

## How to Write a Basic Research Abstract for the SfN Annual Meeting

### **What Is an Abstract?**

An abstract is a short summary of your study. It is a highly-structured writing exercise. Like a paper, it should contain an introduction, methods, results, and conclusions (although these actual headings are not required). The abstract should be written as a single paragraph.

Abstracts have a proscribed length—for the SfN annual meeting, the body of the abstract should be no more than 2,300 characters, including punctuation but not spaces. This makes them deceptively difficult to write, because they need to convey a lot of information in a very small space. If done well, it makes the reader want to learn more about your research. Two example abstracts that incorporate all the key components and meet all the criteria can be found below.

Review the SfN Rules for Abstract Submission before you begin writing to ensure you have a good understanding of what's expected. Any questions? Reach out to [program@sfn.org](mailto:program@sfn.org).

### **Components of an Abstract**

These are the basic components of an abstract in any discipline:

1. *Motivation/problem statement*: What are you studying? Why do we care about the problem? What practical, scientific gap is your research filling?
2. *Methods/approach*: What did you actually do to get your results? Examples include the species, age, and sex of experimental subjects and whether sex differences were assessed. Summarize efforts to ensure scientific rigor, including sample sizes and replication, blinding, and which controls were used. There is no requirement to include full experimental protocols, but sufficient information must be given to indicate how the experiments were performed. Review the SfN Rules for Abstract Submission to familiarize yourself with the specific details required by the Society on efforts to promote transparency & scientific rigor, animal, and human subjects research.
3. *Results*: As a result of completing the above procedure, what did you learn? State a clear description of the outcome measures to support any conclusion you wish to make. If numerical data are presented as mean values, the standard deviations or standard errors should be given; the form used, and the  $n$  values should be stated. When statistical significance is shown, name the statistical test. Data may be conveyed by a combination of Methods and Results, i.e. an outline of the technique followed by the data obtained. Specific details about procedure and results are omitted unless they are very important.
4. *Conclusion/implications*: State the conclusion(s) supported by the results above, without including any metrics (numbers should be restricted to the sentences described above). What are the larger implications of your findings, especially for the problem/gap identified in step 1? Include consequences/impact of the research in the field.
5. *Other components*: Abstracts might also feature tables, figures, abbreviations, and references, but keep in mind that these count toward your 2,300 characters and are not required.

Use standard abbreviations for units of measure. Other abbreviations or acronyms should be fully spelled out on first mention, followed by the abbreviation/acronym in parentheses.

### **The Writing Process**

It helps, as you write your abstract, to write it methodically, step by step, to make sure that it is complete. At this stage, don't worry too much about any length requirements for the abstract.

After the first draft of the abstract is written, check to see if it fits within any length restrictions you have been given. If it is too long (which is usually the case at this stage of writing), look it over to see where it could be made more concise. For each word or phrase, ask yourself "Is this really necessary? Is there a simpler way I can convey the same meaning?" Don't use three words where you can communicate the same idea in one. Remove redundancies and unnecessary details, and substitute concise phrases for wordy passages. Keep editing your abstract until it falls within the length guidelines you have been given.

Have someone else look over your abstract before you are done. Ask a colleague or supervisor to read the abstract and offer criticism. They can often help pinpoint text that is confusing, wordy or redundant. Ensure that all authors have read and approved the abstract before you submit.

Finally, make sure to spell check and proofread carefully. A sloppy abstract leaves the reader with the impression that your research might also be sloppy!

### **Sample SfN Abstracts**

#### **Developmental features of primary sensory cortex and subcortical areas in the prosimian galago (*Otolemur garnettii*)**

##### **Authors**

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##### **Disclosures**

**E.C. Turner:** None. **J.H. Kaas:** None.

##### **Abstract**

The primate cerebral cortex undergoes striking changes during its development, but much is still unknown regarding how the primary sensory systems develop to reach their mature organization. We used a combination of immunohistochemical markers in the prosimian galago (*Otolemur garnettii*, n=8), aged P0 to P72, to follow the cortical and subcortical development of areas connected to primary sensory cortex (visual, somatosensory, and auditory). In addition to standard Nissl and cytochrome oxidase staining, we used vesicular glutamate transporter 2 (VGluT2) protein, which is primarily expressed in glutamatergic feedforward thalamocortical connections, and neuronal nuclei (NeuN) protein, which identifies all neurons, to characterize architectonic features. We also used calcium-binding proteins (CBPs) parvalbumin (PV) and calbindin (CB), as while the function of CBPs remains debated, they are known to be observed in well-

defined subpopulations of neurons. We find that all sensory thalamic nuclei, including the lateral geniculate nucleus (LGN), medial geniculate nucleus (MGN), and ventroposterior nucleus (VPN), show distinct subdivisions, related to the representation of their respective sensory inputs, that differ from expressions in the adult galago. For example, there is evidence for developmental changes in the connections of the magnocellular (M), parvocellular (P), and koniocellular (K) pathways to visual cortex; the P layers of the LGN are the only layers to express PV at P0, in contrast to full expression throughout the LGN layers in the adult. Similarly, VGLUT2 expression at P0 in the LGN is evenly distributed across the M, P, and K layers, in contrast to the adult galago which has the strongest reactivity in the M layers, followed by the P layers, and most weakly in the K layers. In all primary sensory areas, layer IV is easily identifiable with PV reactivity, in contrast to the surrounding non-primary areas which show minimal reactivity across cortex. One exception to this is middle temporal visual area (MT), which contains strong neuropil and cell immunoreactivity for PV in layer IV at P0. These results, similar to those reported for MT in marmosets (Warner et al., 2012), suggest that area MT may serve as a primary-like area early during development. These architectonic maps of galago cortex can reveal more about the hierarchical developmental of cortical areas and the functional roles of these areas in early postnatal development.

## **PV-expressing cells in the mouse spinal dorsal horn gate the transmission of innocuous tactile input to lamina I**

### **Authors**

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### **Disclosures**

**A. Dickie:** None. **K.A. Boyle:** None. **T. Yasaka:** None. **V.E. Abraira:** None. **A.L. Zimmerman:**None. **D.D. Ginty:** None. **M.A. Gradwell:** None. **R.J. Callister:** None. **B.A. Graham:** None. **D.I. Hughes:** None.

### **Abstract**

Chronic pain presents a major unmet clinical problem. One feature of chronic pain is the development of allodynia, where previously innocuous tactile stimuli are perceived as painful. We have shown that inhibitory spinal interneurons which express parvalbumin (PV) form axo-axonic synapses on to the central terminals of myelinated afferents, and have proposed that a loss of the inhibition they mediate could contribute to the development of allodynia following peripheral nerve injury. In this study, we aim to determine the synaptic relationship between low threshold mechanoreceptive (LTMR) afferent input and PV-cell mediated inhibition. We have used *in vitro* targeted recordings in spinal cord slices from PV<sup>Cre</sup>;Ai9 mice to show that stimulation of dorsal roots at A $\delta$  strength elicits monosynaptic EPSCs in PV-expressing cells. We have used tissue from Split<sup>Cre</sup>;Ai34 and TrkB<sup>CreER</sup>;Ai35 mice to show that approximately 30% (mean 28.5% SD 14.9) and 35% (35.2%  $\pm$  6.3) of VGLUT1 inputs on to inhibitory PV neurons are derived from A $\beta$  and A $\delta$  hair afferents, respectively, and also find that central terminals of most A $\beta$  and A $\delta$  hair afferents receive contacts from inhibitory boutons that express PV (70.7%  $\pm$  8.1, and 80.1%  $\pm$  3.8, respectively). We also show that 27.9% ( $\pm$  2.4) of VGAT boutons in contact with the dendrites of vertical cells in lamina II and III are derived from

PV-expressing cells, and that 61.9% ( $\pm$  17.2) of the VGLUT1 terminals that target these dendrites associate directly with PV/VGAT boutons. To determine whether PV cells mediate presynaptic inhibition of LTMRs and postsynaptic inhibition of vertical cells, we have also carried out *in vitro* optogenetic experiments in spinal cord slices from PV<sup>Cre</sup>;Ai32 mice. We find evidence of light-induced bicuculline-sensitive polysynaptic EPSCs in vertical cells, indicative of primary afferent depolarisation mediated by PV cells. We also find evidence of monosynaptic IPSCs that are sensitive to both bicuculline and strychnine, indicative of PV-cell mediated postsynaptic inhibition. Our findings provide anatomical and functional evidence that PV cells mediate both presynaptic inhibition of myelinated afferents that synapse on to vertical cells, and postsynaptic inhibition of the vertical cells themselves. We propose that decreased PV cell-mediated inhibition unmasks a circuit involving vertical cells. This enables LTMR input from lamina III and III to activate lamina I pain circuits, and could result in allodynia. Together, these findings identify PV interneurons as a target for therapeutic intervention to alleviate allodynic conditions.

*Adapted from the Abstract Submission Instructions of The Physiological Society*