D.; Santamaría, G.; Sánchez-Serrano, N.; Lantarón Caeiro, E.; Seco-Calvo, J. Efficacy of Therapeutic Exercise in Reversing Decreased Strength, Impaired Respiratory Function, Decreased Physical Fitness, and Decreased Quality of Life Caused by the Post-COVID-19 Syndrome. *Viruses* **2022**, *14*, 2797. [CrossRef]
106. Lemhöfer, C.; Bahmer, T.; Baumbach, P.; Besteher, B.; Boekel, A.; Finke, K.; Katzer, K.; Lehmann-Pohl, K.; Lewejohann, J.-C.; Loudovici-Krug, D.; et al. Variations and Predictors of Post-COVID Syndrome Severity in Patients Attending a Post-COVID Outpatient Clinic. *J. Clin. Med.* **2023**, *12*, 4013. [CrossRef]