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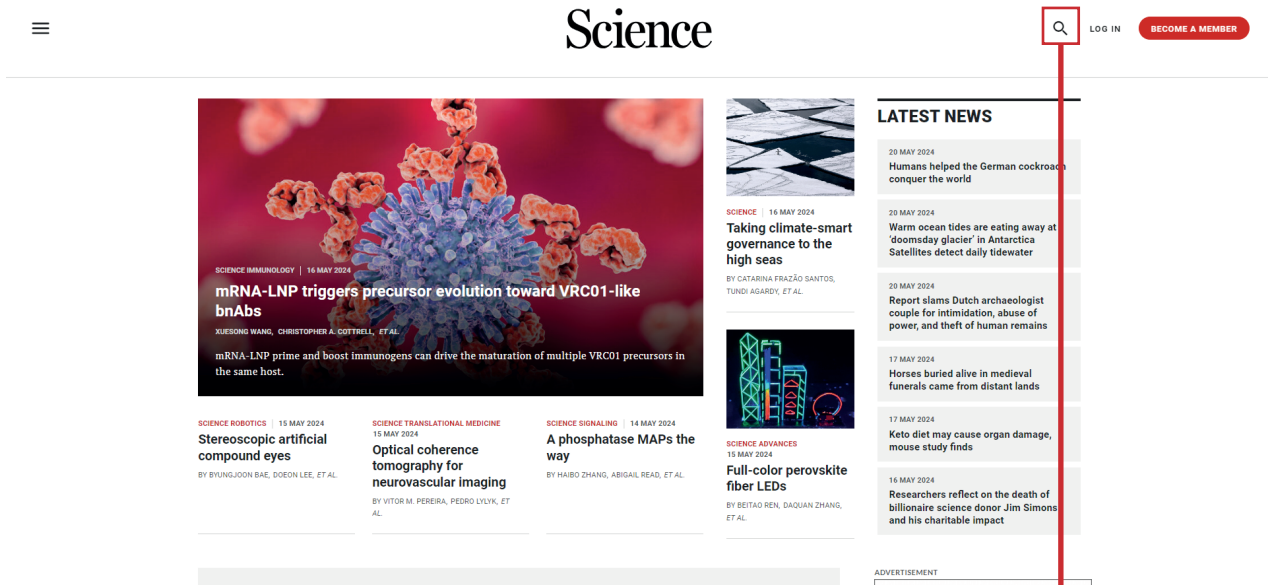


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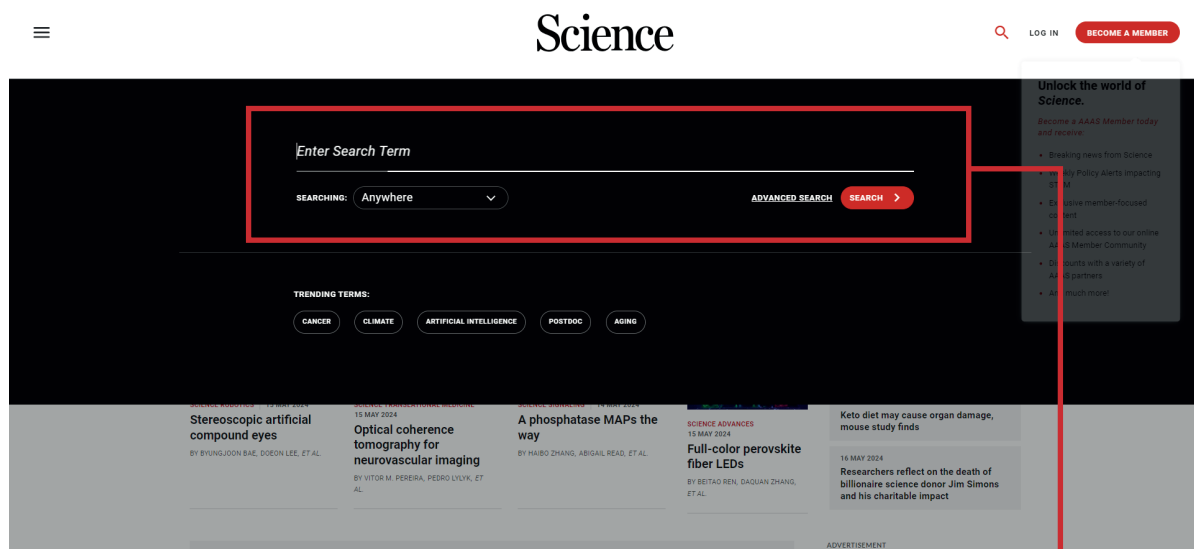
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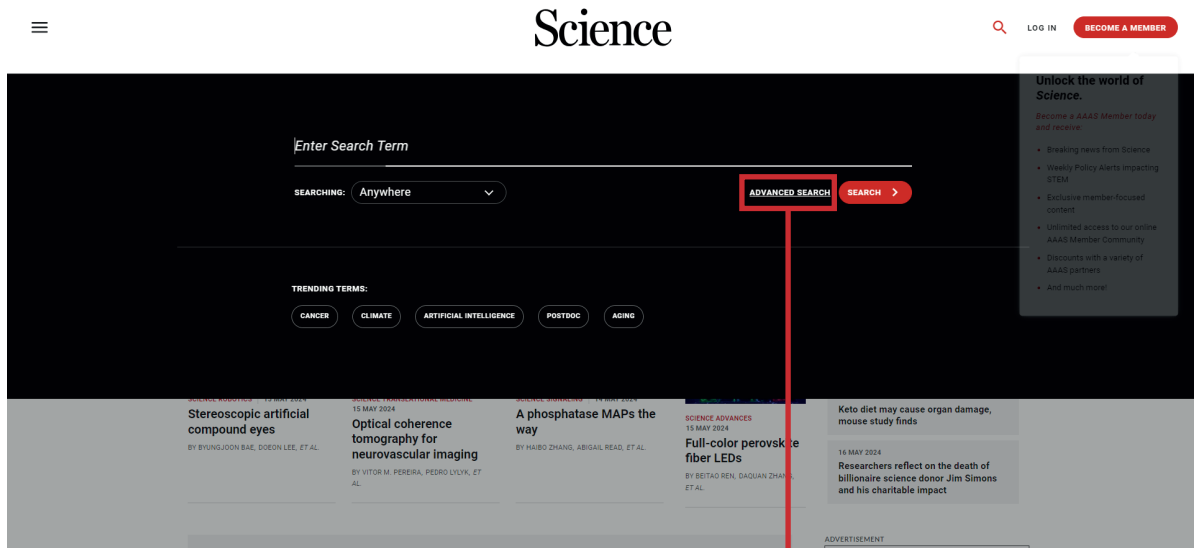


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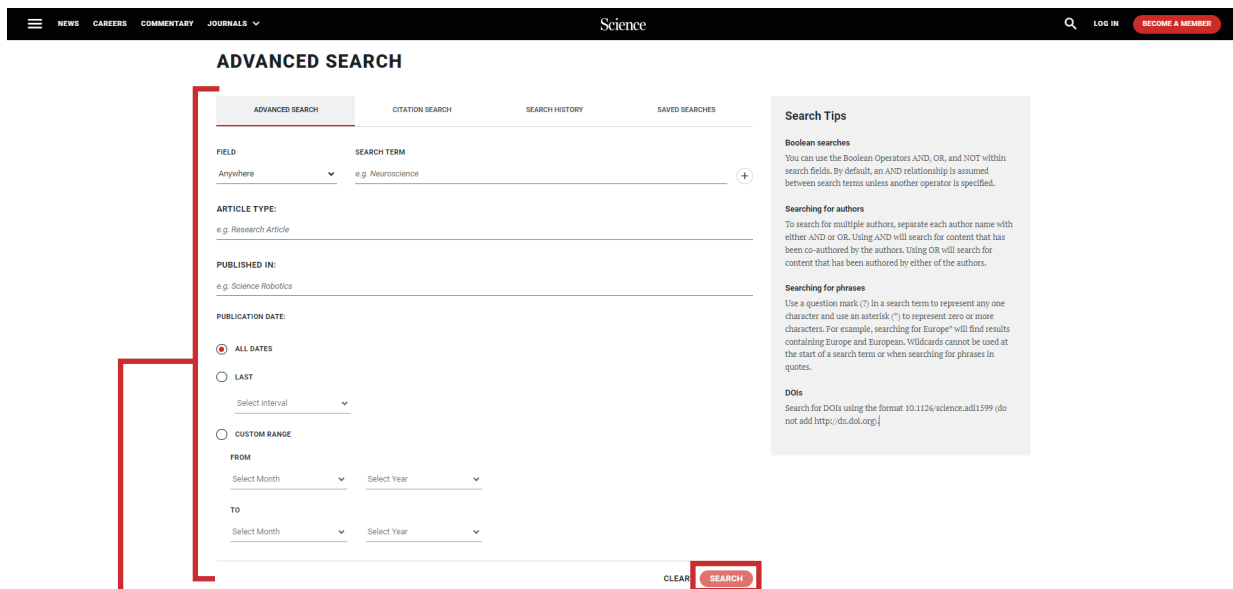
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 COVID-19 AND CHILDREN

| PERSPECTIVE | VIEWPOINTS: COVID-19
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COVID-19 and children

CARL A. PERES¹, DEVAN C. HEROLD¹, BETTY C. HEROLD¹, JONET CHOI², ADRIENNE RANDOLPH³, BINTA KANE⁴, SAIMME MCKINLAY⁴, DEEPTI DUREGANNI⁵, CHRISTINA PHILLIPS^{1, 6} AND SCOTT L. HENSLEY¹ +2 authors [Authors' Info & Affiliations](#)

SCIENCE • 8 SEP 2022 • Vol 377, Issue 6611 • pp. 1160-1169 • DOI: 10.1126/science.adc4679

37 565
 19

Why is COVID-19 generally milder in children?

Why do some children develop MIS-C?

Long Covid in children and young people

What is the role of children in transmission of SARS-CoV-2?


The importance of vaccinating children against COVID-19

Original research
Vaccine priming and laboratory responses against SARS-CoV-2

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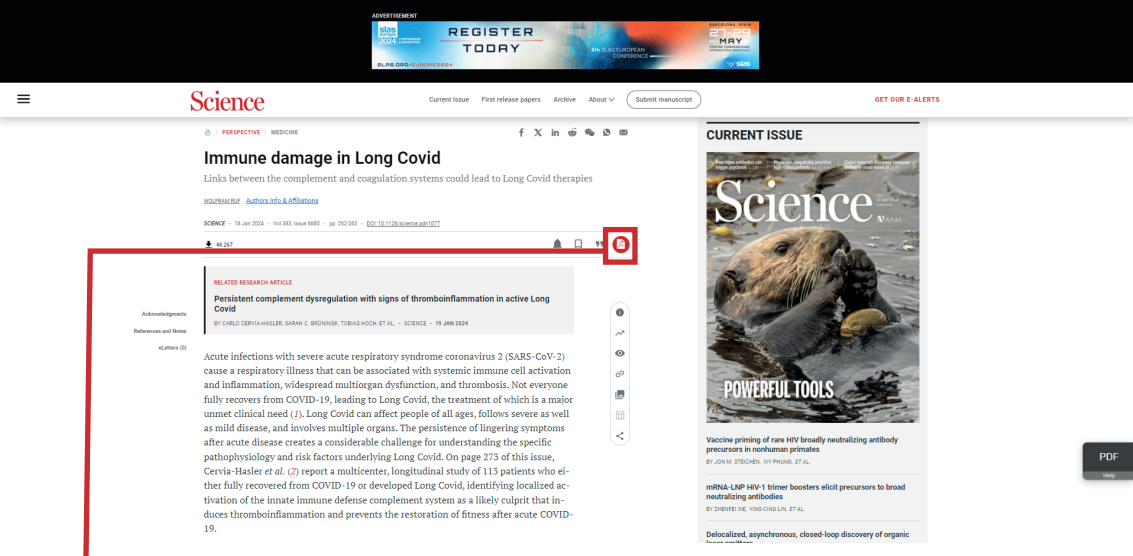
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Acute infections with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) cause a respiratory illness that can be associated with systemic immune cell activation and inflammation, widespread multiorgan dysfunction, and thrombosis. Not everyone fully recovers from COVID-19, leading to Long Covid, the treatment of which is a major unmet clinical need (1). Long Covid can affect people of all ages, follows severe as well as mild disease, and involves multiple organs. The persistence of lingering symptoms after acute disease creates a considerable challenge for understanding the specific pathophysiology and risk factors underlying Long Covid. On page 273 of this issue, Cerva-Hasler et al. (2) report a multicenter, longitudinal study of 115 patients who either fully recovered from COVID-19 or developed Long Covid, identifying localized activation of the innate immune defense complement system as a likely culprit that induces thromboinflammation and prevents the restoration of fitness after acute COVID-19.

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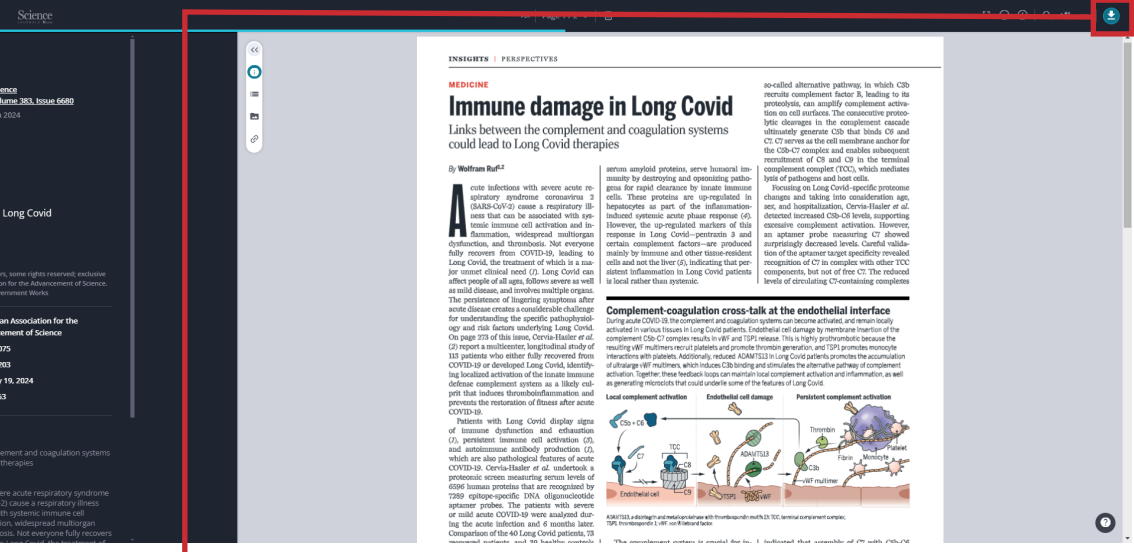
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INSIGHTS | PERSPECTIVES

Immune damage in Long Covid

Links between the complement and coagulation systems could lead to Long Covid therapies

By Wolfrum Ruff^{1,2}

Acute infections with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) cause a respiratory illness that can be associated with systemic immune cell activation and inflammation, widespread multiorgan dysfunction, and thrombosis. Not everyone fully recovers from COVID-19, leading to Long Covid, the treatment of which is a major unmet clinical need (1). Long Covid can affect people of all ages, follows severe as well as mild disease, and involves multiple organs. The persistence of lingering symptoms after acute disease creates a considerable challenge for understanding the specific pathophysiology and risk factors underlying Long Covid. On page 273 of this issue, Cerva-Hasler et al. (2) report a multicenter, longitudinal study of 115 patients who either fully recovered from COVID-19 or developed Long Covid, identifying localized activation of the innate immune defense complement system as a likely culprit that induces thromboinflammation and prevents the restoration of fitness after acute COVID-19.

Patients with Long Covid display signs of immune dysfunction and exhaustion (3), persistent immune cell activation (3), and autoimmune antibody production (4), which are also pathological features of acute COVID-19. Cerva-Hasler et al. undertook a proteomic screen measuring serum levels of 6596 human proteins that are recognized by 7289 rRNA-specific DNA oligonucleotide aptamer probes. The patients with severe or mild acute COVID-19 were analyzed during the acute infection and 6 months later. Comparisons of the 40 Long Covid patients, 79 unmet clinical need (1). Long Covid can affect people of all ages, follows severe as well as mild disease, and involves multiple organs. The persistence of lingering symptoms after acute disease creates a considerable challenge for understanding the specific pathophysiology and risk factors underlying Long Covid. On page 273 of this issue, Cerva-Hasler et al. (2) report a multicenter, longitudinal study of 115 patients who either fully recovered from COVID-19 or developed Long Covid, identifying localized activation of the innate immune defense complement system as a likely culprit that induces thromboinflammation and prevents the restoration of fitness after acute COVID-19.

so-called alternative pathway, in which C3b recruits complement factor B, leading to its proteolytic, can amplify complement activation on cell surfaces. The conservative proteolytic cleavages in the complement cascade ultimately generate C3b that binds C3 and C7, serving as the cell membrane anchor for the C3b-C7 complex and enables subsequent recruitment of C5 and C6 in the terminal complement complex (TCC), which mediates lysis of pathogens and host cells.

Focusing on Long Covid-specific proteome changes and taking into consideration age, sex, and hospitalization, Cerva-Hasler et al. detected increased C3b-C7 levels, supporting excessive complement activation. However, an aptamer probe measuring C7 showed surprisingly decreased levels. Careful validation of the aptamer target specificity revealed recognition of C7 in complex with other TCC components, but not of free C7. The reduced levels of circulating C7-containing complexes

Complement-coagulation cross-talk at the endothelial interface

During acute COVID-19, the complement and coagulation systems can become activated, and remain locally activated in various tissues in Long Covid patients. Endothelial cell damage by membrane insertion of the complement C3b-C7 complex results in vWF and TSP1 release. This is highly prothrombotic because the resulting vWF multimers recruit platelets and promote thrombin generation, and TSP1 promotes monocyte interactions with platelets. Additionally, reduced ADAMTSL1 in Long Covid patients promotes the accumulation of ultrahigh vWF multimers, which induces C3b binding and stimulates the alternative pathway of complement activation. Together, these feedback loops can maintain local complement activation and inflammation, as well as generating microclots that could underlie some of the features of Long Covid.

Local complement activation Endothelial cell damage Persistent complement activation

ADAMTSL1 is a disintegrin and metalloprotease with thrombospondin motifs. TCC, terminal complement complex; TSP1, thrombospondin 1; vWF, von Willebrand factor.

This microclot contains a microclot that is indicated that assembly of C7 with C3b/C7

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