## S17-03

### MEMORY REORGANIZATION BY SYNAPTIC PROTEIN DEGRADATION

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An accumulating body of evidence shows that the retrieval process of long-term memory is not static and requires de novo protein synthesis. Thus long-term memories are dynamic and particularly become fragile during its retrieval. Importantly, memory retrieval is regarded as a step necessary for incorporating new information into preexisting memories. We have examined whether protein degradation is involved in the memory reorganization or not. In this presentation I will present the evidence that synaptic proteins are degraded by polyubiquitination and proteasome pathway in the hippocampus after the retrieval of contextual fear conditioning. In addition, we found that the infusion of a proteasome inhibitor into the hippocampus prevented the memory impairment induced by anisomycin, a protein synthesis inhibitor. This indicates that ubiquitin/proteasome-dependent protein degradation is involved in destabilization processes accompanying the memory retrieval. It also supports our hypothesis that preexisting memory is disrupted by synaptic protein degradation before updated memory is strengthened by protein synthesis. Our data also showed that synaptic protein degradation plays a critical role in fear memory extinction, a simple form of memory reorganization. Taken together, synaptic protein degradation is critically involved in the reorganization of the preexisting memories.

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