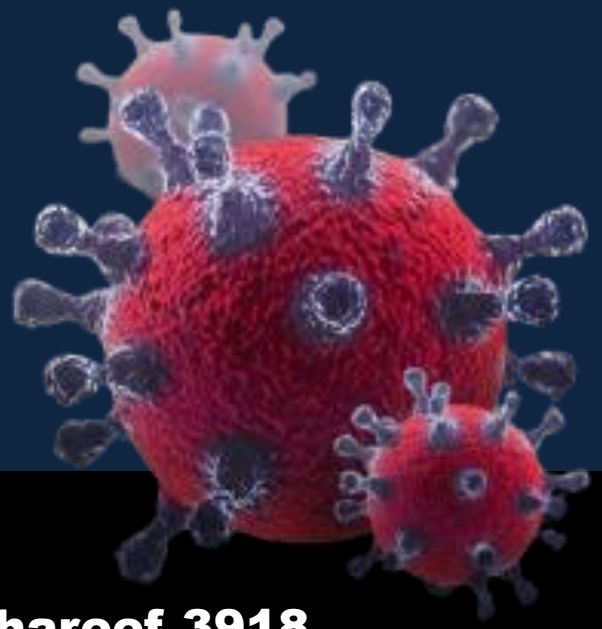
Long COVID

Presented By:
Aisha Alshebani 3250,
Ahmed Alabbar3234
Fatima Mellad 3456,
Seraj Hussine 4009
Mariam Abdalkader 4055 , Areej Alshareef 3918



Davis, H.E., McCorkell, L., Vogel, J.M. *et al.* (2023). Long COVID: major findings, mechanisms and recommendations. *Nat Rev Microbiol* **21**, 133–146. https://doi.org/10.1038/s41579-022-00846-2

Aims: 🎯

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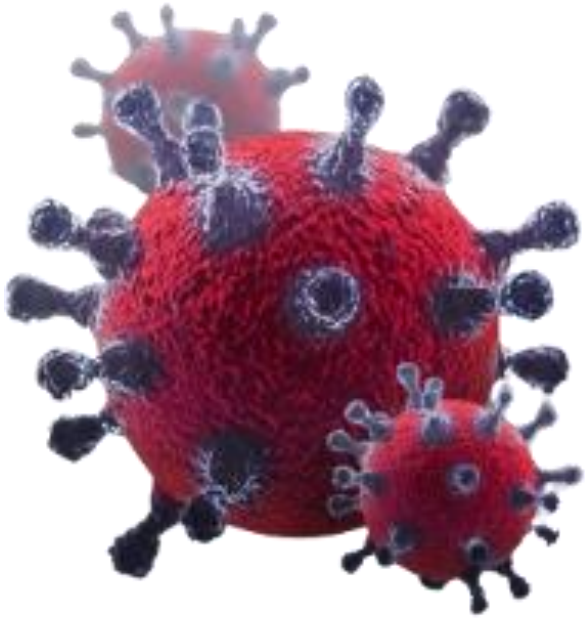

Introduction

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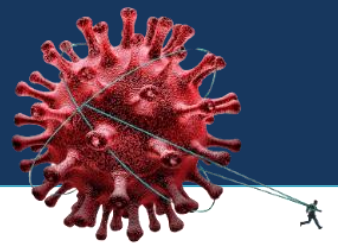
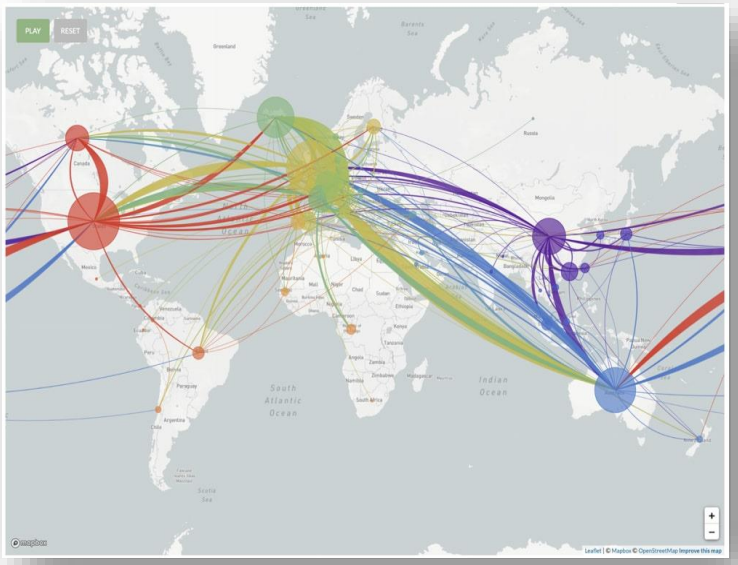


Long COVID (sometimes referred to as ‘post-acute sequelae of COVID-19’) is a multisystemic condition comprising often severe symptoms that follow a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection.

Epidemiology

At least 65 million individuals around the world have long COVID, based on a conservative estimated incidence of 10% of infected people and more than 651 million documented COVID-19 cases worldwide the number is likely much higher due to many undocumented cases.



Introduction

The incidence is estimated at 10–30% of non-hospitalized cases, 50–70% of hospitalized cases and 10–12% of vaccinated cases.

Long COVID is associated with all ages and acute phase disease severities, with the highest percentage of diagnoses between the ages of 36 and 50 years, and most long COVID cases are in non-hospitalized patients with a mild acute illness.

Clinical finding

Hundreds of biomedical findings have been documented, with many patients experiencing dozens of symptoms across multiple organ Systems (Fig. 1)

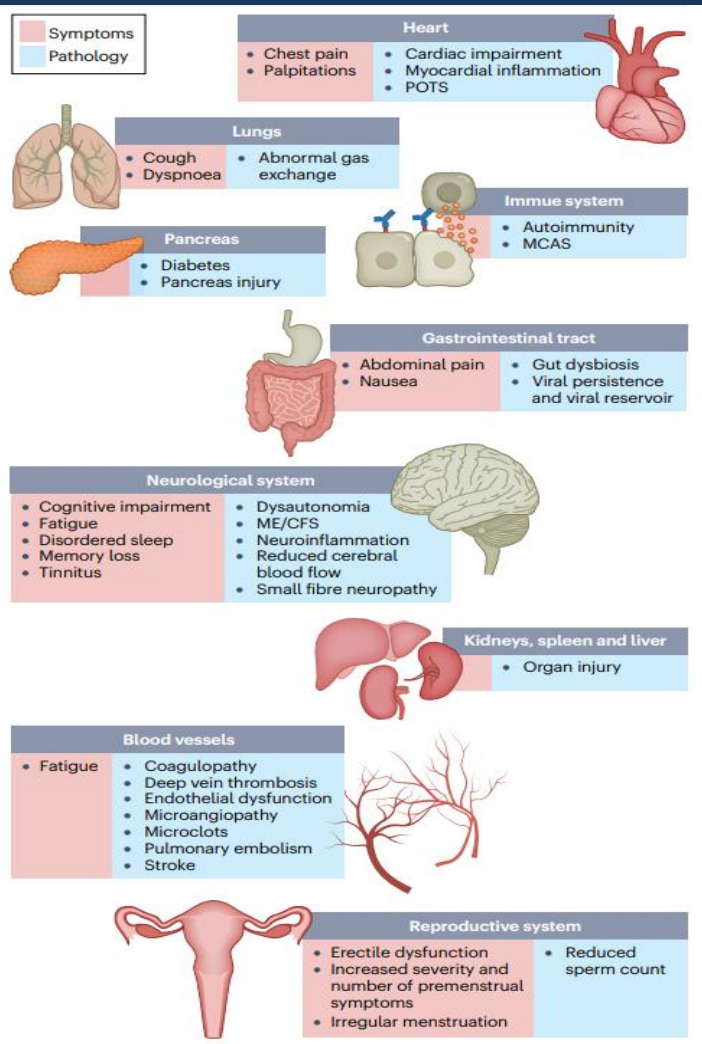


Fig. 1 | Long COVID symptoms and the impacts on numerous organs with differing pathology. The impacts of long COVID on numerous organs with a wide variety of pathology are shown. The presentation of pathologies is often

Introduction

Adverse effect

Long COVID encompasses multiple adverse outcomes with common new-onset conditions including:

- Cardiovascular and cerebrovascular disease
- Type 2 diabetes
- Myalgic encephalomyelitis/chronic fatigue syndrome
- Orthostatic tachycardia syndrome (POTS)

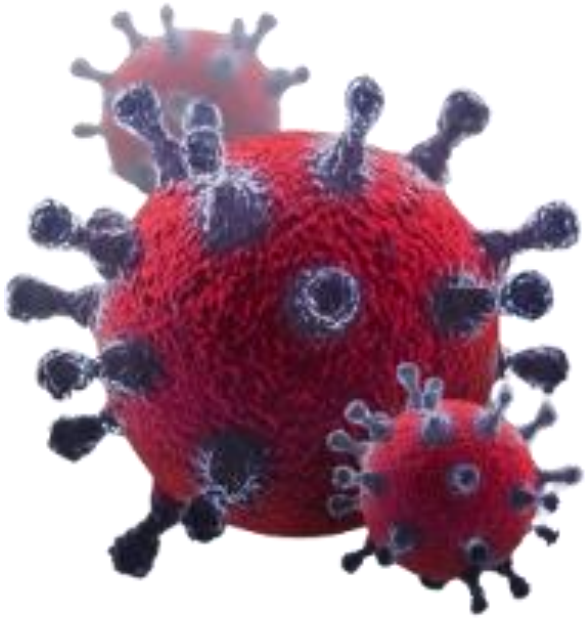
Symptoms can last for years, and particularly in cases of new-onset ME/CFS are expected to be lifelong.

Risk factor

Risk factor:

- Female sex
- Type 2 diabetes
- EBV reactivation
- Attention deficit hyperactivity disorder
- Chronic urticaria and allergic rhinitis
- Ethnicities including Latino heritage
- Low Socio-economic

Aim of the study To investigate immune dysregulation in long COVID patients



Methods

Methods

This study investigates immune dysregulation in long COVID patients. Here's a breakdown of the methods used:

T cell analysis:

Researchers examined the types and functions of T cells in long COVID patients compared to healthy controls and recovered COVID-19 individuals.

This likely involved isolating and analyzing T cells using techniques like flow cytometry and measuring markers like CD4 and CD8.

B cell analysis:

Studies looked at the number and activation state of B cells in long COVID patients.

Flow cytometry and specific antibody tests might have been employed.

Methods

Cytokine and interferon measurement:

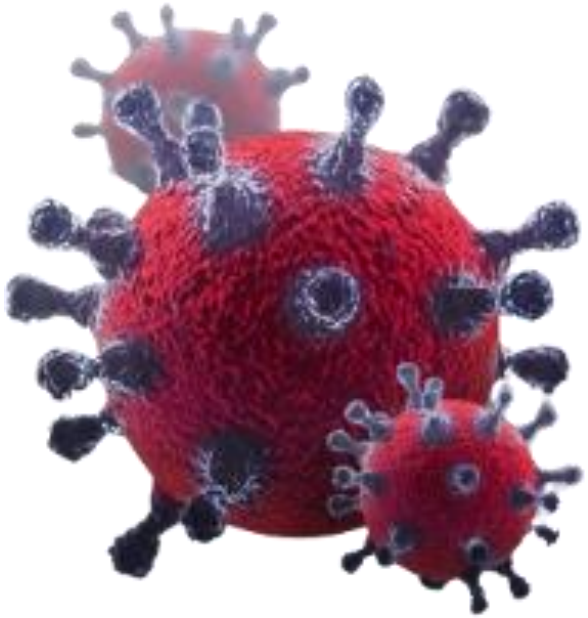
Researchers measured the levels of various immune signaling molecules like cytokines (IL-1β, IL-6, TNF-α, IP10) and interferons (IFN-β, IFN-λ1) in long COVID patients compared to controls.

This likely involved blood tests using immunoassays like ELISA.

Cortisol measurement:

Cortisol is a hormone involved in immune regulation.

Blood tests were likely used to measure cortisol levels in long COVID patients.



Result

Result

Long COVID and Immune Dysregulation: Key Findings; These studies reveal significant immune dysfunction in individuals with long COVID, even after mild initial COVID-19 infection. Here's a summary of the key findings:

T-cell abnormalities:

Exhausted T cells (less effective fighters) and reduced memory T cells (important for long-term immunity) persist for over a year.

Cortisol:

Low cortisol levels, potentially weaken the body's ability to regulate inflammation.

Result

Cytokines and interferons:

Elevated levels of inflammatory cytokines (IL-1β, IL-6, TNF-α, IP10) and interferons (IFN-β, IFN-λ1), potentially contribute to chronic inflammation.

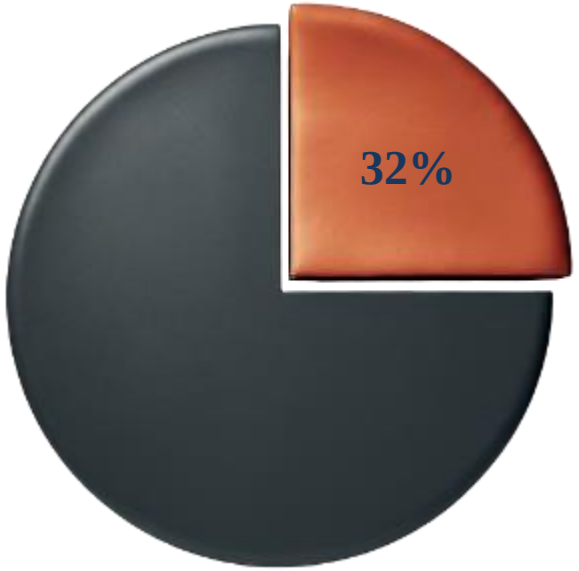
Autoimmunity:

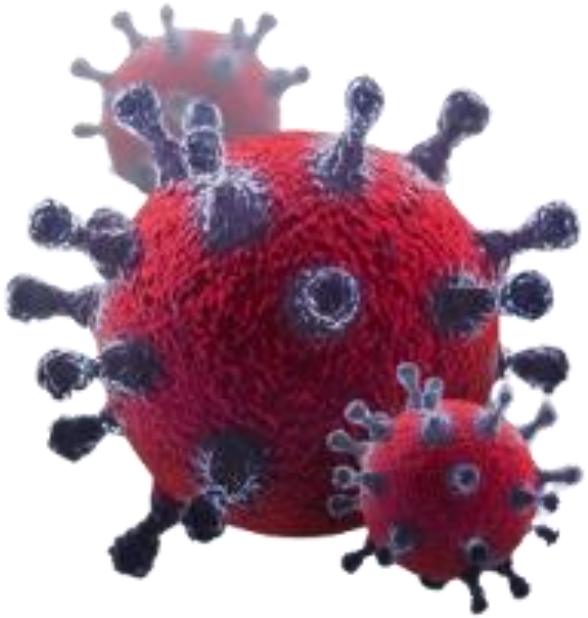
Conflicting evidence on autoantibodies (antibodies targeting the body's own tissues). Some studies show elevated autoantibodies in long COVID, potentially contributing to tissue damage.

A large study suggests autoimmunity may not be a major driver.

Result

In a meta-analysis, fatigue was found in 32% and cognitive impairment was found in 22% of patients with COVID-19 at 12 weeks after infection.





Discussion

Discussion

Chronic Inflammation: The persistent activation of innate immune cells and elevated inflammatory cytokines suggest a state of chronic low-grade inflammation in long COVID.

This could explain fatigue, pain, and other widespread symptoms.

Impaired Immune Response: The reduction in memory T and B cells, along with exhausted T cells, indicates a weakened ability to fight new infections and maintain long-term immunity.

This might explain increased susceptibility to other illnesses in long COVID patients.

Discussion

T-cell Exhaustion:

The link between cytotoxic T-cell expansion and gut problems is interesting.

It suggests a specific immune response may be driving some long COVID symptoms.

Cortisol and Immune Regulation:

The low cortisol levels observed could be a contributing factor or a consequence of the ongoing immune dysregulation.

Cortisol plays a role in suppressing inflammation, so understanding this relationship is important.

Discussion

There are likely multiple, potentially overlapping, causes of long COVID. Several hypotheses for its pathogenesis have been suggested, including:

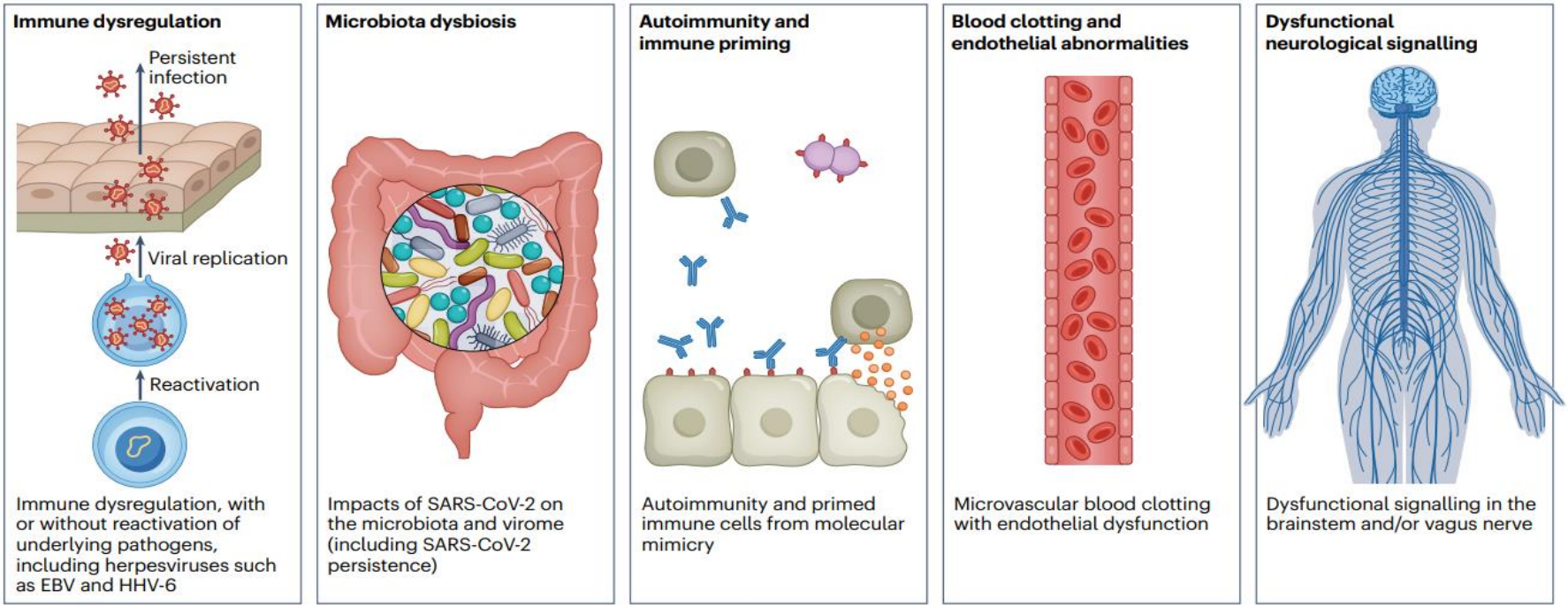
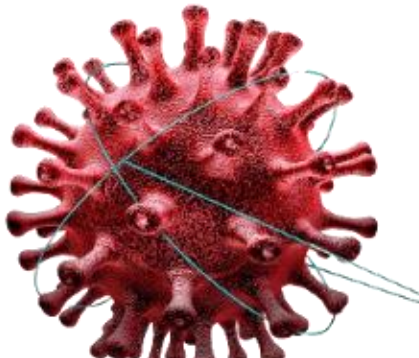


Fig. 3 | Hypothesized mechanisms of long COVID pathogenesis. There are several hypothesized mechanisms for long COVID pathogenesis, including immune dysregulation, microbiota disruption, autoimmunity, clotting and endothelial abnormality, and dysfunctional neurological signalling. EBV, Epstein–Barr virus; HHV-6, human herpesvirus 6; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

Discussion

Diagnostic and treatment:

Diagnostic tools for long COVID are mostly in development, including imaging to detect micro clots, corneal microscopy to identify small fiber neuropathy, new fragmentation of QRS complex on electrocardiograms as indicative of cardiac injury. Although there are currently no broadly effective treatments for long COVID.



Conclusion

Long COVID is a multisystemic illness encompassing ME/CFS, impacts on multiple organ systems, and vascular and clotting abnormalities.

It has already debilitated millions of individuals worldwide, and that number is continuing to grow.

Diagnostic and treatment options are currently insufficient, and many clinical trials are urgently needed to rigorously test treatments that address hypothesized underlying biological mechanisms, including viral persistence, neuroinflammation, excessive blood clotting, and autoimmunity.

Reference

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