

# Exploring how activity in the hippocampal CA2 region encodes social interactions

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People with neuropsychiatric disorders sometimes exhibit anomalous social behaviors, such as antisocial tendencies or dramatic behavioral changes. These changes could be the manifestation of deficits in social memory, the ability to encode and retain information related to social interactions.

Past neuroscientific studies suggest that the hippocampus region in the brain is responsible for the formation of different types of memories, including social memories. While the role it plays in the formation of social [memory](#) is now well documented, how it encodes and represents social information is still poorly understood.

Researchers at Columbia University and the National Institute of Mental Health (NIH) have recently carried out a study aimed at determining how social information is encoded within the hippocampus and more specifically in the hippocampal CA2 region. This brain region has been often found to be associated with the encoding of social information into declarative memory, a form of long-term memory that allows

humans or animals to consciously recall specific facts or events.

Studies that examined tissue samples extracted postmortem from individuals with schizophrenia or bipolar disorder have found that they presented 30% less parvalbumin-positive (PV<sup>+</sup>) interneurons in the hippocampal CA2 region than those extracted from individuals with no neuropsychiatric conditions. Moreover, research identified the selective loss of PV<sup>+</sup> neurons in the CA2 region in a specific mouse model, called Df(16)<sup>+/-</sup>.

Interestingly, these mice appeared to have profound social memory deficits even if they interacted with other mice with low levels of inhibition. A possible explanation for this could be a simultaneous hyperpolarization and lower excitability of their pyramidal neurons (PNs) in the hippocampal CA2 region, which could be in turn be caused by an increased current running through TREK-1 two-pore potassium (K<sup>+</sup>) channels. PNs are multipolar brain cells that can be found in many regions of the brain, while TREK-1 K<sup>+</sup> channels are membrane proteins expressed in cells that selectively control the flow of potassium ions.

"Whether and how the opposing actions of decreased CA2 PN inhibition and enhanced TREK-1 hyperpolarizing current affect in vivo CA2 PN firing and/or contribute to social memory deficits of the Df(16)A<sup>+/-</sup> is unknown," the researchers wrote in their paper. "In this study, we addressed these questions using extracellular electrophysiological recordings from dorsal CA2 PNs and behavioral analysis in both wild-type and Df(16)<sup>+/-</sup> mice during spatial exploration and social interactions."

In their study, the researchers surgically implanted electrode bundles in the brains of nineteen male mice. This allowed them to observe activity in the CA2 hippocampal region of the mice's brains as they interacted with other mice.

Overall, the findings gathered in these experiments suggest that neuronal firing in the CA2 region accurately encoded changes in social context. For instance, the CA2 region appeared to encode memories that allow mice to determine whether they are interacting with new mice or mice they socialized with before.

"In the Df(16) <sup>+/-</sup> mouse model of the human 22q11.2 microdeletion, which confers a 30-fold increased risk of schizophrenia, CA2 social coding was impaired, consistent with the social memory deficit observed in these mice; in contrast, spatial coding accuracy was greatly enhanced," the researchers explained in their paper.

In addition to confirming the key role of the hippocampal CA2 region in encoding social memories, the findings gathered by this team of researchers show that the TREK-1 channel could rescue [social memory](#) and coding in the CA2 in Df(16) <sup>+/-</sup> [mice](#). In the future, their findings could enhance the current understanding of how social stimuli are encoded in the hippocampal CA2 region.

Ultimately, the results of this recent study could shed some light on the specific neural deficits and processes that may underpin the social behavior dysfunctions observed in people with [neuropsychiatric disorders](#), such as [bipolar disorder](#) or schizophrenia. This could in turn inform the development of alternative and more effective treatment strategies for these disorders that promote positive changes in social behavior.

**More information:** Coding of social novelty in the hippocampal CA2 region and its disruption and rescue in a 22q11.2 microdeletion mouse model. *Nature Neuroscience*(2020). DOI: [10.1038/s41593-020-00720-5](https://doi.org/10.1038/s41593-020-00720-5).

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