RUHR-UNIVERSITÄT BOCHUM

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IGSN - COLLOQUIUM

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Processing and storage of synaptic information by CaMKII

The Ca2+/calmodulin(CaM)-dependent protein kinase II (CaMKII) can become partially Ca2+-independent ("autonomous") after auto-phosphorylation at T286, a mechanism described as form of molecular memory and indeed important in long-term potentiation (LTP) of synaptic strength and in memory. Contrary to previous hypothesis, our results indicate that autonomous CaMKII activity is required for information processing leading to LTP induction and memory formation, rather than in LTP maintenance or memory storage. However, our recent results indicate that storage of synaptic information may instead be mediated by CaMKII interaction with the NMDA-receptor complex. These effects and the underlying mechanisms will be discussed, also in connection with two clinically relevant findings: (i) inhibition of autonomous CaMKII activity was neuroprotective even when done hours after stroke model insults, and (ii) an intervention to interfere with the CaMKII/NMDA-receptor complex reversed pre-established addiction behavior.

Host:

Rolf Dermietzel
Neuroanatomy, Faculty of Medicine, Ruhr-University Bochum

Guests are welcome!

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