

# SPRINGER NATURE Link

**Quick User Guide**

November 2024

**SPRINGER NATURE**

# INTRODUCING SPRINGER NATURE LINK

Home for all research

2

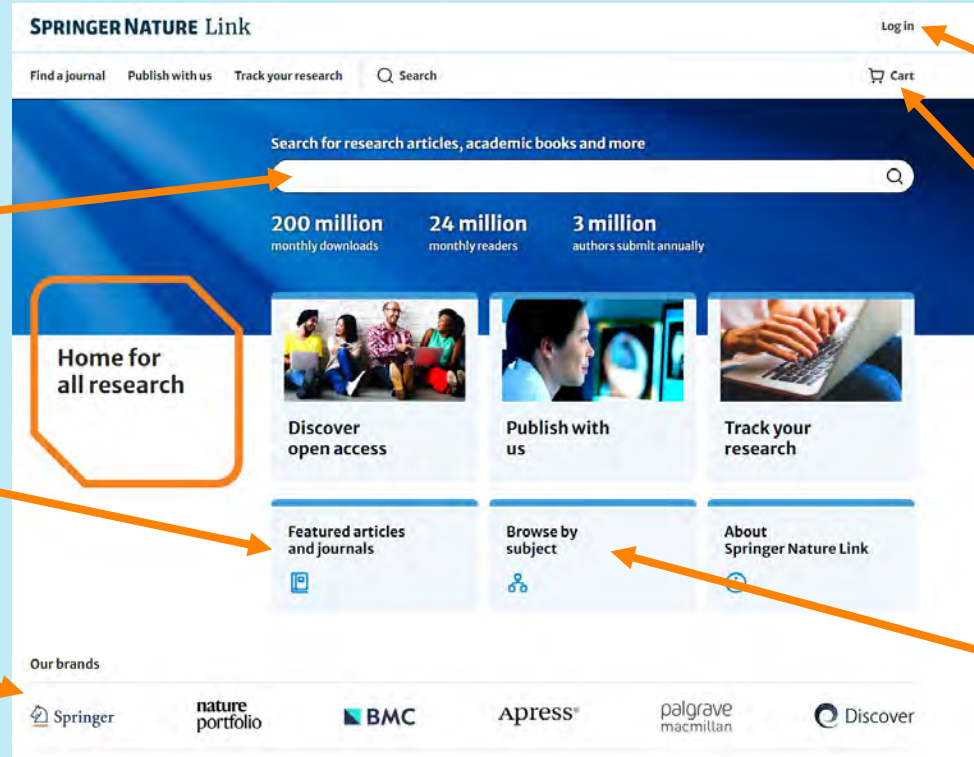
Visit

[link.springer.com](https://link.springer.com)

Responsive search  
function

Jump to highlighted  
content

Includes content from  
across our brands



Sign-in


Purchase  
content  
online

Browse  
by  
discipline

# LOGGING IN

Home for all research

3


 Enter your email address to update your account to the new Springer Nature login on Springerlink.


**Log in, or register a new account to continue**

Email address

Continue →

OR

 Continue with Google →

 Continue with ORCID →

Registered users can  
log-in directly

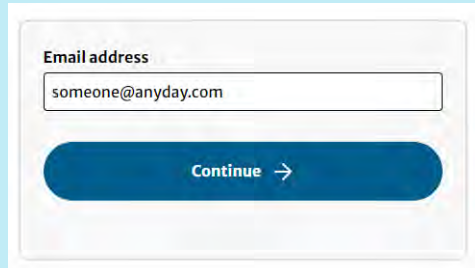
Or log-in using your  
Google account or  
ORCID

# CREATING AN ACCOUNT WITH EMAIL ADDRESS

For the first time

4

1) Enter email address on Log in page and press continue

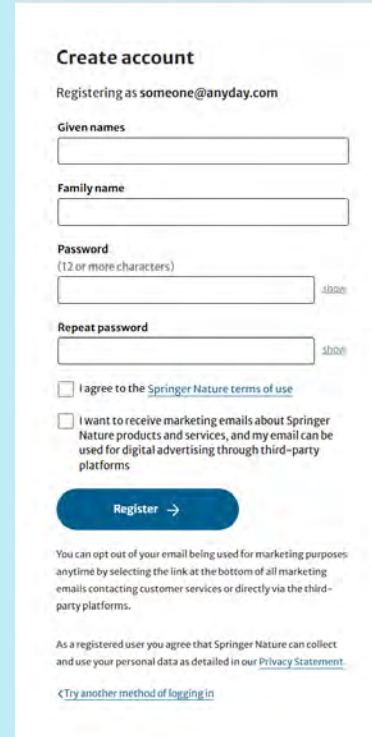


Email address

someone@anyday.com

Continue →

2) Fill in form and confirm terms of use. Press register.



**Create account**

Registering as someone@anyday.com

Given names

Family name

Password  
(12 or more characters)

Repeat password

☐ I agree to the [Springer Nature terms of use](#)

☐ I want to receive marketing emails about Springer Nature products and services, and my email can be used for digital advertising through third-party platforms

Register →

You can opt out of your email being used for marketing purposes anytime by selecting the link at the bottom of all marketing emails contacting customer services or directly via the third-party platforms.

As a registered user you agree that Springer Nature can collect and use your personal data as detailed in our [Privacy Statement](#).

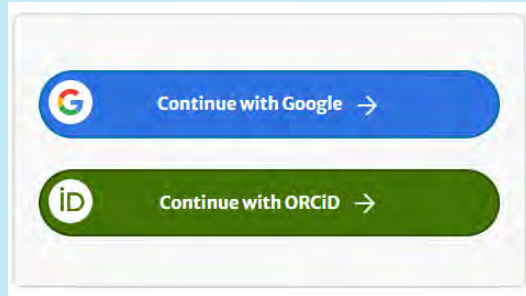
[Try another method of logging in](#)

# CREATING AN ACCOUNT WITH GOOGLE ACCOUNT

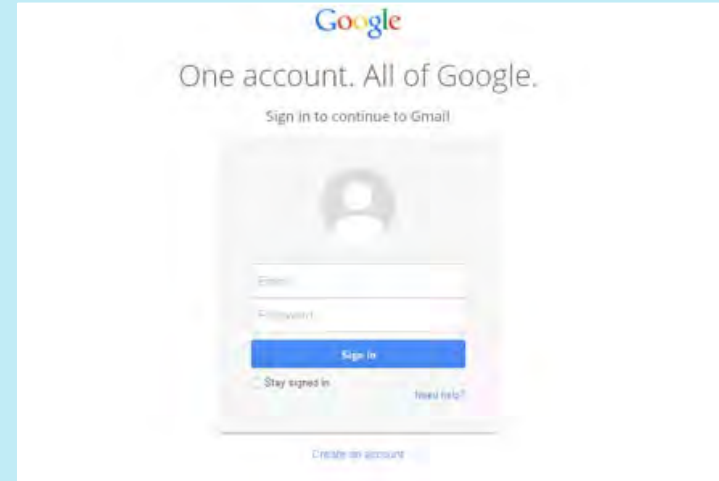
5

For the first time

1) Choose 'Continue with Google'



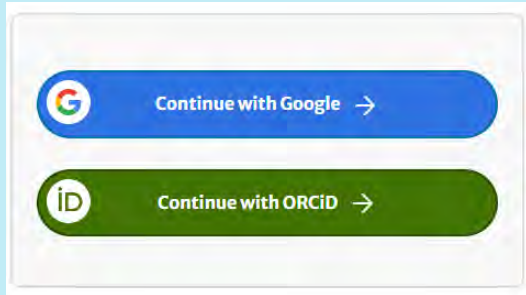
2) Sign in to Google using your email and password.



# CREATING AN ACCOUNT WITH ORCID ACCOUNT

For the first time

1) Choose 'Continue with ORCID'



2) Sign in to ORCID using your email/ID and password.

A screenshot of the ORCID 'Sign in to ORCID' form. At the top is the ORCID logo. Below it, the text 'Sign in to ORCID' is displayed, followed by a link: 'Don't have your ORCID ID yet? Register now'. The form contains two main input sections: 'Email or ORCID ID' with a text box and an example 'For example: joe@institution.edu or 0000-1234-5678-9101', and 'Password' with a text box labeled 'Your ORCID password'. A dark blue 'Sign in to ORCID' button is positioned below these fields. Underneath the button is a link: 'Forgot your password or ORCID ID?'. Further down, separated by an 'OR' divider, are two additional options: 'Sign in through your institution' (with a building icon) and 'Sign in with Google' (with the Google logo). An orange arrow points from a text box to the 'Sign in through your institution' option.

Or click on this option to authenticate yourself through your institution.

# SEARCHING FOR CONTENT

Finding what you need

7

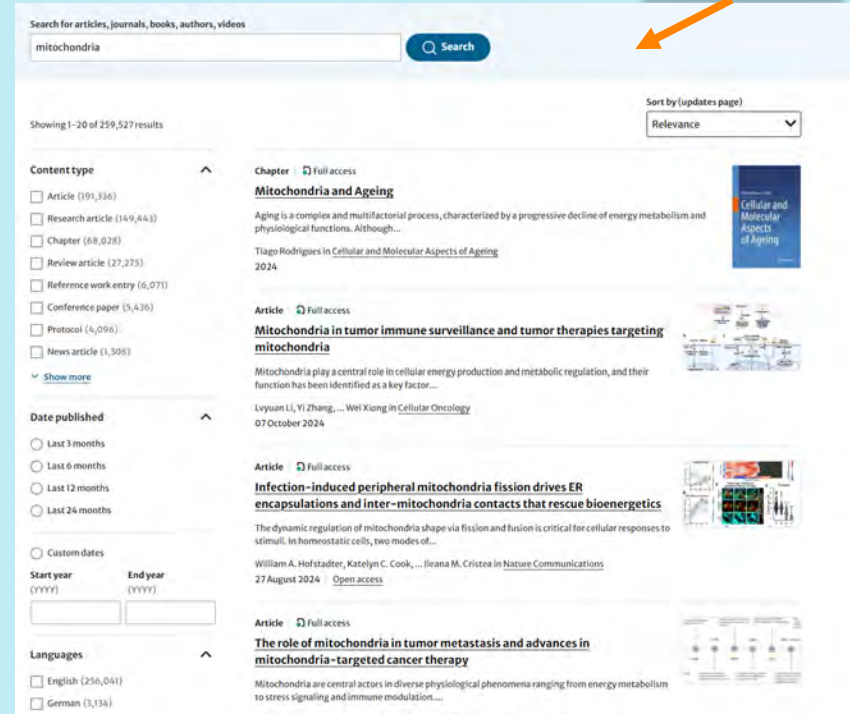
1) Enter key word into search bar



2) Refine search using left hand menu, filtering results by:

- Content type
- Date published
- Language
- Subject
- Disciplines
- Subdisciplines

Then click **Update Results**





# SEARCHING FOR CONTENT

Finding what you need

8

2) Review refined results

3) Sort by date

Full-access content doesn't require a subscription to read or download

The screenshot shows a search results page for the term 'mitochondria'. The search bar at the top contains 'mitochondria' and a 'Search' button. Below the search bar, there are filters for 'Research article', 'Last 3 months', 'English', and 'Life sciences'. The results are sorted by 'Relevance'. The first result is titled 'Stress triggers gut dysbiosis via CRH-CRH1-mitochondria pathway' and is marked as 'Full access'. The second result is titled 'Mitochondria transfer-based therapies reduce the morbidity and mortality of Leigh syndrome' and is also marked as 'Full access'. The third result is titled 'Mitochondria facilitate neuronal differentiation by metabolising nuclear-encoded RNA' and is marked as 'Full access'. The page also shows a 'Content type' filter with 'Research article' selected, and a 'Date published' filter with 'Last 3 months' selected. A 'Full access' label is highlighted with an orange box around the first result.



The screenshot shows the article page for "Stress triggers gut dysbiosis via CRH-CRHR1-mitochondria pathway" in the journal npi Biofilms and Microbiomes. The page includes a title, authors, abstract, and a diagram illustrating the CRH-CRHR1-mitochondria pathway. Annotations with orange arrows point to various elements: "Download full PDF article" points to the PDF download button; "Click to find out more about the author(s)" points to the author list; "Explore metrics to see how many times it has been accessed, cited and mentioned on social media" points to the "Explore all metrics" link; "Jump to citation details and download citation" points to the citation icon; "Review Figures and References" points to the "Figures" and "References" tabs; and "Jump to sections of interest" points to the "Sections" tab.

Find a journal Publish with us Track your research Search

Home > npi Biofilms and Microbiomes > Article

## Stress triggers gut dysbiosis via CRH-CRHR1-mitochondria pathway

Article | Open access | Published: 30 September 2024  
Volume 10, article number 93, (2024) | [View this article](#)

[Download PDF](#) [No, I don't have access to this open access article](#)

Yining Zhang, Xiaoyang Li, Song Li, Hualin Guo, Zhongyi Zhang, Haonan Zheng, Cuihong Zhang, Indong Zhang, Kun Wang, Fei Pei & Liping Quat

1536 Accesses 2 Altmetric [Explore all metrics](#)

### Abstract

Stress can lead to gut dysbiosis in brain-gut axis disordered diseases as irritable bowel syndrome (IBS), yet the mechanisms how stress transfer from the brain to the gut and disrupt gut microbes remain elusive. Here we describe a stress-responsive brain-to-gut axis which triggers colonocytes' mitochondria to trigger gut dysbiosis. Patients with IBS exhibit significantly increased facultative anaerobes and decreased obligate anaerobes, related to increased serum corticotropin-releasing hormone (CRH) level and detected colonocytes' mitochondria ultrastructure. Mice exposed to acute stress experienced enhanced CRH-CRH receptor type 1 (CRHR1) signaling, which impaired mitochondria and epithelium hypoxia in the colon, subsequently triggered gut dysbiosis. Antagonizing CRHR1 expression to inhibit cAMP/Pka/MAK signaling or activating mitochondria respiration conferred resilience against stress-induced mitochondria damaging and epithelium hypoxia impairment, ultimately improving gut dysbiosis. These results suggest that the CRH-CRHR1-mitochondria pathway plays a pivotal role in stress-induced gut dysbiosis that could be therapeutically targeted for stress-induced gastrointestinal diseases.

### Sections

- [Abstract](#)
- [Introduction](#)
- [Methods](#)
- [Results](#)
- [Discussion](#)
- [Data availability](#)
- [References](#)
- [Acknowledgements](#)
- [Author information](#)
- [Ethics declarations](#)
- [Additional information](#)
- [Supplementary information](#)
- [Rights and permissions](#)
- [About this article](#)

Advertisements

**EXPLORE SMOLDERING NEUROINFLAMMATION**  
[www.nature.com/npi](#)  
KONOFI

Download full PDF article

Click to find out more about the author(s)

Explore metrics to see how many times it has been accessed, cited and mentioned on social media

Jump to citation details and download citation

Review Figures and References

Jump to sections of interest

# HOW TO CITE AN ARTICLE

10

Find a journal Publish with us Track your research Search

Home > npj Biofilms and Microbiomes > Article

## Stress triggers gut dysbiosis via CRH-CRHR1-mitochondria pathway

Article | Open access | Published: 30 September 2024  
Volume 10, article number 93 (2024) | [Cite this article](#)

[Download PDF](#) | You have full access to this open access article

Yiming Zhang, Xiaoxiao Li, Sujia Lu, Huaili Guo, Zhuangyi Zhang, Haonan Zheng, Cunsheng Zhang, Lindong Zhang, Kun Wang, Fei Pei & Liping Qian

1154 Accesses | 2 Altmetrics | [Explore all metrics](#)

### Abstract

Stress can lead to gut dysbiosis in brain-gut axis disordered diseases as irritable bowel syndrome (IBS), yet the mechanisms how stress transfer from the brain to the gut and disrupt gut microbiota remain elusive. Here we describe a stress-responsive brain-to-gut axis which impairs colonocytes' mitochondria to trigger gut dysbiosis. Patients with IBS exhibit significantly increased facultative anaerobes and decreased obligate anaerobes, related to increased serum corticotropin-releasing hormone (CRH) level and defected colonocytes' mitochondria ultrastructure. Mice exposed to acute stress experienced enhanced CRH-CRH receptor type 1 (CRHR1) signaling, which impaired mitochondria and epithelium hypoxia in the colon, subsequently triggering gut dysbiosis. Antagonizing CRHR1 expression to inhibit cAMP/Pka/MAPK signaling or activating mitochondria respiration conferred resilience against stress-induced mitochondria damaging and epithelium hypoxia impairment, ultimately improving gut dysbiosis. These results suggest that the CRH-CRHR1-mitochondria pathway plays a pivotal role in stress-induced gut dysbiosis that could be therapeutically targeted for stress-induced gastrointestinal diseases.

**EXPLORE SMOLDERING NEUROINFLAMMATION**

[Advertisement](#)

1) Click “Cite this article”

2) Copy citation information or click “Download citation”



### Cite this article

Zhang, Y., Li, X., Lu, S. et al. Stress triggers gut dysbiosis via CRH-CRHR1-mitochondria pathway. *npj Biofilms Microbiomes* 10, 93 (2024). <https://doi.org/10.1038/s41522-024-00571-z>

[Download citation](#)

Received  
13 May 2024

Accepted  
16 September 2024

Published  
30 September 2024

DOI  
<https://doi.org/10.1038/s41522-024-00571-z>

[Data availability](#)

[References](#)

[Acknowledgements](#)

[Author information](#)

[Ethics declarations](#)

[Additional information](#)

[Supplementary information](#)

[Rights and permissions](#)

[About this article](#)

# REFERENCES

11

1) Click "References"

2) Explore list of literature the author used to write the article.

Most references are linked to their source.

Find a journal Publish with us Track your research Search

Home > *npj Biofilms and Microbiomes* > Article

## Stress triggers gut dysbiosis via CRH-CRHR1-mitochondria pathway

Article | Open access | Published: 30 September 2024  
Volume 10, article number 93, (2024) | [Cite this article](#)

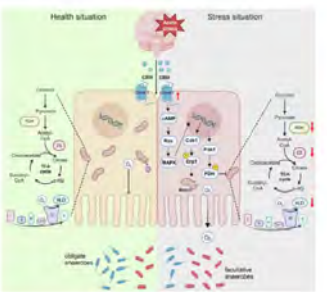
[Download PDF](#) | You have full access to this open access article

Yining Zhang, Xiaoxi Li, Siqi Lu, Huaili Guo, Zhuangyi Zhang, Haonan Zheng, Cunheng Zhang, Lindong Zhang, Kun Wang, Fei Pei & Liping Qian

1154 Accesses | 2 Altmetrics | [Explore all metrics](#)

### Abstract

Stress can lead to gut dysbiosis in brain-gut axis disordered diseases as irritable bowel syndrome (IBS), yet the mechanisms how stress transfer from the brain to the gut and disrupt gut microbiota remains elusive. Here we describe a stress-responsive brain-to-gut axis which impairs colonocytes' mitochondria to trigger gut dysbiosis. Patients with IBS exhibit significantly increased facultative anaerobes and decreased obligate anaerobes, related to increased serum corticotropin-releasing hormone (CRH) level and defected colonocytes' mitochondria ultrastructure. Mice exposed to acute stress experienced enhanced CRH-CRH receptor type 1 (CRHR1) signaling, which impaired mitochondria and epithelium hypoxia in the colon, subsequently triggered gut dysbiosis. Antagonizing CRHR1 expression to inhibit cAMP/Pka/MAPK signaling or activating mitochondria respiration conferred resilience against stress-induced mitochondria damaging and epithelium hypoxia impairment, ultimately improving gut dysbiosis. These results suggest that the CRH-CRHR1-mitochondria pathway plays a pivotal role in stress-induced gut dysbiosis that could be therapeutically targeted for stress-induced gastrointestinal diseases.



**EXPLORE SMOLDERING NEUROINFLAMMATION**

**sanofi**

Sections Figures **References**

1. Morais, L. H., Schreiber, H. L. T. & Mazmanian, S. K. The gut microbiota-brain axis in behaviour and brain disorders. *Nature reviews. Microbiology* **19**, 241–255, <https://doi.org/10.1038/s41579-020-00460-0> (2021).  
[Article](#) [CAS](#) [PubMed](#) [Google Scholar](#)
2. Camilleri, M. Diagnosis and Treatment of Irritable Bowel Syndrome: A Review. *JAMA* **325**, 865–877, <https://doi.org/10.1001/jama.2020.22532> (2021).  
[Article](#) [CAS](#) [PubMed](#) [Google Scholar](#)
3. Stewart Campbell, A. et al. Safety and target engagement of an oral small-molecule sequestrant in adolescents with autism spectrum disorder: an open-label phase 1b/2a trial. *Nat. Med* **28**, 528–534, <https://doi.org/10.1038/s41591-022-01683-9> (2022).  
[Article](#) [CAS](#) [PubMed](#) [Google Scholar](#)
4. Dalile, B., Van Oudenhove, L., Vervliet, B. & Verbeke, K. The role of short-chain fatty acids in microbiota-gut-brain communication. *Nat. Rev. Gastroenterol. Hepatol.* **16**, 461–478, <https://doi.org/10.1038/s41575-019-0157-3> (2019).  
[Article](#) [PubMed](#) [Google Scholar](#)

# SPRINGER NATURE ACCESSIBILITY STATEMENT

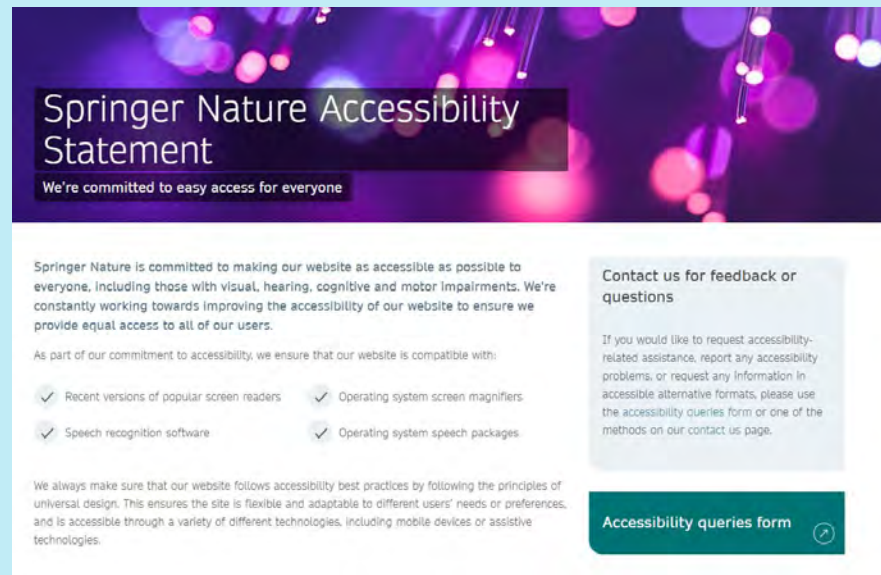
12

We're committed to easy access for everyone

We ensure that our website is compatible with:

- Recent versions of popular screen readers
- Operations systems screen magnifiers
- Speech recognition software
- Operation system speech packages

We welcome your feedback or questions, please use the [Accessibility queries form](#) to raise any accessibility concerns.



**Springer Nature Accessibility Statement**  
We're committed to easy access for everyone

Springer Nature is committed to making our website as accessible as possible to everyone, including those with visual, hearing, cognitive and motor impairments. We're constantly working towards improving the accessibility of our website to ensure we provide equal access to all of our users.

As part of our commitment to accessibility, we ensure that our website is compatible with:

- ✓ Recent versions of popular screen readers
- ✓ Operating system screen magnifiers
- ✓ Speech recognition software
- ✓ Operating system speech packages

We always make sure that our website follows accessibility best practices by following the principles of universal design. This ensures the site is flexible and adaptable to different users' needs or preferences, and is accessible through a variety of different technologies, including mobile devices or assistive technologies.

**Contact us for feedback or questions**

If you would like to request accessibility-related assistance, report any accessibility problems, or request any information in accessible alternative formats, please use the accessibility queries form or one of the methods on our contact us page.

[Accessibility queries form](#)

To find out more about our approach to accessibility visit [springernature.com/gp/info/accessibility](https://springernature.com/gp/info/accessibility)