JNC
The Official Journal of the International Society for Neurochemistry

# Journal of Neurochemistry

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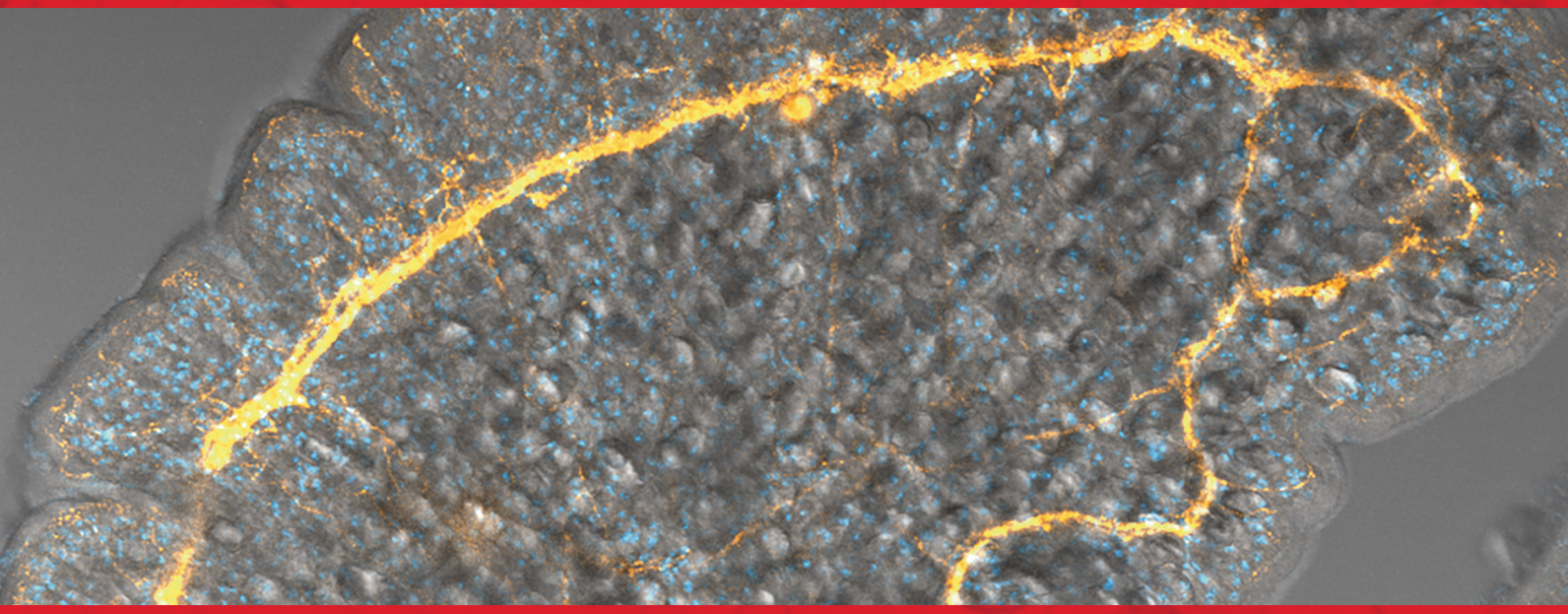

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**Aims & Scope**

*Journal of Neurochemistry* focuses on molecular, cellular and biochemical aspects of the nervous system, the pathogenesis of neurological disorders and the development of disease specific biomarkers. It is devoted to the prompt publication of original findings of the highest scientific priority and value that provide novel mechanistic insights, represent a clear advance over previous studies and have the potential to generate exciting future research.

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**Front cover**

Many anthelmintics target the neuromuscular system, in particular by interfering with signalling mediated by classical neurotransmitters. In this work, we have characterised the neuropeptide complement of the model cestode Hymenolepis microstoma. Analysis of gene expression of ten npp genes by in situ hybridization confirmed that all of them are expressed in the nervous system and identified cryptic features, including the first evidence of dorsoventral asymmetry, as well as a new population of peripheral peptidergic cells that appears to be conserved in the trematode Schistosoma mansoni. Finally, we characterised in greater detail Attachin, an SIFamide homolog. Although its expression is largely restricted to the longitudinal nerve cords and cerebral commissure in H. microstoma, it shows widespread localization in the larval nervous system of Echinococcus multilocularis and Mesocestoides corti. Altogether, this work provides a robust experimental foothold for the characterization of peptidergic signalling in parasitic flatworms.

**Image content**

Attachin immunoreactivity in Mesocestoides larvae.

**Read the full article** '*Global analysis of neuropeptides in cestodes identifies Attachin, a SIFamide homolog, as a stimulant of parasite motility and attachment*' by M. Preza, S. Van Bael, L. Temmerman, I. Guarnaschelli, E. Castillo and U. Koziol (J. Neurochem. 2022, vol. 162 (6), pp. 467–482) on doi:10.1111/jnc.15654

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# In this issue

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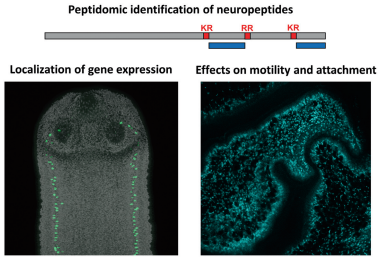
## Global analysis of neuropeptides in cestodes identifies Attachin, a SIFamide homolog, as a stimulant of parasite motility and attachment

*M. Preza, S. Van Bael, L. Temmerman, I. Guarnaschelli, E. Castillo and U. Koziol*

Many anthelmintics target the neuromuscular system of parasites. Although peptidergic signaling has been proposed as a target of novel anthelmintics, our knowledge of the neuropeptides of many helminths is still limited. In this work, we have characterized the neuropeptide complement of the cestode *Hymenolepis microstoma* by peptidomics. Analysis of gene expression of neuropeptide precursors confirmed their expression in the nervous system and identified novel populations of nerve cells. We characterized a peptide homologous to SIFamide, which stimulated motility and attachment (an activity essential for parasite establishment and survival) in the larvae of the related cestode *Mesocestoides corti*.

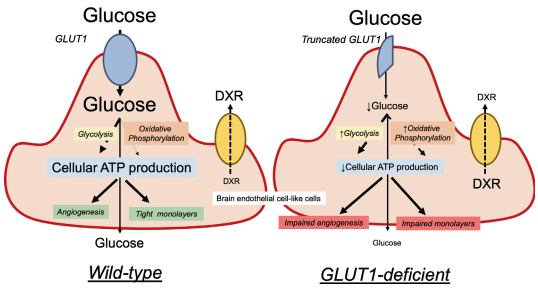



Read the full article on page 467.

## An in vitro model of glucose transporter 1 deficiency syndrome at the blood–brain barrier using induced pluripotent stem cells

*P. Iqra, Z. F. Tuz, M. Constantinos Marios and A-A. Jacob*

Glucose transporter 1 deficiency syndrome (GLUT1DS) is a neurological disorder characterized by mutations in the SLC2A1 gene, resulting in early onset of epilepsies, intellectual deficit, and movement disorders. Yet, the impact of such mutations on brain endothelial cells (BMECs) function remains unclear. In this study, we developed an in vitro model of GLUT1DS using human-induced pluripotent stem cells (iPSCs) differentiated into BMECs. These GLUT1-BMECs showed impaired glucose uptake, altered glucose metabolism, compromised barrier function, and impaired angiogenesis. In conclusion, this model provides a potent tool to better understand the impact of GLUT1DS on glucose metabolism at the neurovascular unit.

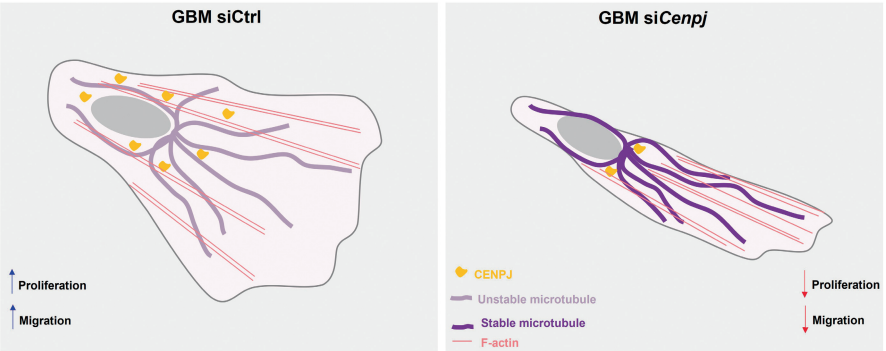



Read the full article on page 483.

## Centromere protein J is overexpressed in human glioblastoma and promotes cell proliferation and migration

*G. P. A. de Freitas, L. H. M. Geraldo, B. M. Faria, S. V. Alves-Leon, J. M. de Souza, V. Moura-Neto, B. Pontes, L. F. Romão and P. P. Garcez*

Here we have examined the of Centromere protein J (CENPJ) expression and function in human glioblastoma cells. We found that CENPJ is overexpressed in glioblastoma cells. Through gain and loss of function of Cenpj, we show that it regulates morphology, cell proliferation and migration in glioblastoma. Our data suggest that CENPJ regulates glioblastoma migration through its PN2-3 domain, known to destabilize microtubules. This research contributes to the understanding of relevant aspects of the glioblastoma biology, suggesting that CENPJ could be a potential target for therapeutic intervention.




Read the full article on page 501.