

# SPRINGER NATURE Link

Quick User Guide

A hand is shown pointing towards a vibrant, glowing digital structure. The structure consists of numerous overlapping, colorful lines (blue, purple, orange, yellow) that form a complex, swirling pattern, reminiscent of a galaxy or a data visualization. The background is dark with scattered, colorful bokeh lights. The overall aesthetic is futuristic and high-tech.

**SPRINGER NATURE**

# INTRODUCING SPRINGER NATURE LINK

Home for all research

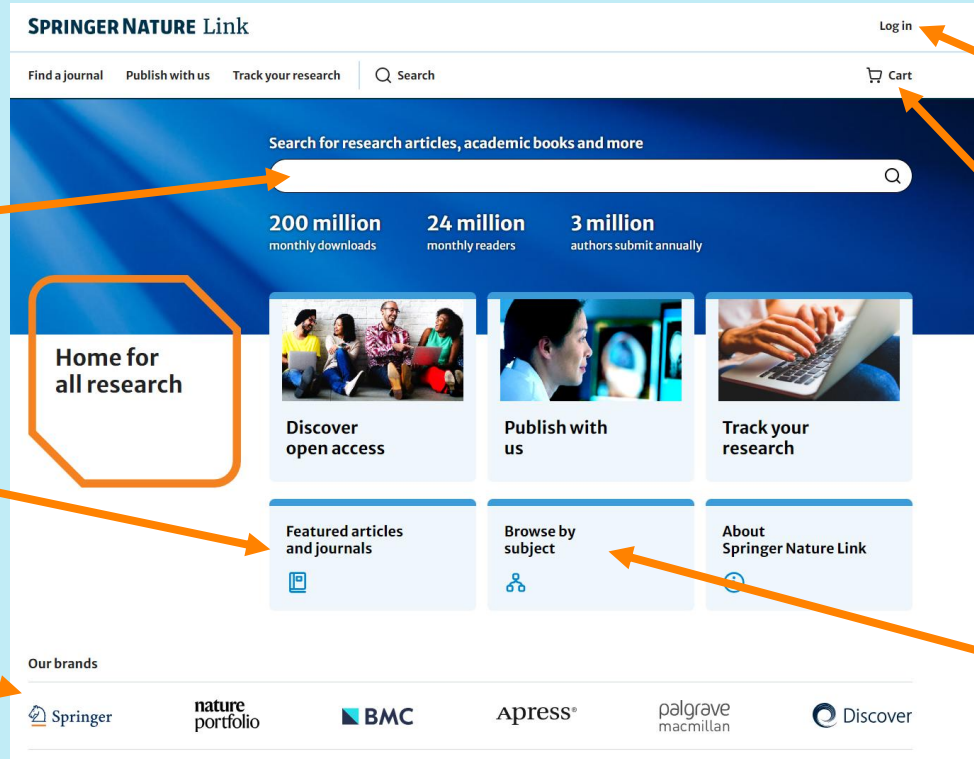
Visit

[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


 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 →](#)

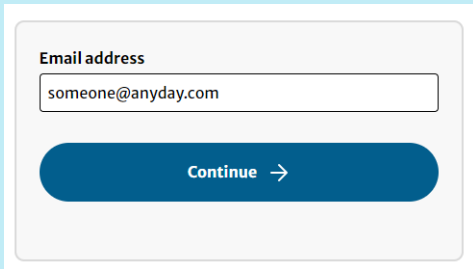
Registered users can log-in directly

Or log-in using your Google account or ORCID

# CREATING AN ACCOUNT WITH EMAIL ADDRESS

For the first time

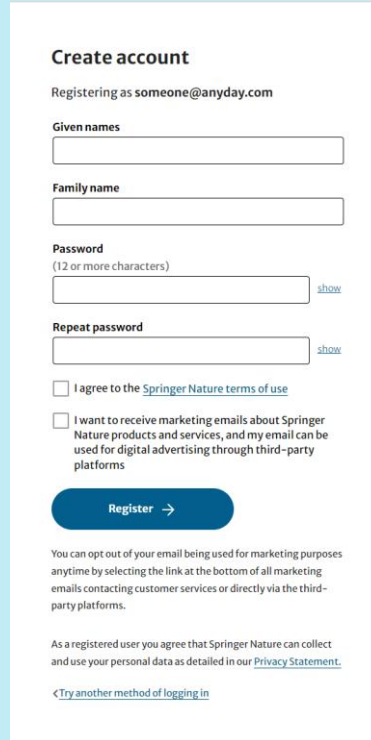
1) Enter email address on Log in page and press continue



Email address

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)

[show](#)

Repeat password

[show](#)

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](#)

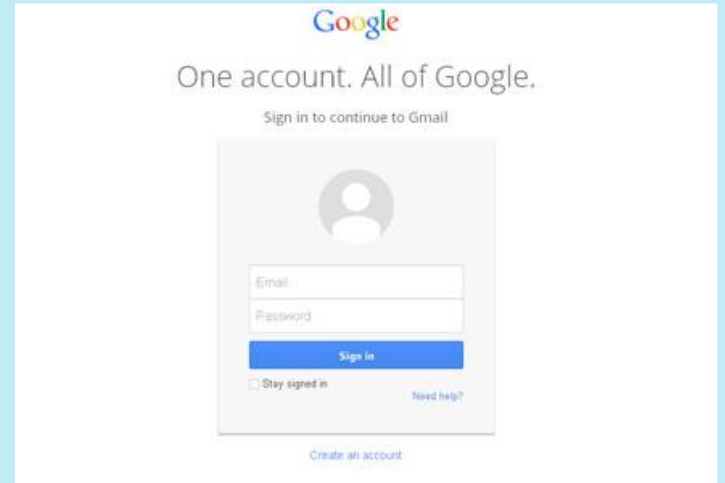
# CREATING AN ACCOUNT WITH GOOGLE ACCOUNT

For the first time

1) Choose 'Continue with Google'



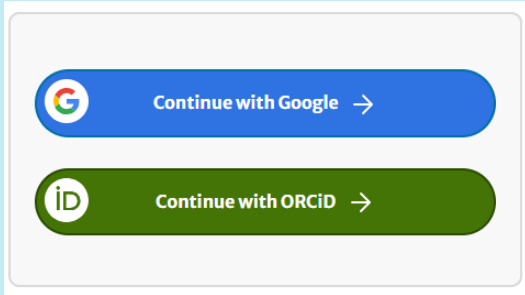
2) Sign in to Google using your email and password.



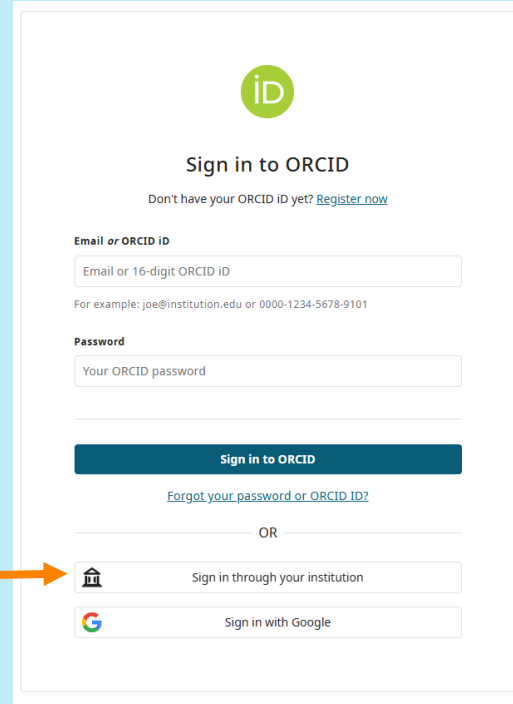
# CREATING AN ACCOUNT WITH ORCID ACCOUNT

For the first time

1) Choose 'Continue with ORCID'



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



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



# SEARCHING FOR CONTENT

## Finding what you need

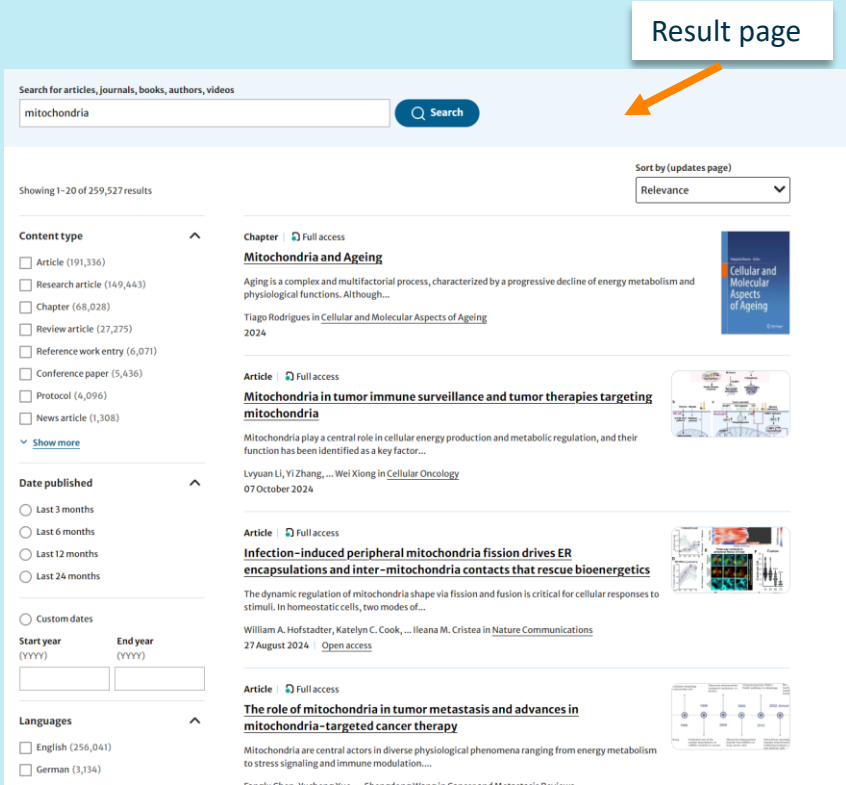
1) Enter key word into search bar



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

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

Then click **Update Results**



Result page



# SEARCHING FOR CONTENT

Finding what you need

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 an article titled "Stress triggers gut dysbiosis via CRH-CRH1-mitochondria pathway" published on 30 September 2024. The second result is "Mitochondria transfer-based therapies reduce the morbidity and mortality of Leigh syndrome" published on 02 September 2024. The third result is "Mitochondria facilitate neuronal differentiation by metabolising nuclear-encoded RNA" published on 26 September 2024. The page also shows a sidebar with filters for "Content type", "Date published", "Start year", "End year", and "Subjects".

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](#) or you have full access to this open access article

Yiming Zhang, Xiaogang Li, Siqi Liu, Hualin Guo, Zhuangyi Zhang, Haonan Zheng, Cunheng Zhang, Jindong Zhang, Kun Wang, Fei Pei & Liping Du (✉)

1156 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 microbiota remain elusive. Here we describe a stress-responsive brain-to-gut axis which involves 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/Ras/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.

Health situation Stress situation

EXPLORING SMOLDERING NEUROINFLAMMATION  
www.nature.com/scientificreports/

konofi

Download full PDF article

Jump to citation details and download citation

Review Figures and References

Click to find out more about the author(s)

Jump to sections of interest

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

# HOW TO CITE AN ARTICLE

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, Xiaoyang Li, Siqi Lu, Hualizhu Guo, Zhuangqi Zhang, Haonan Zheng, Cunzhen Zhang, Jindong Zhang, Kun Wang, Fei Pei & Liping Duan

1156 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 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 triggered gut dysbiosis. Antagonizing CRHR1 expression to inhibit cAMP/Ras/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.

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    | Accepted          | Published         |
| 13 May 2024 | 16 September 2024 | 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](#)

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) | [View this article](#)

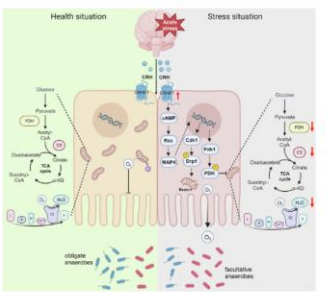
[Download PDF](#) | You have full access to this open access article

Yiming Zhang, Xiaoyang Li, Siqi Lu, Huaili Zhu Guo, Zhuangqi Zhang, Haonan Zheng, Cunzhen Zhang, Jindong Zhang, Kun Wang, Fei Pei & Liping Duan


1156 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 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 triggered gut dysbiosis. Antagonizing CRHR1 expression to inhibit cAMP/Ras/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.



Advertisement



1) Click "References"

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

Most references are linked to their source.

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

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)