2nd BEB/Bluepharma Award Guidelines

The **Title** should capture the conceptual significance for a broad audience. As a general guideline, the most effective titles are no more than 10–12 words and should readily give readers an overall view of the paper's significance rather than the detailed contents of the paper, which can be elaborated upon in the Summary. Titles should also avoid use of jargon, uncommon abbreviations, and punctuation. They should be no more than a total of 85 characters, including spaces (Font: Arial,12).

A **graphical abstract** should allow readers to quickly gain an understanding of the main take-home message of the paper and is intended to encourage browsing, promote interdisciplinary scholarship, and help readers identify more quickly which papers are most relevant to their research interests.

Technical requirements are as follows:

- Size: 5.5 inches square at 300 dpi.
- Font: Arial, 12–16 points. Smaller fonts will not be legible online.
- Content: the abstract should consist of one single panel.

Examples of this feature can be seen in the online version of articles published in Cell from January 2010 onwards.

The **"In Brief"** is a short summary of the main take-home message of the paper and should describe the context and significance of the findings for the broader readership.

Specifications: This blurb should be 80 words or fewer (Font: Arial, 12).

Highlights are a short collection of bullet points that convey the core findings of the article.
Specifications: up to four bullet points can be included; the length of an individual bullet point should not exceed 85 characters (including spaces); only the core results of the paper should be covered.

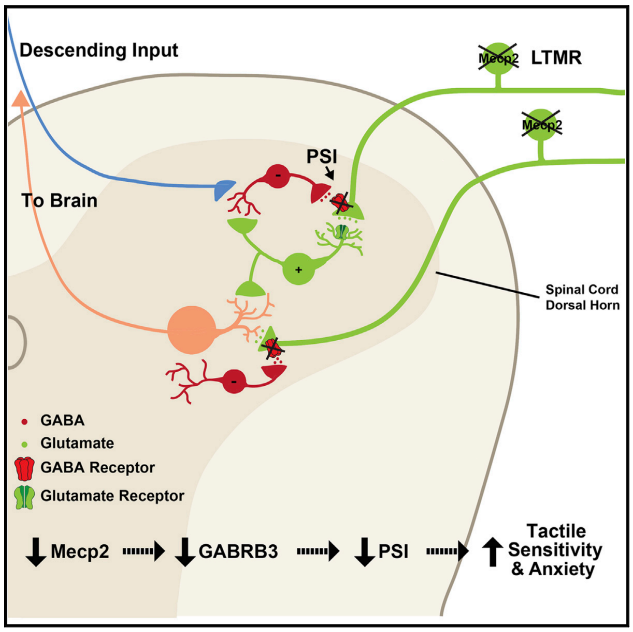
The **Summary** consists of a single paragraph of fewer than 150 words (Font: Arial, 12). We recommend that effective abstracts include the following elements: (1) a brief background of the question, while avoiding common yet information-poor clauses stating that a certain process has not been well understood; (2) a description of the results and approaches/model systems framed in the context of their conceptual interest; and (3) an indication of the broader significance of the work. As the adjective "novel" tends to be overused and rarely adds much meaning to a sentence, we generally try to avoid its use. The same applies to priority claims such as "the first" that can also be difficult to verify exhaustively. The description and interpretation of findings should be able to convey the study's interest and importance. References should not be cited in the Summary.

“What motivates me?” For this open question we challenge you to write about your background and what motivates, excites and seeds your curiosity in science and scientific research. In not more than 150 words (Font: Arial, 12), you should get your evaluator engaged and see your potential as a prospective researcher.

Example

Peripheral Mechanosensory Neuron Dysfunction Underlies Tactile and Behavioral Deficits in Mouse Models of ASDs

Graphical Abstract



In Brief

Changes in the brain are thought to underlie behaviors associated with autism, but now evidence from mouse models indicates that deficits in peripheral sensory neurons can contribute to the syndrome.

Highlights

- Several ASD mouse models exhibit aberrant tactile sensitivity
- *Mecp2* and *Gabrb3* function in somatosensory neurons for normal tactile behaviors
- *Mecp2* and *Gabrb3* function in somatosensory neurons to control presynaptic inhibition
- Developmental tactile abnormalities contribute to behaviorial deficits in adult mice

Summary: Patients with autism spectrum disorders (ASDs) commonly experience aberrant tactile sensitivity, yet the neural alterations underlying somatosensory dysfunction and the extent to which tactile deficits contribute to ASD characteristics are unknown. We report that mice harboring mutations in Mecp2, Gabrb3, Shank3, and Fmr1 genes associated with ASDs in humans exhibit altered tactile discrimination and hypersensitivity to gentle touch. Deletion of Mecp2 or Gabrb3 in peripheral somatosensory neurons causes mechanosensory dysfunction through loss of GABAA receptor-mediated presynaptic inhibition of inputs to the CNS. Remarkably, tactile defects resulting from Mecp2 or Gabrb3 deletion in somatosensory neurons during development, but not in adulthood, cause social interaction deficits and anxiety-like behavior. Restoring Mecp2 expression exclusively in the somatosensory neurons of Mecp2-null mice rescues tactile sensitivity, anxiety-like behavior, and social interaction deficits, but not lethality, memory, or motor deficits. Thus, mechanosensory processing defects contribute to anxiety-like behavior and social interaction deficits in ASD mouse models. (≤ 150 words)

What motivates me? (≤ 150 words)