ACS Chemical Neuroscience

Editorial

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Neuroinflammation: Brain on Fire?

If you ask our guest editor, Dr. Richard Ransohoff, for a definition of neuroinflammation—the topic of this *ACS Chemical Neuroscience* Special Issue—he may very well start by pointing to the greatest source of all information, Wikipedia. "Neuroinflammation is inflammation of the nervous tissue." He will then point out that Wikipedia also defines that "a circular definition is one that uses the term being defined as a part of the definition." Of course, Dr. Ransohoff uses this somewhat tongue-in-cheek remark to make a very clear and important point. The concept of neuroinflammation is quite poorly defined. While the word neuroinflammation conjures a picture of a "brain on fire" represented on the cover of our issue, it is clear that neuroinflammation does not necessarily conform to the hallmarks of acute inflammation—pain, heat, redness, swelling, and loss of function. Dr. Ransohoff instead defines neuroinflammation as "non-cell-autonomous processes which dictate CNS cell death, dysfunction, or recovery for neurons and oligodendrocytes during the course of neurodegenerative disease." This agnostic view recognizes that neuroinflammation encompasses "good and bad" actions. We used this definition of neuroinflammation to guide our solicitation of manuscripts for the Special Issue, although we certainly did not limit contributions to only those that explicitly comply. We tried where possible to challenge authors to help define "neuroinflammation" in their contributions in order to provide readers with an opportunity to formulate an independent definition.

Contributions to the Special Issue on neuroinflammation span a great number of disciplines including neurobiology, translational neuroimaging and neuroimmunology. You will note a collection of articles related to microglial cells, their functions and some of the protein targets that might provide therapeutics opportunities. While these are not the only cells that contribute to the process of "neuroinflammation", they are arguably the most important, perhaps the most poorly understood; fortunately, the field is on the cusp of major advances in knowledge. We were fortunate to receive contributions from academia and the pharmaceutical industry, which we hope provides some level of insight into how each currently views neuroinflammation. We are excited by the opportunity to reach additional researchers studying neuroinflammation—however you define it—and hope that *ACS Chemical Neuroscience* is recognized as an appropriate journal home to communicate your science.

As a community of researchers studying "neuroinflammation", we eventually need to reach a consensus definition to avoid misconception and misinformation. We hope that, in some small way, this *ACS Chemical Neuroscience* Special Issue can contribute to the dialogue that leads us there.

Jacob M. Hooker, Associate Editor

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Notes

Views expressed in this editorial are those of the author and not necessarily the views of the ACS.

Figure 1. Neuroinflammation author map created using VOSviewer (www.vosviewer.com) and a PubMed search for "neuroinflammation". *ACS Chemical Neuroscience* guest editor, Richard Ransohoff, is a large star and gravitational force in a dense constellation of researchers.

Special Issue: Neuroinflammation

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