



2026 USASP Annual Scientific Meeting

March 23-26, 2026 | Loews Philadelphia Hotel

Call for Abstracts

- Length of abstract (body of abstract): 250 words max
- Abstracts must be submitted using the designated link (see below)
- Each abstract submitted must have a unique corresponding/presenting author
- The first author will be the main contact for the submission.
- Abstracts will be published in the Journal of Pain.
- Members and non-members are eligible to submit abstracts.
- Abstracts of unpublished work will be accepted if the abstract is **not** currently in Press (i.e., at the time of abstract submission)

Please organize the body of the abstract as follows:

1. A statement of the purpose of the study.
2. An outline of the methods used.
3. A summary of the results is presented in sufficient detail to support the conclusions.
4. A statement of the conclusions reached. It is not satisfactory to state that "the results will be discussed."

NOTE: Abstracts submitted that do not follow these formatting guidelines will be rejected and sent back to the main contact.

Formatting (numbers correspond to the example below):

1. Only the first author's affiliations should be included at the end of the author list due to space limitations for publication in the Journal of Pain.
2. The abstract should not have subheadings (such as introduction, results, methods, etc.) and should be one fluid paragraph.
3. No figures or tables are permitted.
4. If possible, please avoid using special characters in the text.

5. Do not include numbered references/bibliography. Authors who wish to cite specific research may do so parenthetically such as, (Palermo, 2019).

6. Funded sources may be included at the end of the abstract. Example – Funding: RO1AGO4891.

You will be prompted to insert the text of the abstract in the submission portal as well as to upload a Word document of your abstract.

EXAMPLE:

Sensory Neuron-Derived Ccl2 Corresponds To Inflammation In Ulcerative Colitis

Aaditya Adlakha, Elizabeth M. Howerton, Namrata GR Raut and Michael P. Jankowski; Cincinnati Children's Hospital

~~Introduction~~

Ulcerative colitis (UC), characterized by chronic inflammation of colonic mucosa and abdominal pain is associated with inappropriate migration or activation of macrophages. CCL2 plays a significant role in regulating macrophage recruitment and mediating abdominal pain (Author, XXXX). Our RNA sequencing data implicate that sensory neurons express CCL2. We, therefore, hypothesize that sensory neuron-derived CCL2 correlated with inflammation through recruitment and maintenance of macrophages, in a mouse model of UC (oral 4% dextran sodium sulfate (DSS)).

~~Methods and Results~~

The degree of colonic injury was confirmed by hematoxylin and eosin staining and inflammation was determined by immunohistochemical staining of colons harvested from sensory neuron reporter (PirtCre;tdTomato) mice for macrophages 2-7 days post-DSS. At day 2, DSS mice showed minimal colonic injury but a significant surge in the number of macrophages in the colonic mucosa compared to the controls. Increases in macrophages were observed in the vicinity of neurons innervating the mucosa, however, innervation density in the mucosal layer was decreased. A significant increase in pain-like behavior was also observed, but only at later time points (day 4-7) as assessed using abdominal von frey test and mouse grimacing scale.

~~Conclusion~~

The initiation of inflammation and the increase in pain-like behavior on day 2 correlated with an upregulation of CCL2 mRNA in neurons of the lumbosacral ganglia. These results suggest that sensory neuron-derived CCL2 could be involved in colonic macrophage recruitment and subsequent pain in UC, and may have implications for future development of pain and anti-inflammatory therapeutics. NIH (R01NS113965 and R01NS105715) and University Research Council Fellowship from the University of Cincinnati.

