Forgetting is For getting

2023-03-30

健忘小队

“If we remembered everything, we should on most occasions be as ill off as if we remembered nothing.” —— William James(1890)

Highly superior Autobiographical Memory

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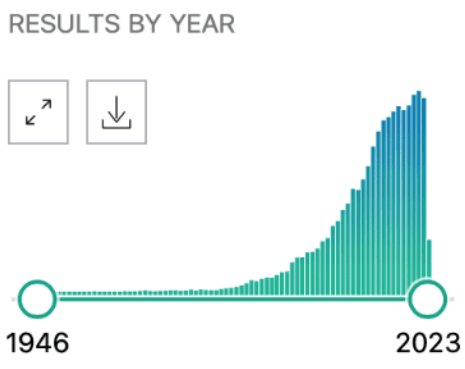
A Case of Unusual Autobiographical Remembering

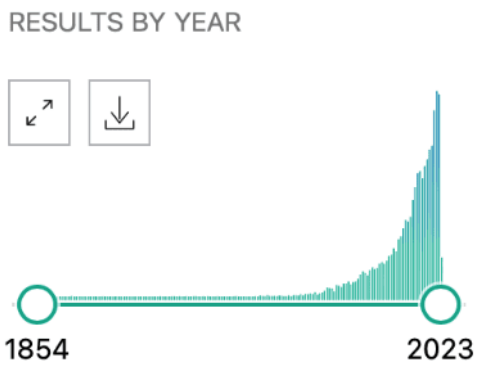
ELIZABETH S. PARKER[1], LARRY CAHILL[2] and JAMES L. McGAUGH[2]

Some people call me the human calendar while others run out of the room in complete fear but the one reaction I get from everyone who eventually finds out about this “gift” is total amazement. Then they start throwing dates at me to try to stump me… . I haven’t been stumped yet. Most have called it a gift but I call it a burden. I run my entire life through my head every day and it drives me crazy!!!… .

Learning & Memory

Forgetting





PART 1 : The overview of forgetting: Definition and Types ——JSH

PART 2 : The neuroscience of forgetting in *Drosophila*——MMZ

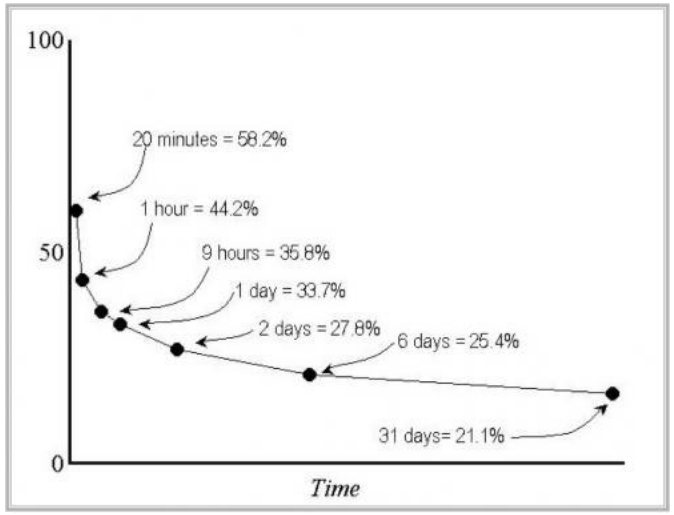
PART 3 : The potential factors associated forgetting processing——LZQ

Why forgetting is commonly happened in our everyday lives?

Pioneer of forgetting

1885 Herman Ebbinghaus

“forgetting curve”



Theories of forgetting

1885 | Forgetting Curve

1913 | Decay theory

Memory is forgotten simply by the dissipation of memory over time.

Passive Forgetting

Theories of forgetting

1885 | Forgetting Curve

1913 | Decay Theory

1932 | John McGeoch

“In time iron, when unused, may rust,
but oxidation, not time, is responsible”

Brain activity

Theories of forgetting

1885 | Forgetting Curve

1913 | Decay Theory

Interference Theory

other competing memories before (proactive) or after (retroactive) the learning event

Motivated Forgetting

an emotional motive to suppress the memories

Retrieval-induced Forgetting

recalling some items of a memory inhibits recollection of other items

Active Forgetting

What is the neuroscience of active forgetting?

1885 | Forgetting Curve

1913 | Decay Theory

1932
Interference Theory
Motivated Forgetting
Retrieval-induced Forgetting

2010 | Rac

钟毅 博士

教授

教育经历：

1978-1982年，清华大学工程物理系	学士
1982-1985年，清华大学生物科学与技术系	硕士
1985-1991年，美国爱荷华大学生物科学系	博士

工作经历：

1992-1997年，美国冷泉港实验室	助理教授
1997-2004年，美国冷泉港实验室	副教授
2004-2013年，美国冷泉港实验室	教授
2001-2013年，清华大学生命科学学院	兼职教授
2013年至今，清华大学生命科学学院	教授

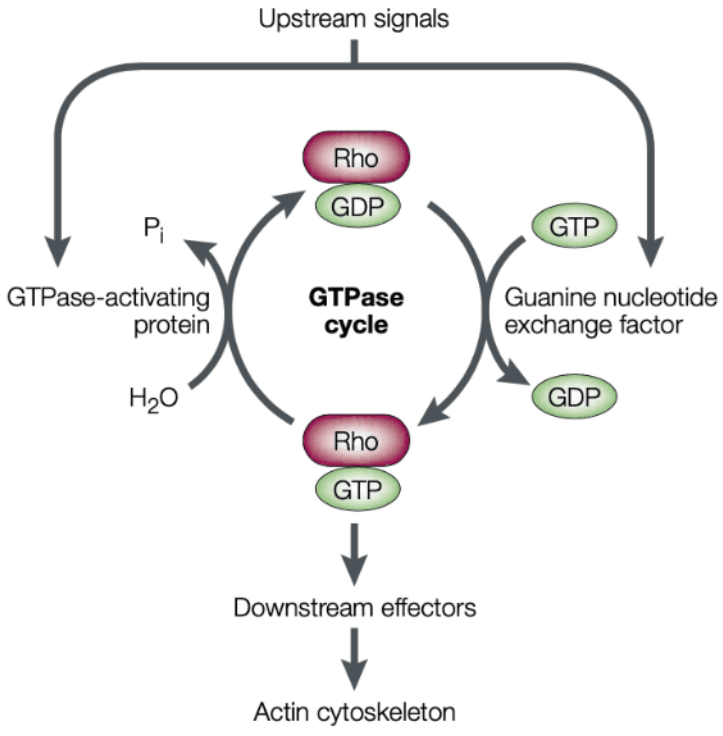
- 综合利用果蝇和小鼠等模式生物的研究优势，探究学习、记忆和遗忘的生物学机制。
- 在此基础上，一方面致力于推进人类记忆相关疾病病理机制的研究和潜在治疗药物的开发，另一方面致力于理解生物智能的深层机理并为人工智能的研究提供启发。

Forgetting Is Regulated through Rac Activity in *Drosophila*

Yichun Shuai,[1] Binyan Lu,[1] Ying Hu,[1] Lianzhang Wang,[1] Kan Sun,[1] and Yi Zhong[1,2,*]
[1]Department of Biological Sciences and Biotechnology, Tsinghua University, Beijing 100084, P.R. China
[2]Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA
*Correspondence: zhongyi@cshl.edu
DOI 10.1016/j.cell.2009.12.044

What is Rac?

The Rho family of small GTPases (Rho, Rac and Cdc4)



Dominant-negative N17 mutant (T17N): sequester the rate-limiting guanine nucleotide exchange factor
Constitutively active V12 mutant (G12V) : defective GTPase function

Published: 28 March 2002

Rac function and regulation during *Drosophila* development

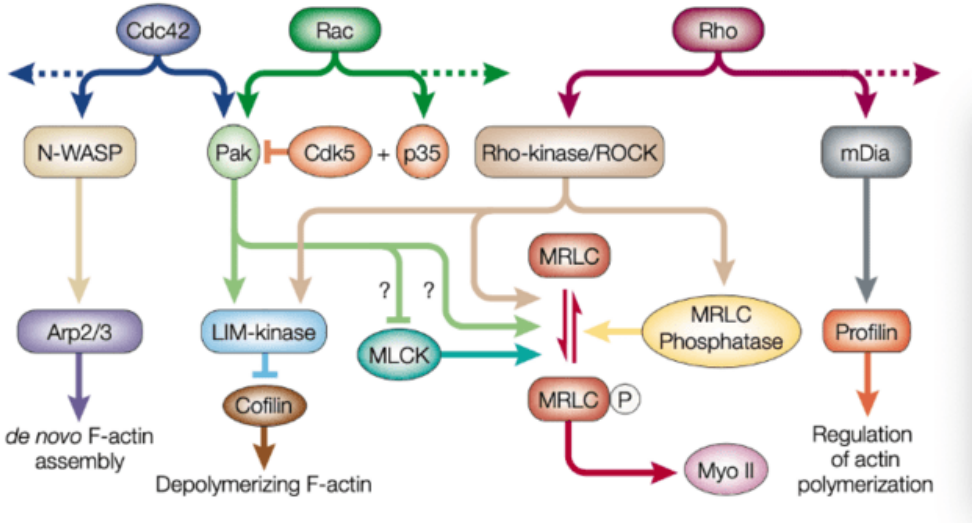
Satoko Hakeda-Suzuki, Julian Ng, Julia Tzu, Georg Dietzl, Yan Sun, Matthew Harms, Tim Nardine, Liqun Luo & Barry J. Dickson

Nature **416**, 438–442 (2002) | Cite this article

> Nat Commun. 2022 Oct 12;13(1):6014. doi: 10.1038/s41467-022-33727-6.

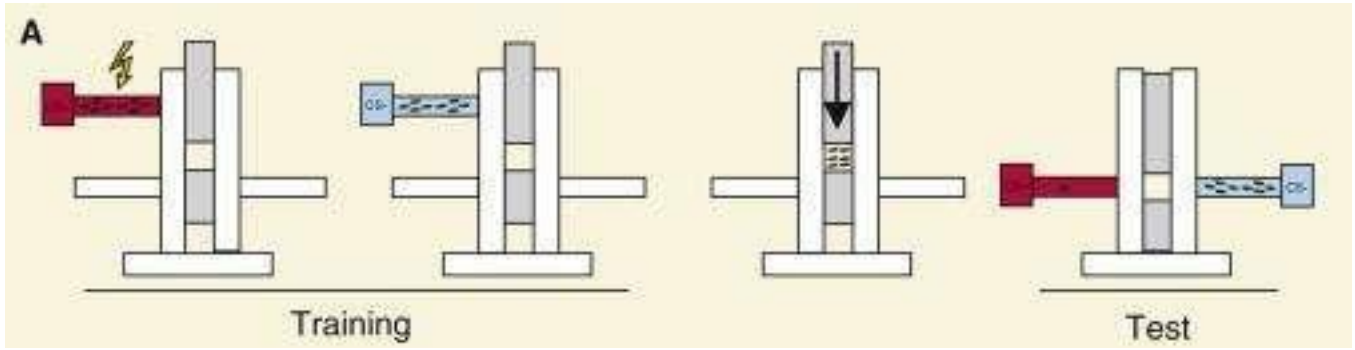
Two Rac1 pools integrate the direction and coordination of collective cell migration

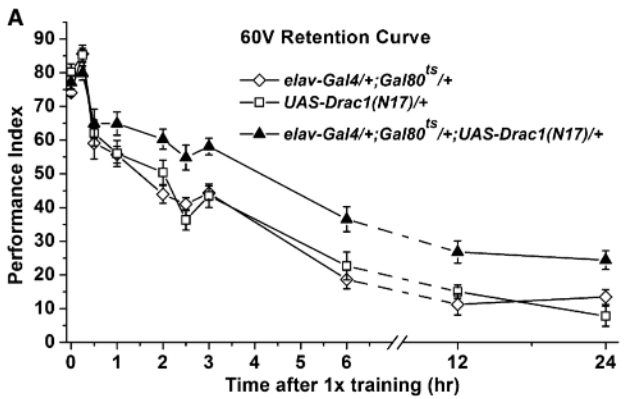
Sijia Zhou [1], Peng Li [2], Jiaying Liu [1 3], Juan Liao [4], Hao Li [1], Lin Chen [3], Zhihua Li [5], Qiongyu Guo [5], Karine Belguise [1], Bin Yi [6], Xiaobo Wang [7]

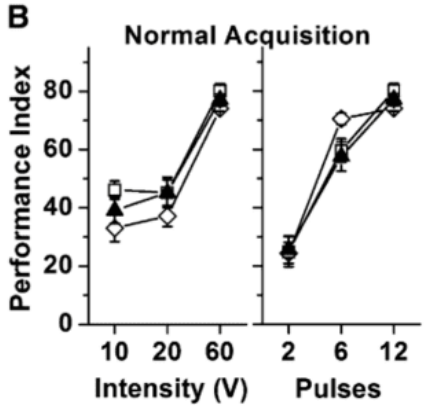


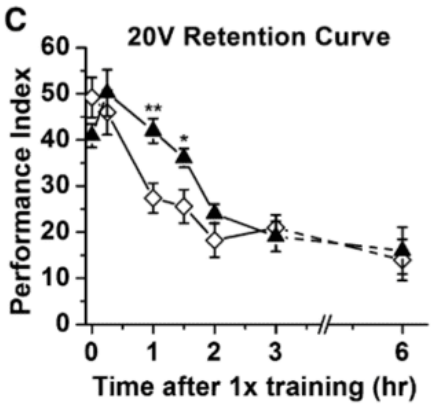
Inhibition of Rac Activity has no effect on memory formation but functions specifically on memory decaying

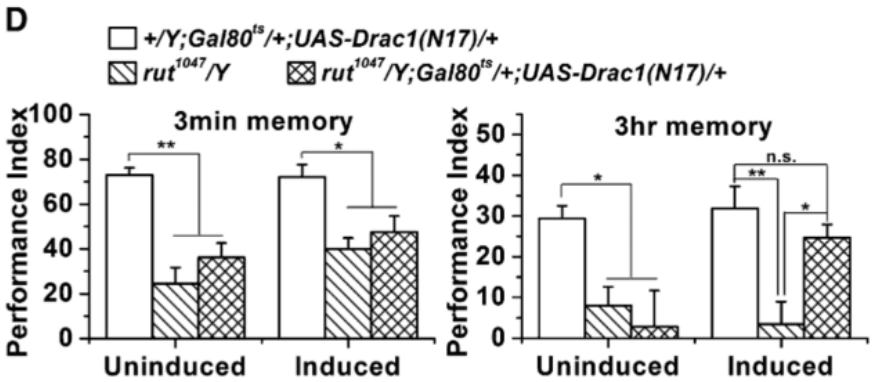
Pavlovian Olfactory Aversive Conditioning





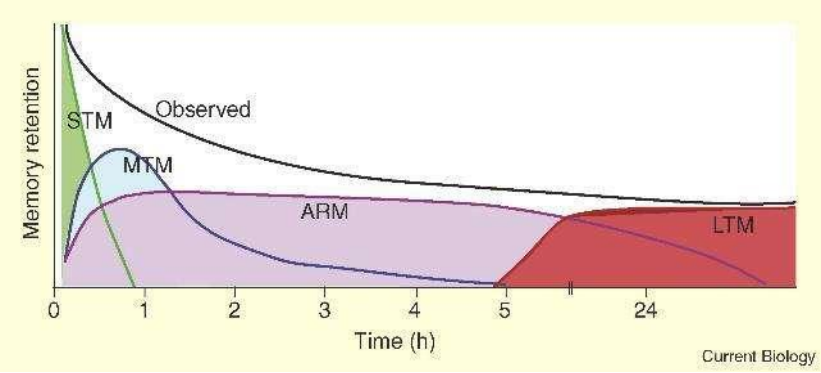




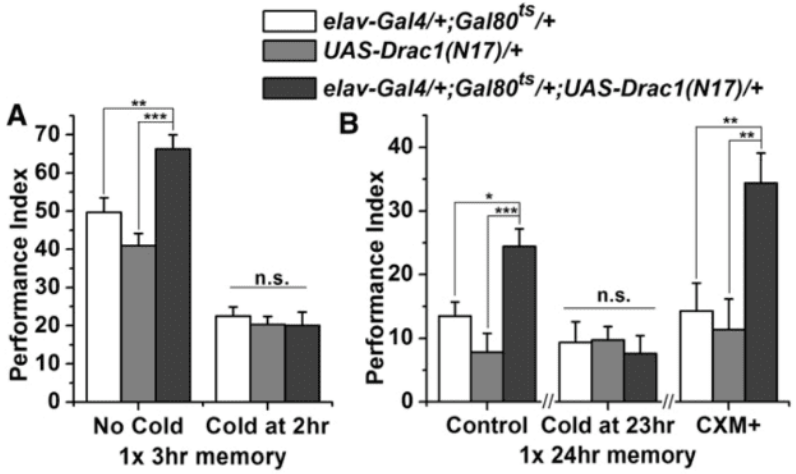


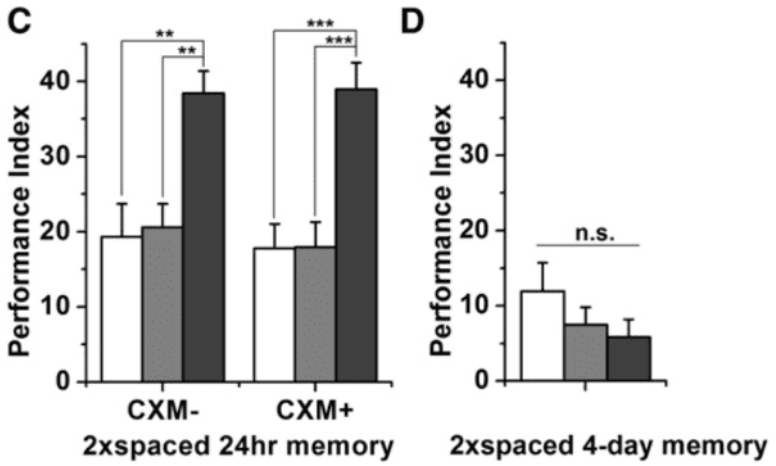
Shuai Y, et al. Cell. 2010

The prolonged memory is distinct from known consolidated memory components



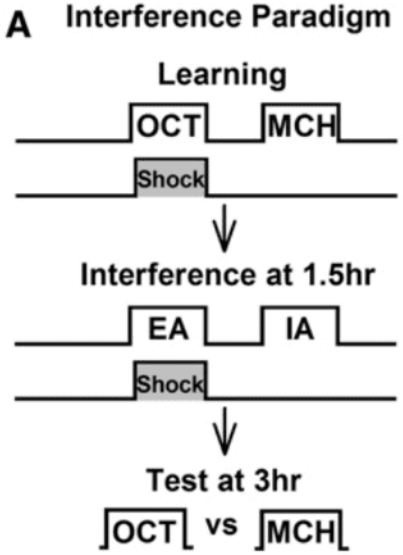
STM: short-term memory
MTM: middle or intermediate-term memory
ARM: anesthesia-resistant memory
LTM: protein-synthesis-dependent long-term memory

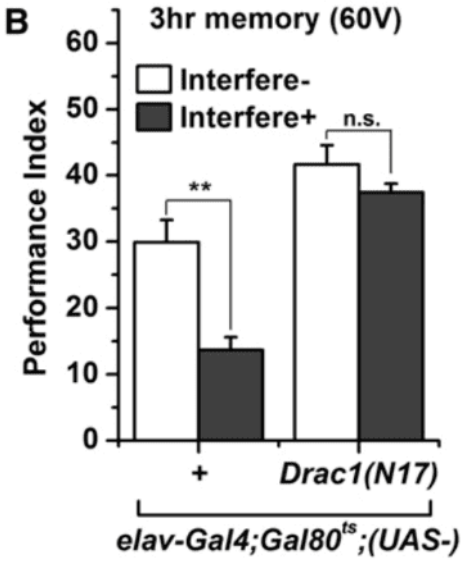


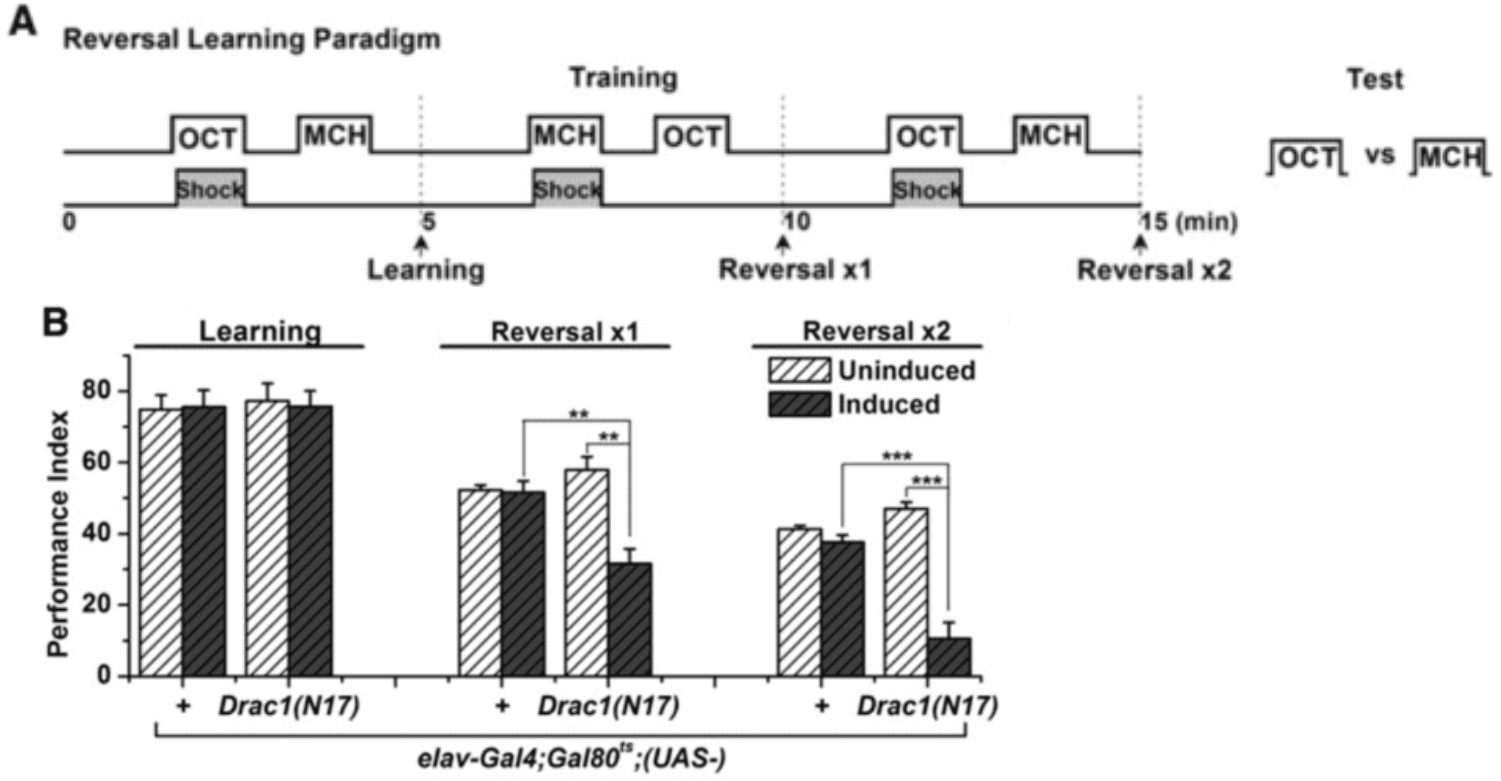


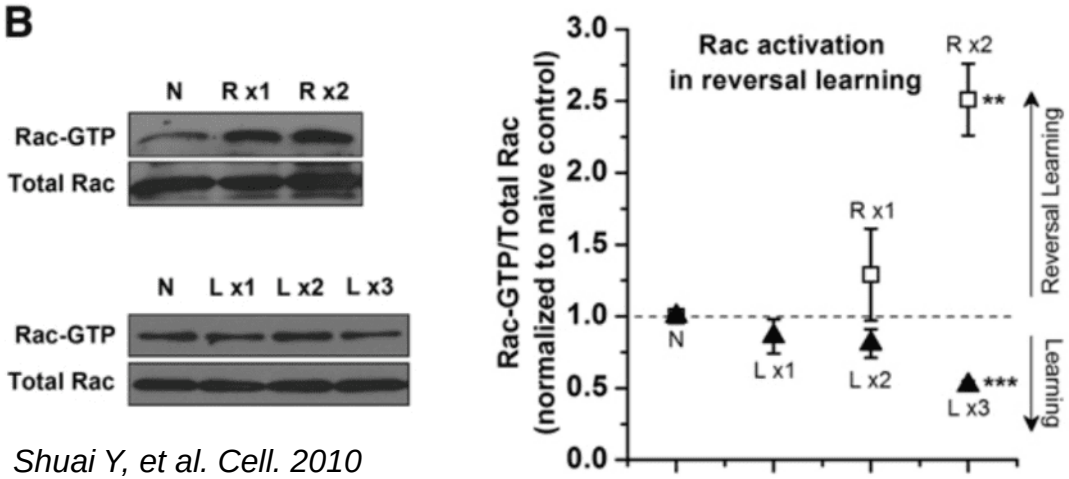
Shuai Y, et al. Cell. 2010

Rac1 also mediates interference-induced forgetting and reversal learning-activated forgetting



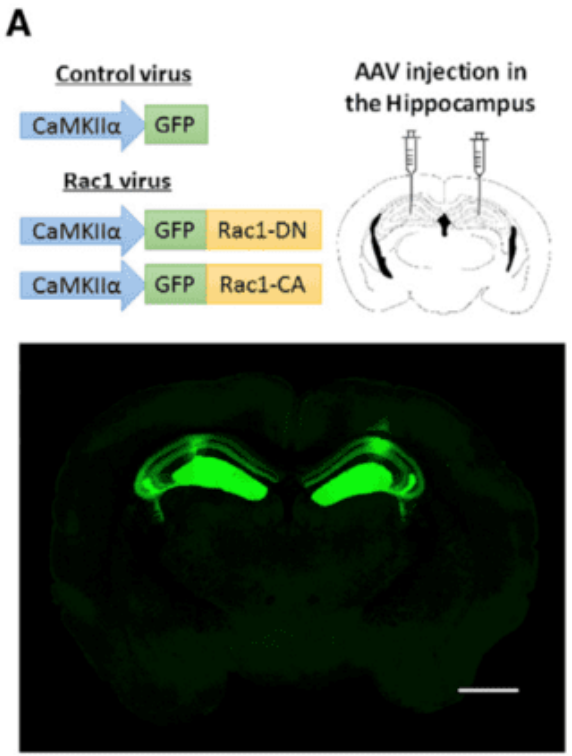


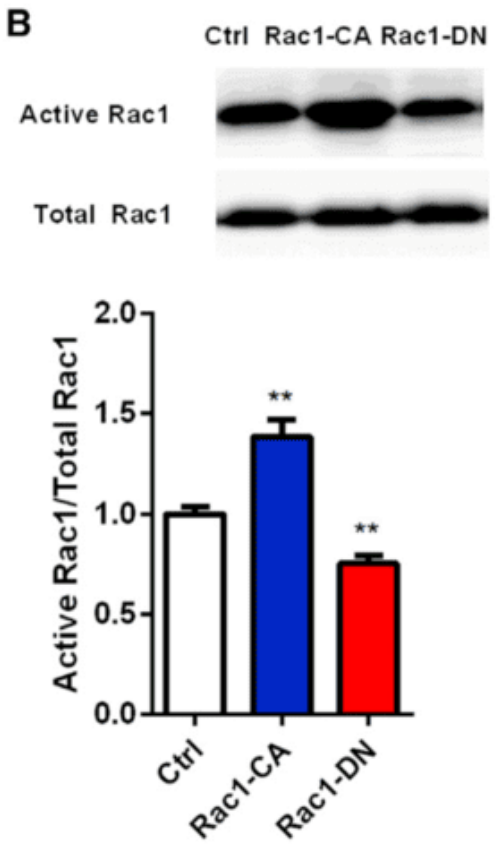


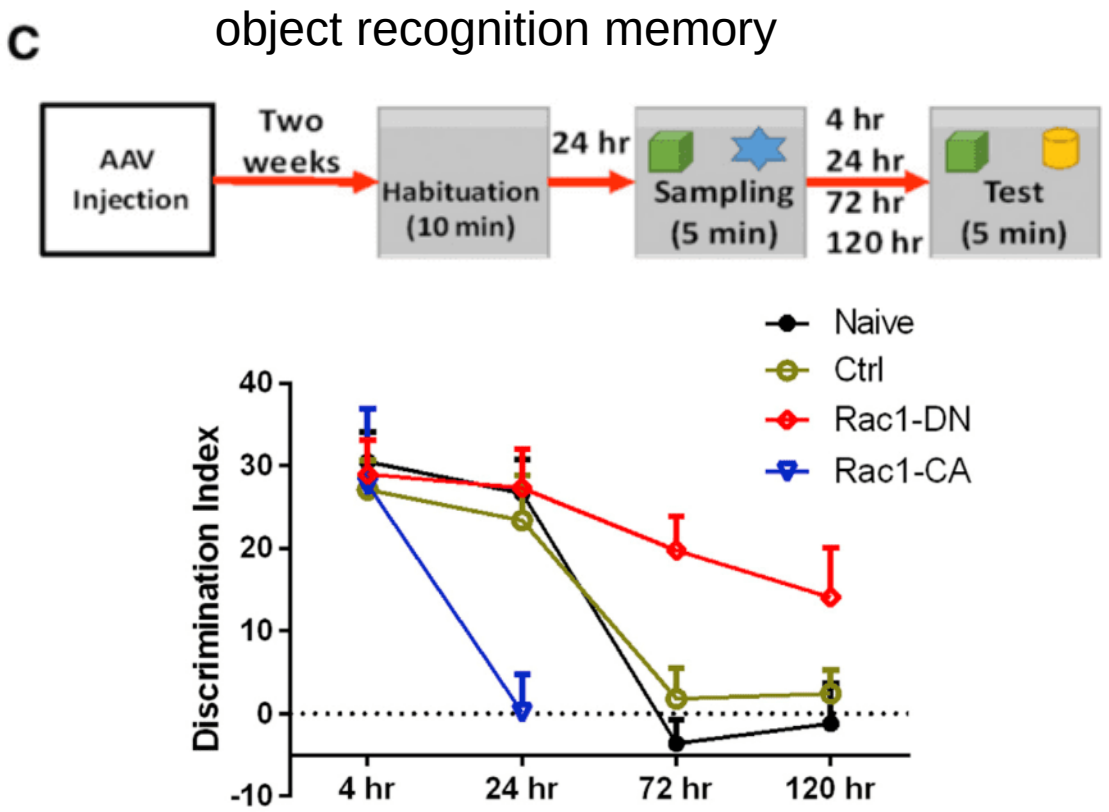


Shuai Y, et al. Cell. 2010

Decay of object recognition memory is bi-directionally regulated by Rac1 activity

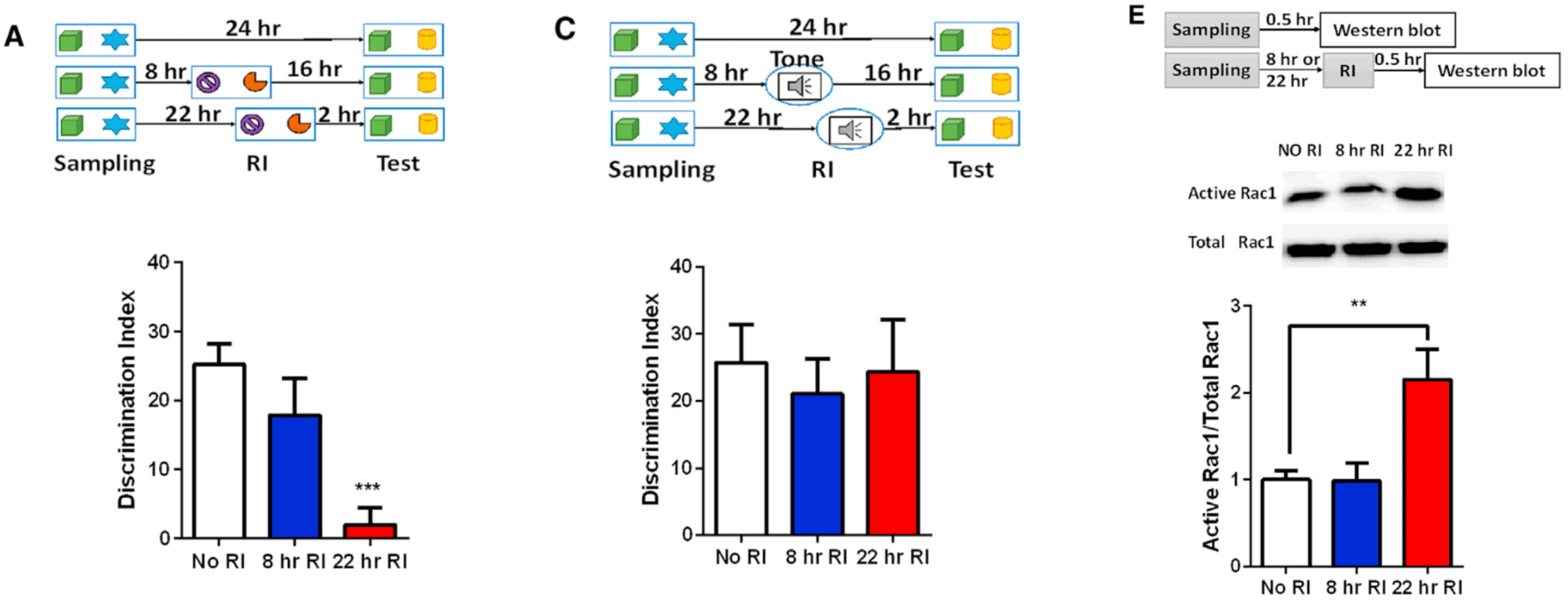






Yunlong Liu, et al. Current Biology. 2016

Rac1 activation regulates the interference-based forgetting of object recognition memory

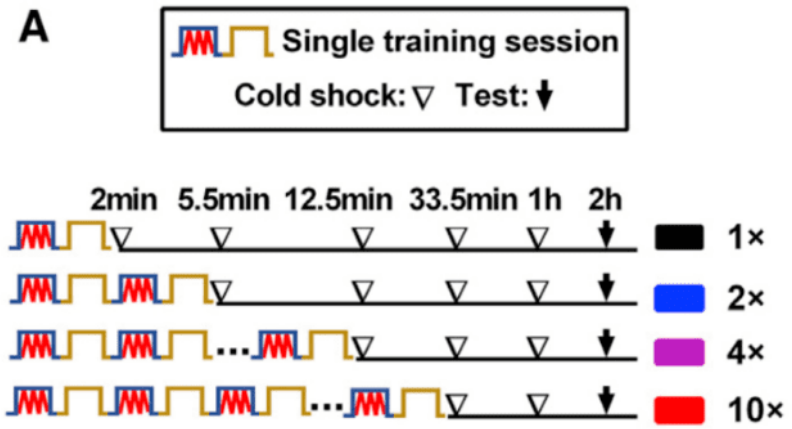


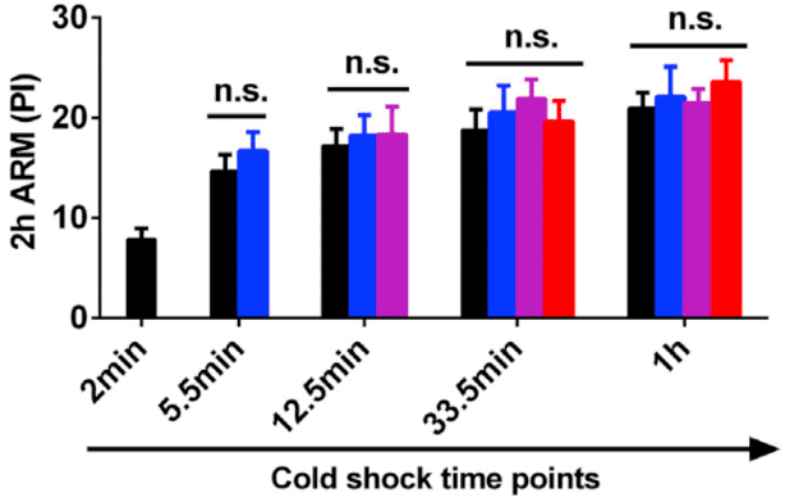
Rac1-dependent forgetting is evolutionarily conserved from invertebrates to vertebrates.

Yunlong Liu, et al. Current Biology. 2016

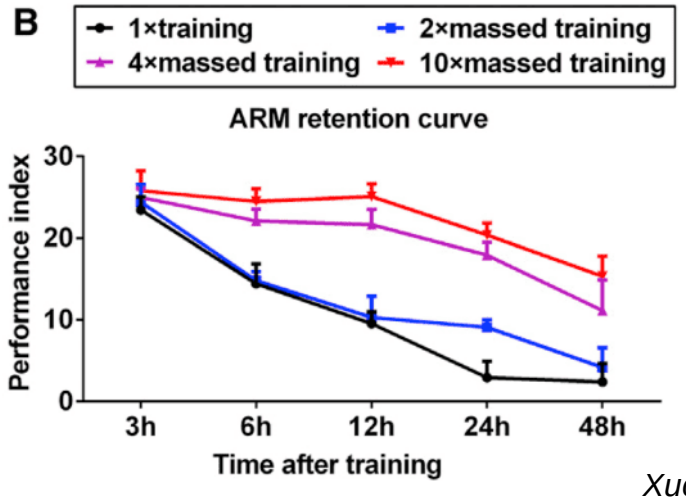
Repetition-induced ARM improvement is due to slower decay but not enhanced formation

The time course of ARM formation is solely determined by the first session of training.



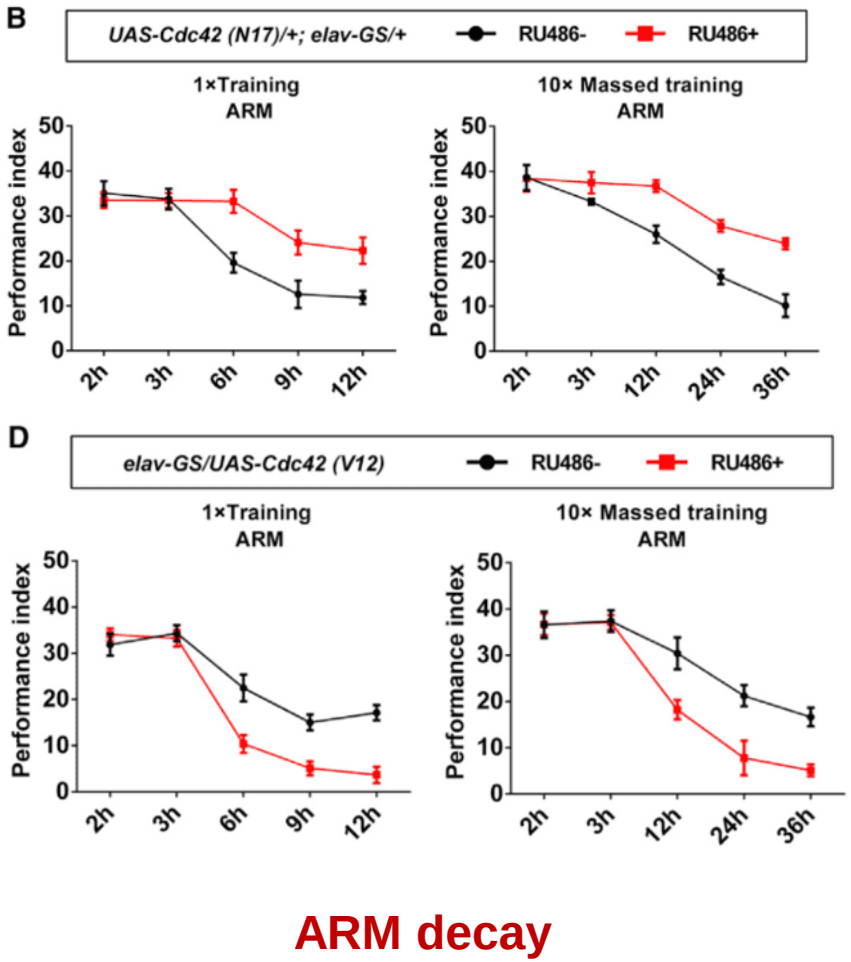


The subsequent trials are devoted not to acquiring information but to inhibiting forgetting of the ARM.

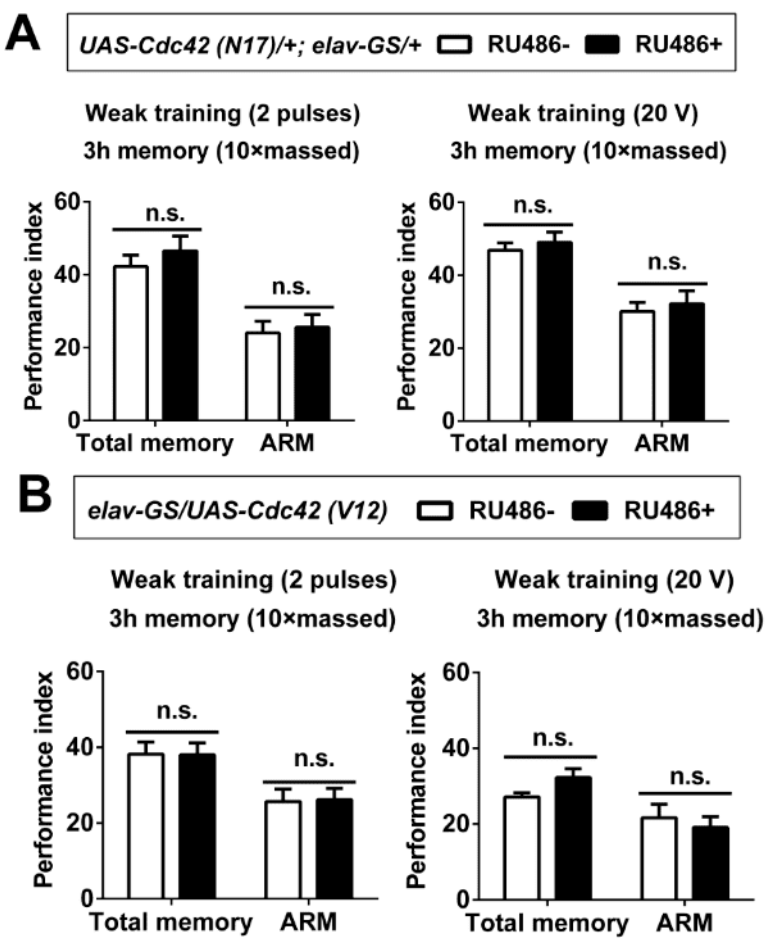


Xuchen Zhang, et al. Cell Reports. 2016.

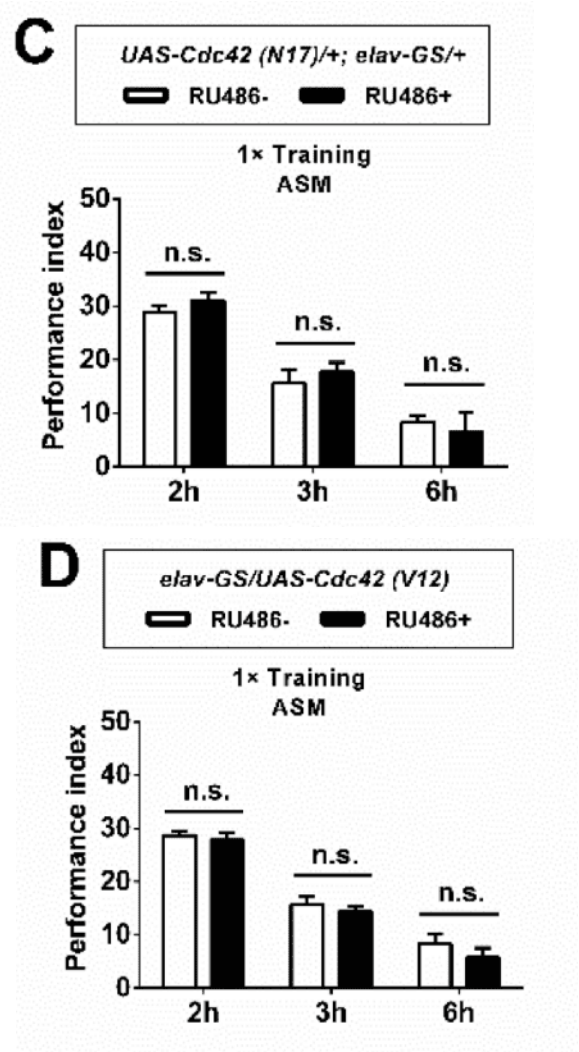
Cdc42 activity specifically regulates time-based ARM decay



ARM decay



ARM formation

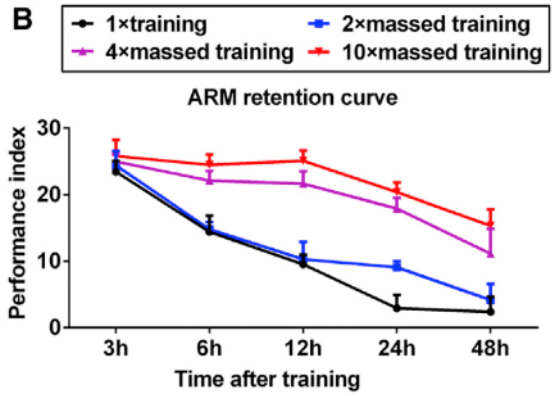


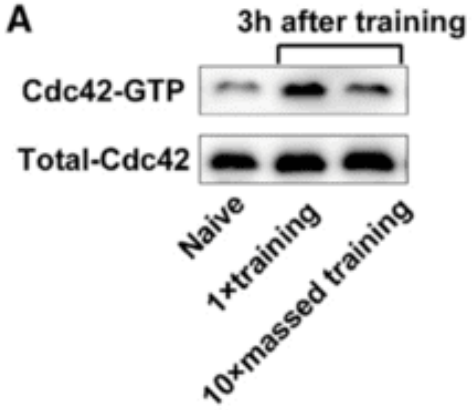
ASM formation

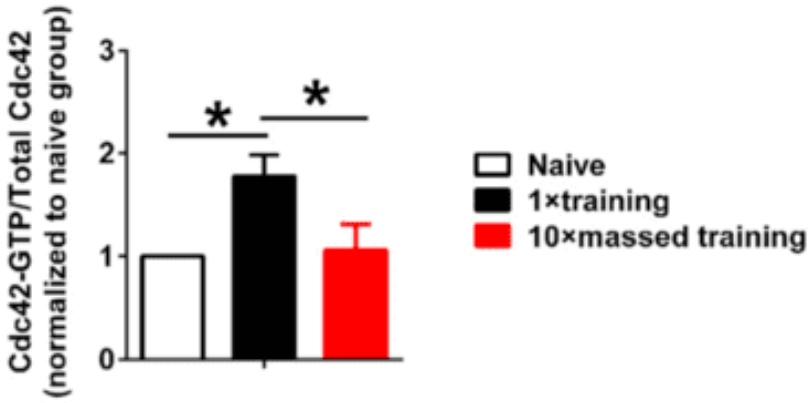
ASM decay

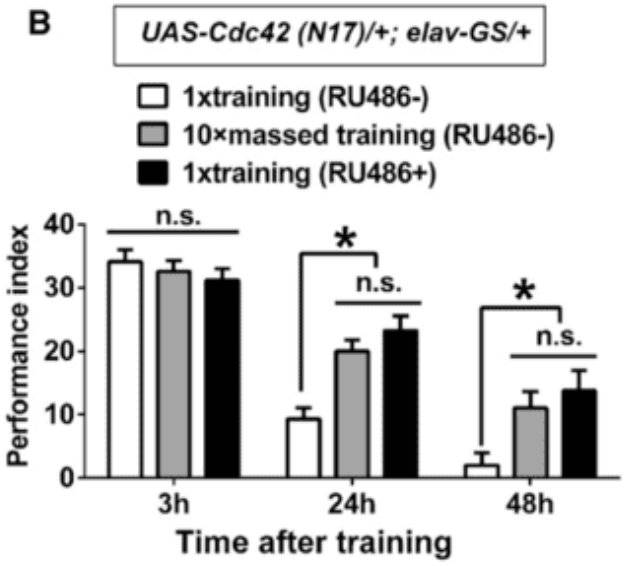
Xuchen Zhang, et al. Cell Reports. 2016.

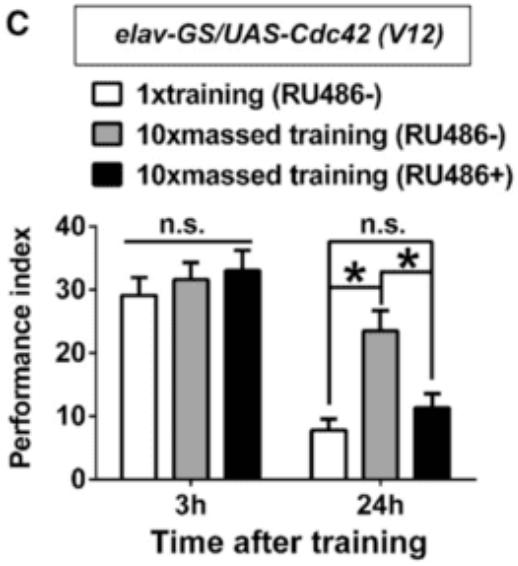
Repeated learning prolongs ARM retention through suppression of Cdc42-dependent forgetting

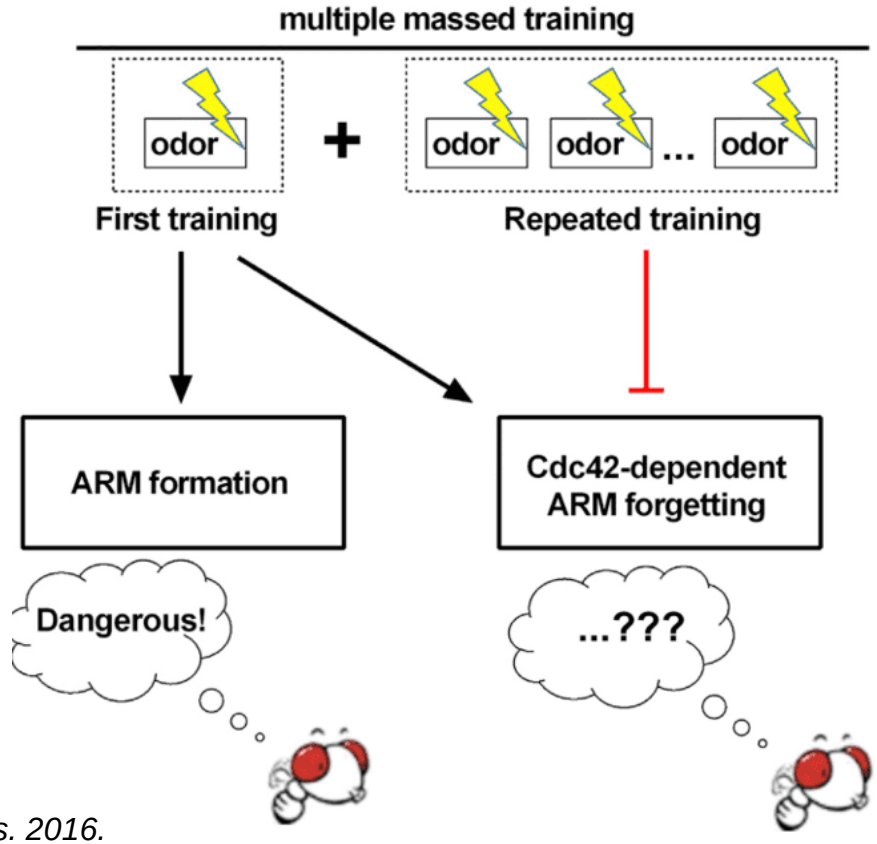






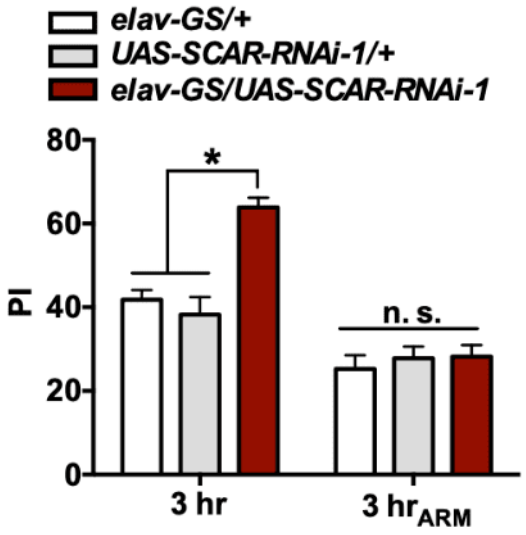


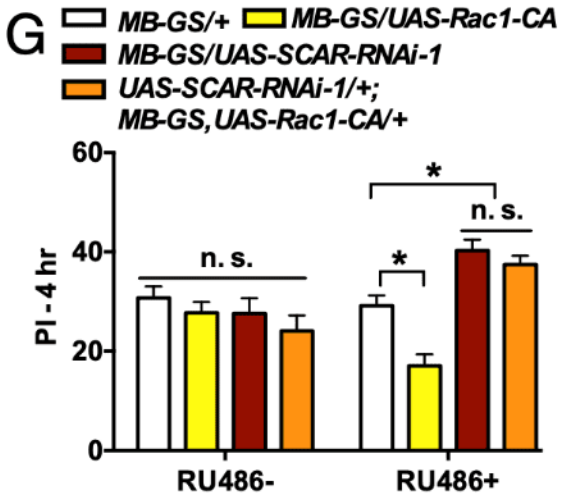




Xuchen Zhang, et al. Cell Reports. 2016.

SCAR/WAVE and WASp, act downstream of Rac1 and Cdc42 separately to regulate ASM and ARM forgetting





ASM Forgetting

Rac1

↓

SCAR/WAVE Complex

Rho GTPases

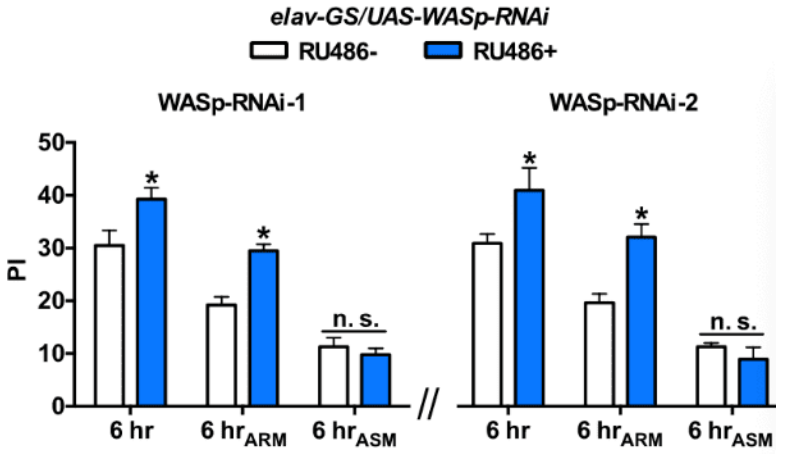
WASP family proteins

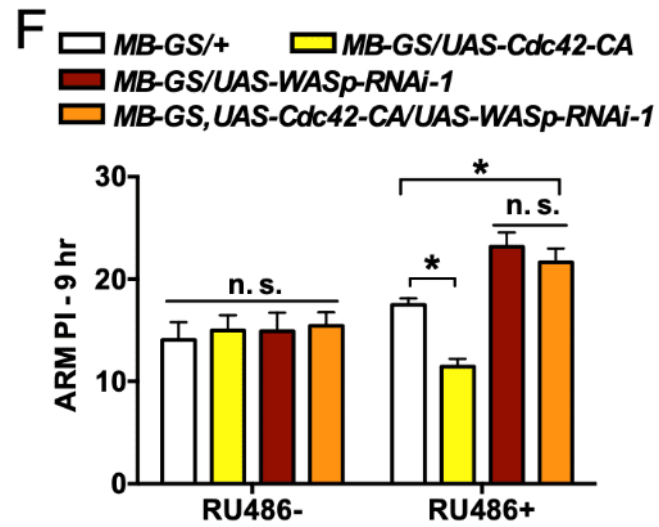
ARM Forgetting

Cdc42

↓

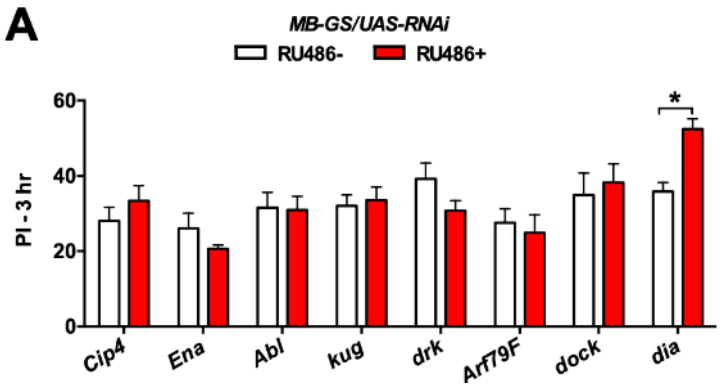
WASp

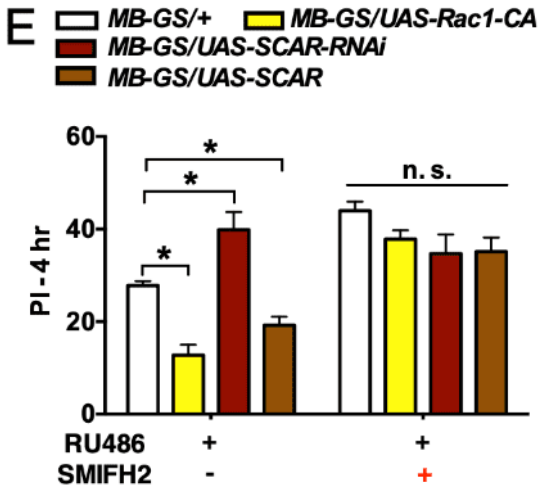




Gao Y, et al. PNAS. 2019

Rac1 and Cdc42 regulate ASM and ARM forgetting by different actin polymerization mechanisms





ASM Forgetting

ARM Forgetting

Rac1

Rho GTPases

Cdc42

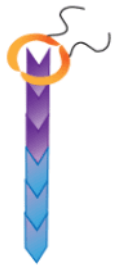
SCAR/WAVE Complex

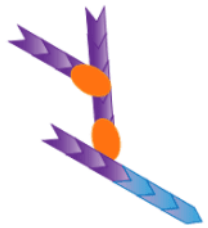
WASP family proteins

WASp

Dia

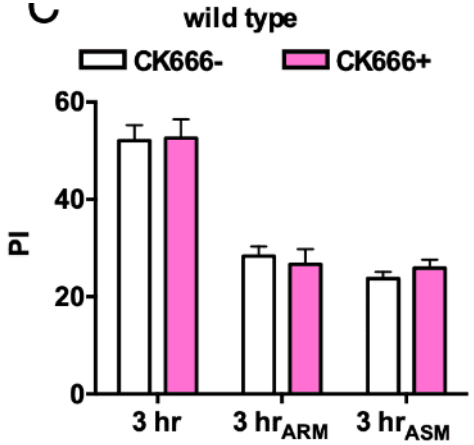
Arp2/3 Complex

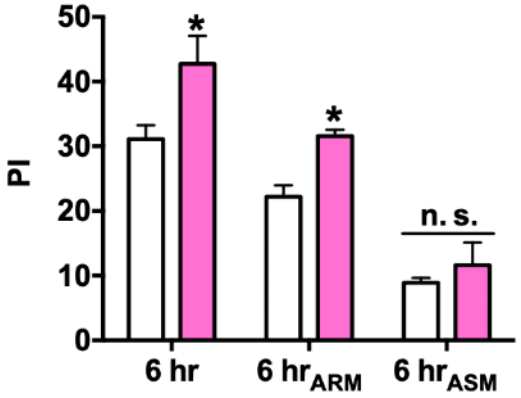


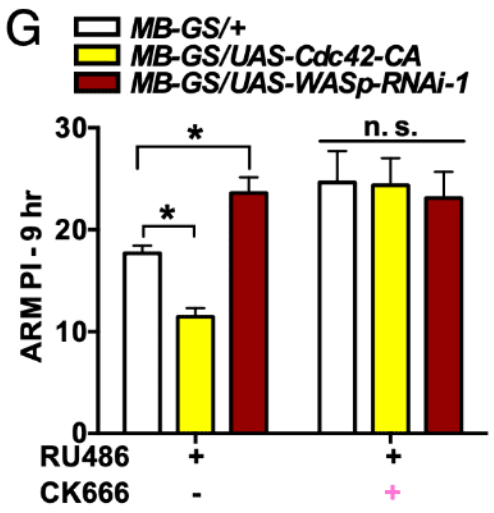


linear actin polymerization

branched actin polymerization

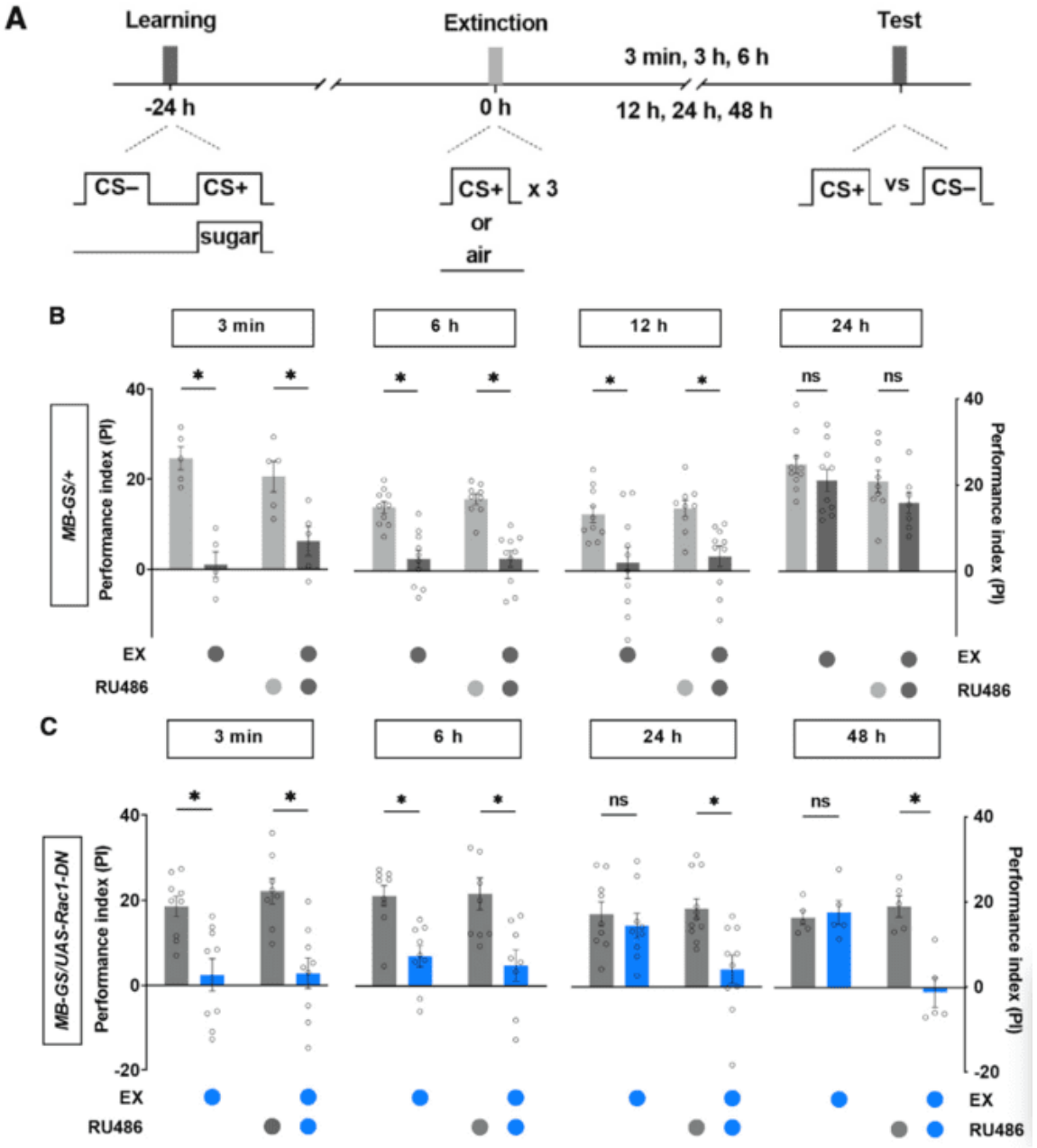


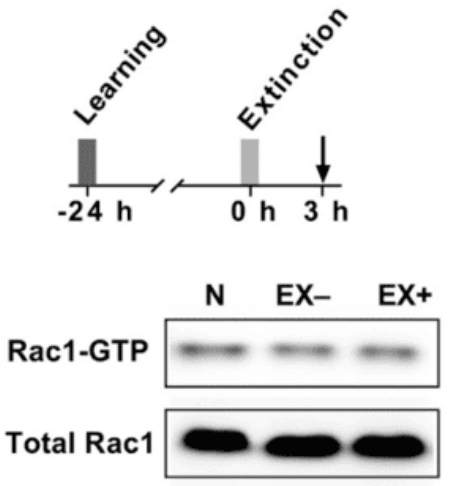


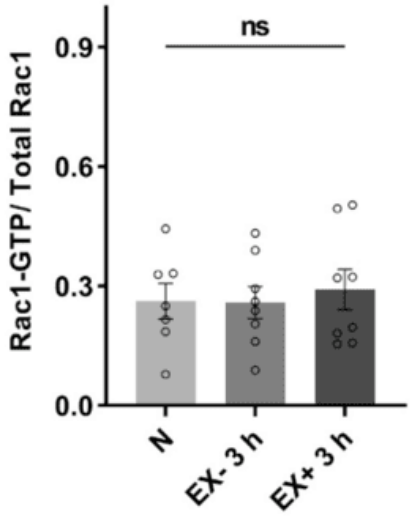


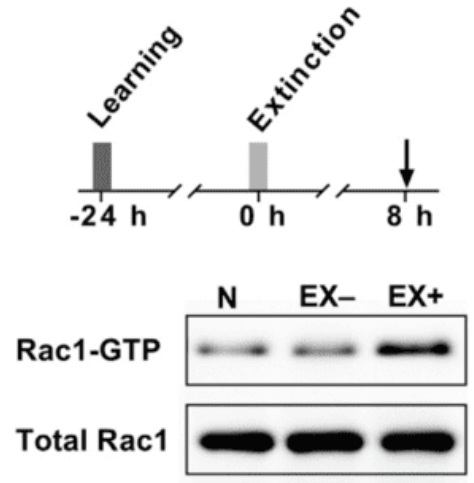
Gao Y, et al. PNAS. 2019

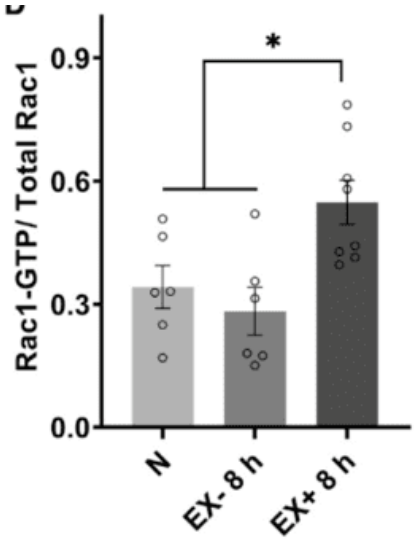
Rac1 regulates spontaneous recovery of extinguished appetitive memory





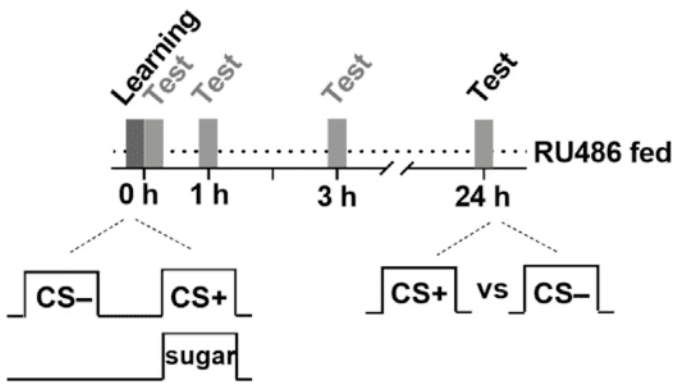


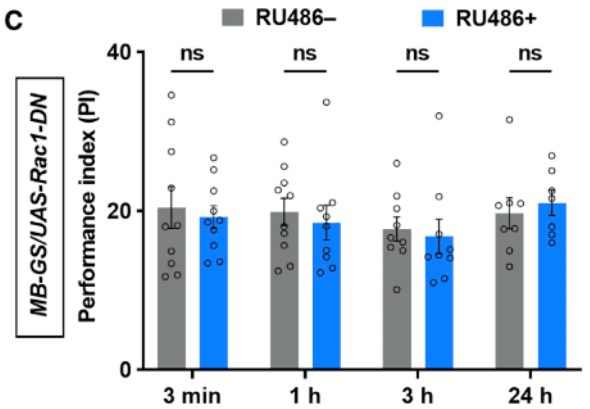


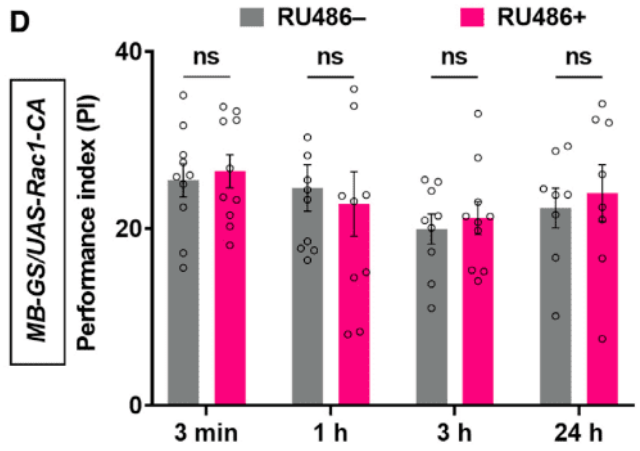


Yang Q, et al. Current Biology. 2023.

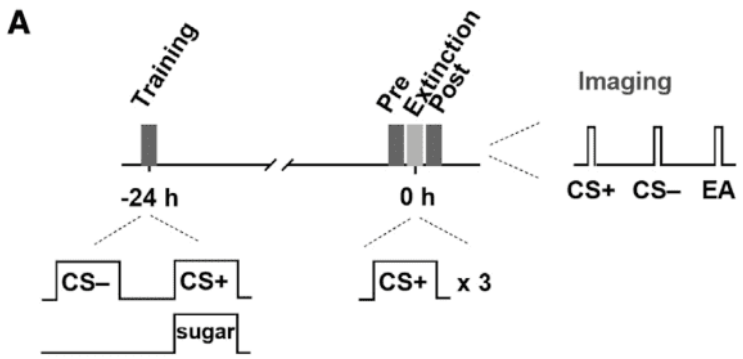
Manipulating Rac1 activity does not affect reward memory

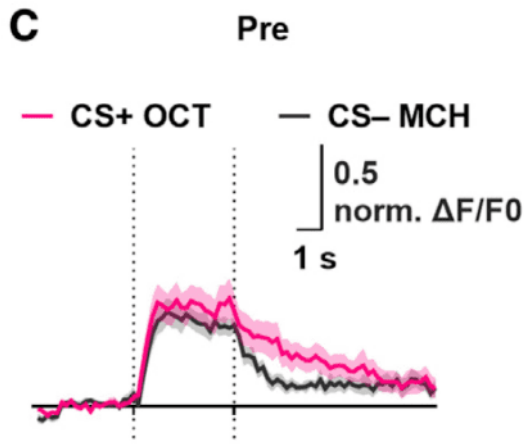


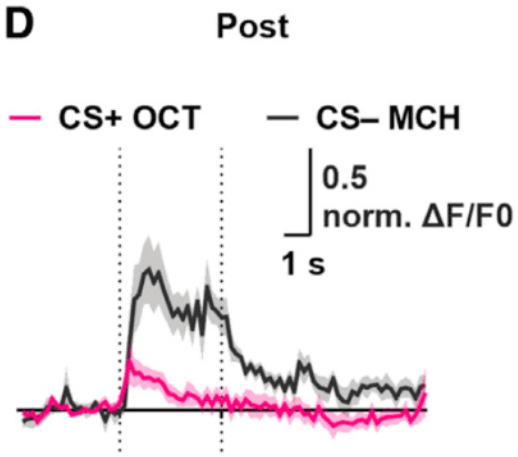




The extinction of sugar reward memory is encoded as aversive memory

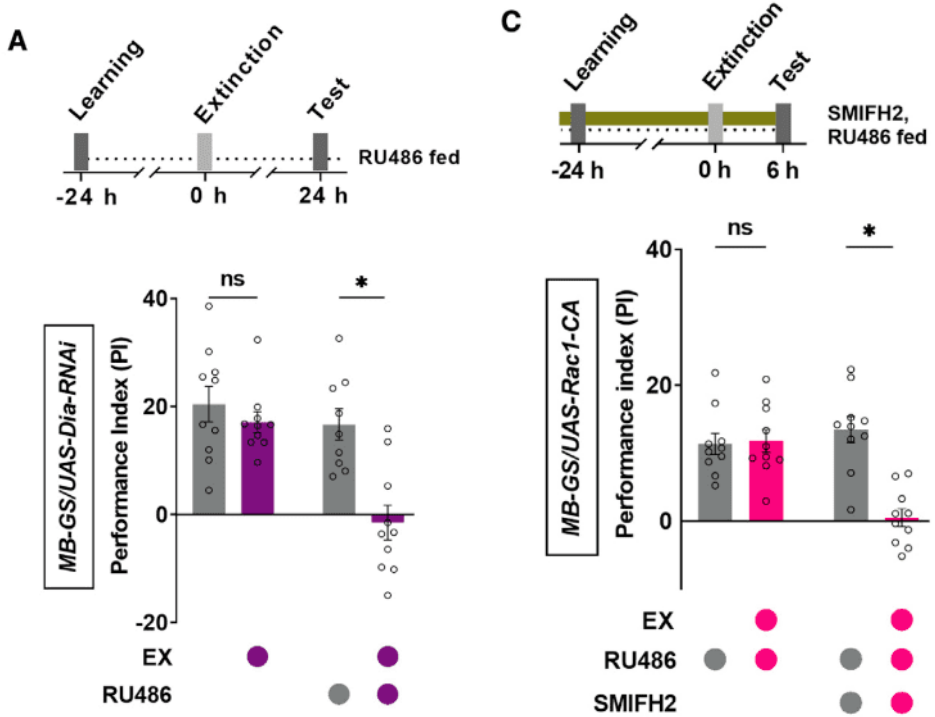


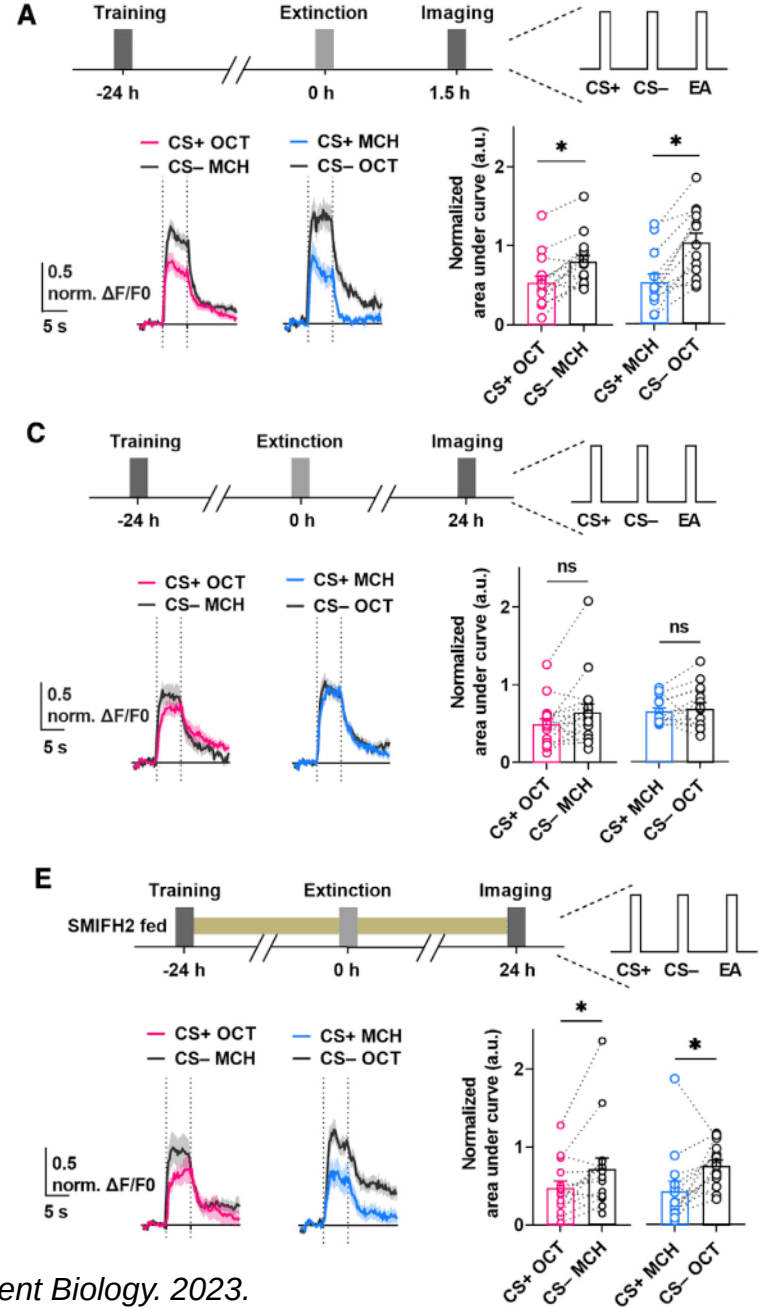




Yang Q, et al. Current Biology. 2023.

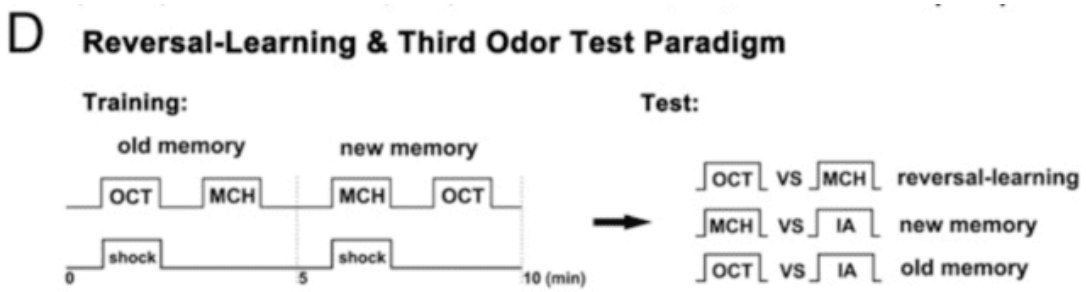
Spontaneous recovery occurs via the Rac1/Dia pathway of aversive memory forgetting

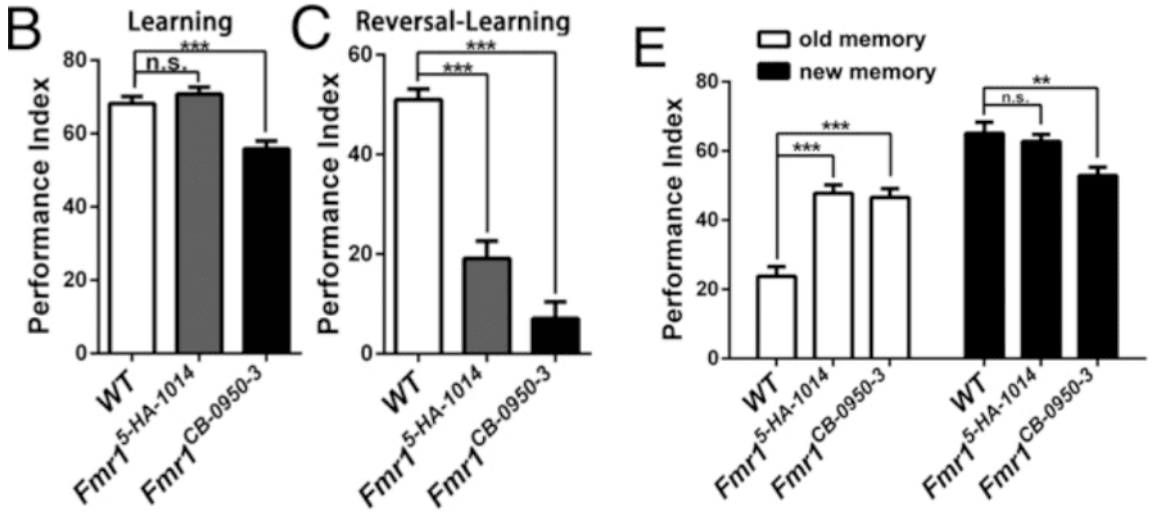


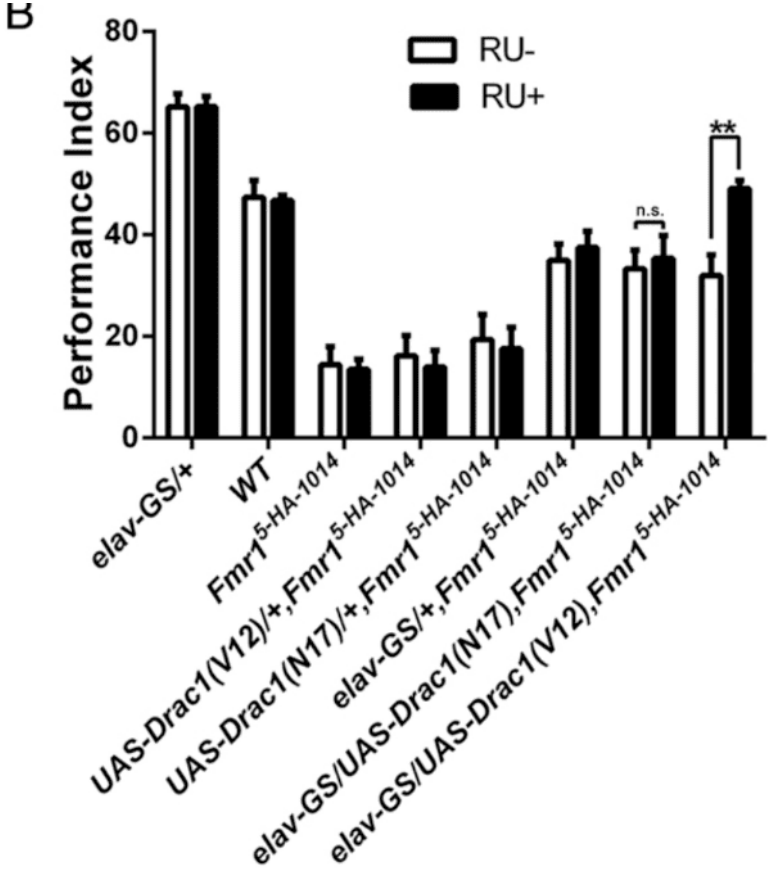


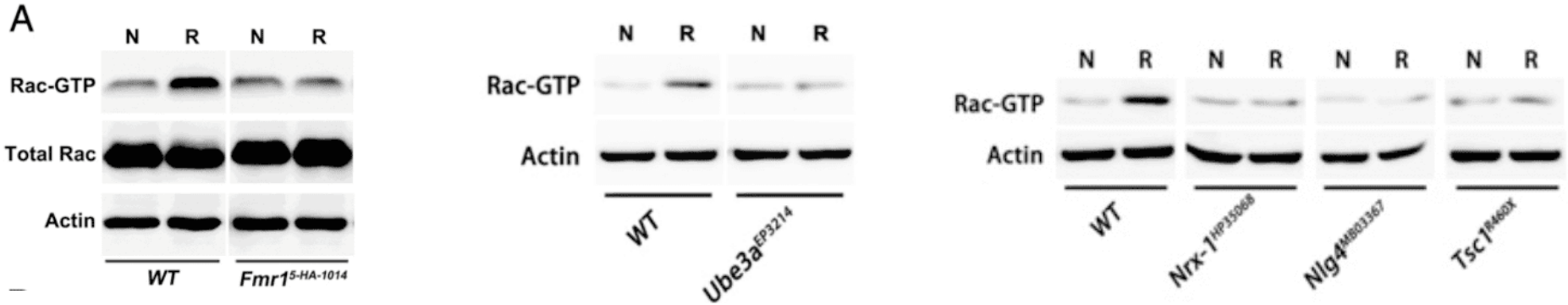
Yang Q, et al. Current Biology. 2023.

Impairment of Rac1-dependent forgetting results in behavioral inflexibility in mutants of autism-risk genes









Dong T, et al. PNAS. 2016.

Summary:

- ✓ Rac activity is critically involved in active regulation of early memory forgetting
- ✓ Rac1-dependent forgetting is evolutionarily conserved from invertebrates to vertebrates
- ✓ Cdc42 specifically regulates forgetting of anesthesia-resistant memory (ARM)
- ✓ Rac1 and Cdc42 regulate different types of memory forgetting by different actin polymerization mechanisms
- ✓ Spontaneous recovery occurs via the Rac1/Dia pathway of aversive memory forgetting
- ✓ Brain disorders are associated with Rac-dependent forgetting

What are neuronal circuits underlying Rac1-dependent forgetting?

The neuroscience of forgetting in *Drosophila*

——a simple circuit of ASM

MMZ

Neuroscience

Can human memory be stored, manipulated, and transplanted digitally?

Science fiction movies depict a day when we can upload our memories to the cloud and then have them downloaded into some form of a computer–would this be a robot perhaps, or an artificial life form? Neuroscientists and cognitive science researchers don't laugh at this prospect, but rather use it as inspiration to push the boundaries of knowledge and better understand how memory works and how it is stored in the bundles of neurons within our skulls.

Memory is the way in which the brain retains information. Our brain can record all that we, as a conscious being, experience–the feelings something evokes; the smells, sounds, sights, and thoughts it engenders; and the actions that we take and observe. Memory is a foundational aspect of our cognition that guides the way we interact with and navigate the world. Its inner workings are generally believed to rely on a dual process of two different systems, in which unconscious and routine thought processes cooperate with more conscious, problem-solving thought processes. In a certain sense, memory parallels a computer-like model, in that it involves inputs, encoding, storage, and retrieval.

But how close are we to copying our memories onto a thumb drive? Closer than ever before–but still many years away from this reality. And yet recently, scientists have made fascinating progress. In 2019, a team reported that they were able to reverse-engineer a natural memory in a mouse's brain. Essentially, they mapped the neural circuits that helped form and store the memory and then "trained" another mouse by "stimulating the brain cells in the pattern of the natural memory," writes neurodegeneration specialist Robert Martone. "Doing so created an artificial memory that was retained and recalled in a manner indistinguishable from a natural one."

The achievement of forging artificial memories could impact theoretical and experimental cognitive research as well as clinical medical science: Perhaps we can leverage our understanding of memory storage and manipulation for emancipation from devastating ailments such as Alzheimer's and posttraumatic stress disorder.

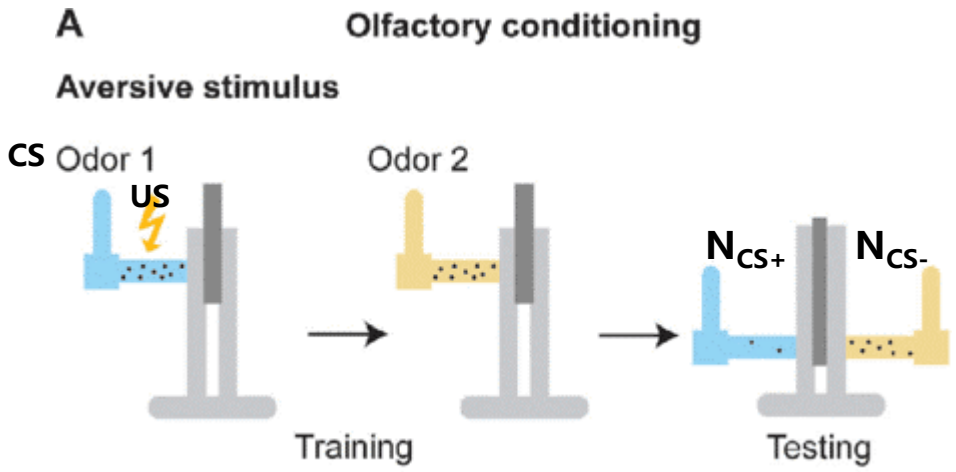
Why should we forget our memory?

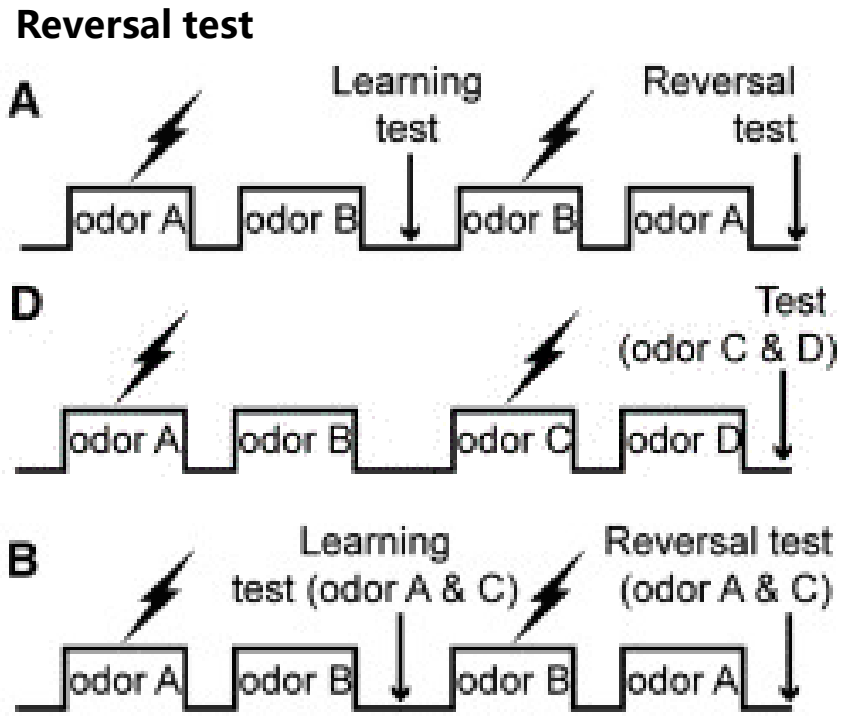
Is forgetting just a passive occurrence?

How does forgetting happen?

- The importance of Rac1 in active forgetting
- Neural circuits involved in forgetting
- How does Rac1 work in neurons

Classical olfactory and courtship conditioning assays



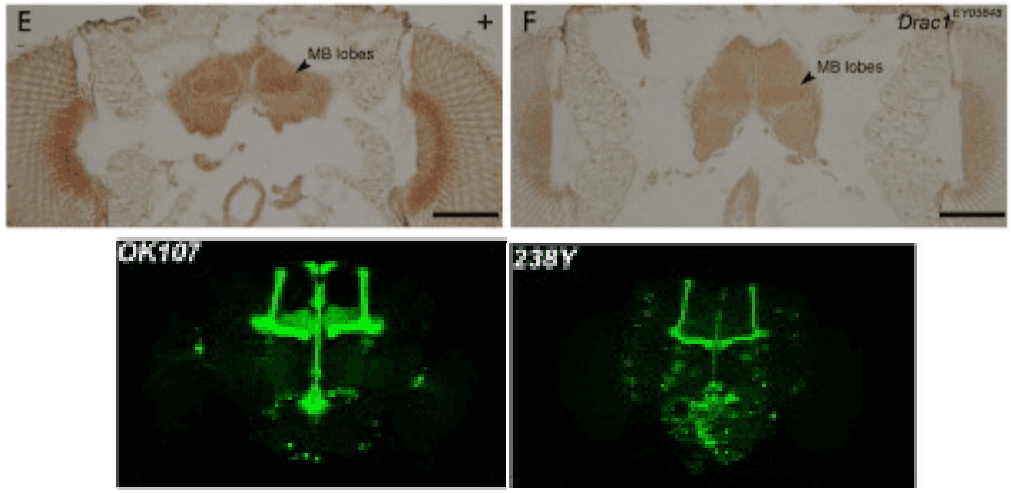


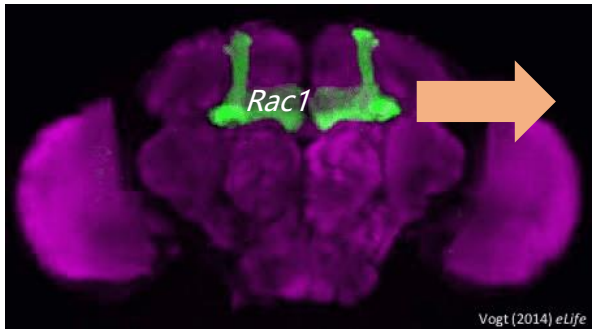
$$PI=(N_{CS-}-N_{CS+})/N$$

0 ⟷ 1

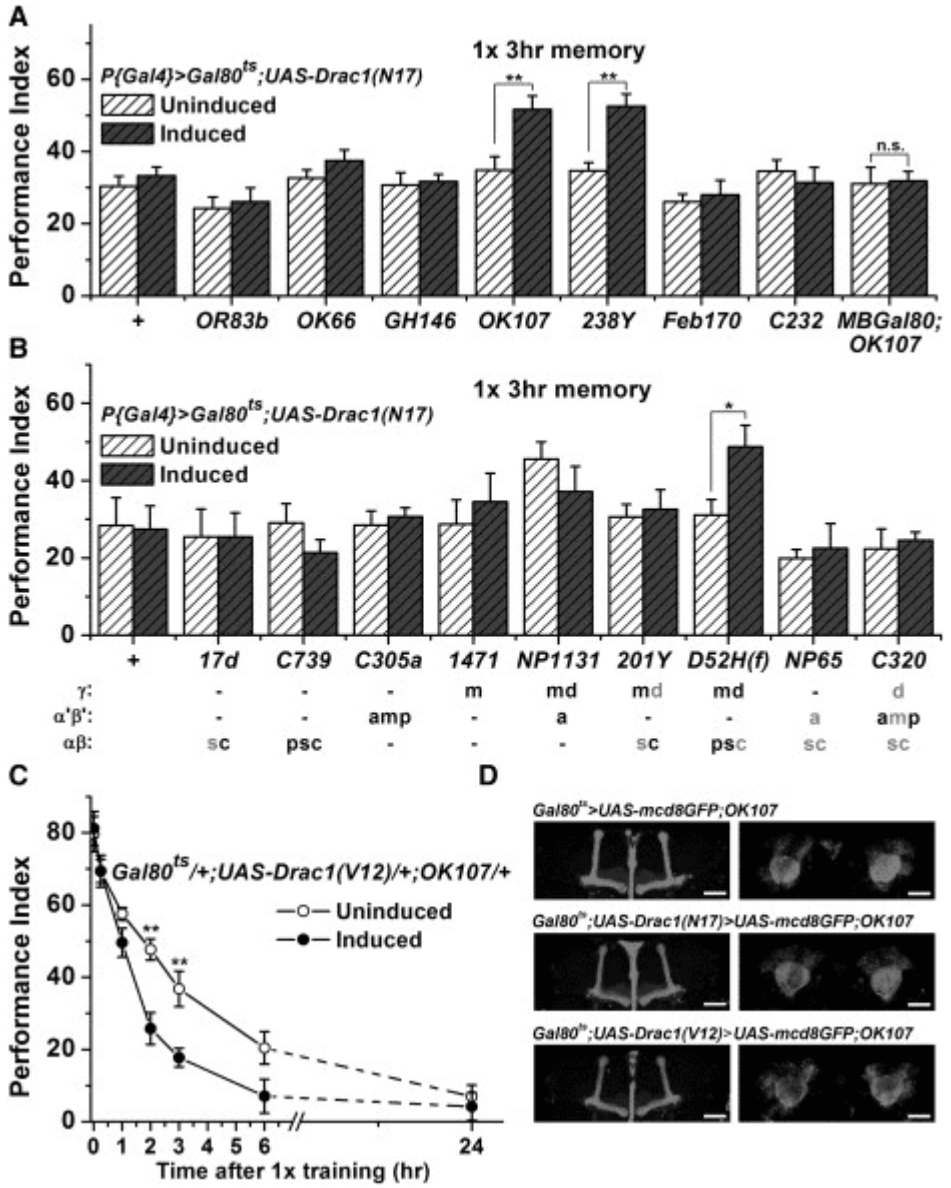
- The importance of Rac1 in active forgetting
- Neural circuits involved in forgetting
- How does Rac1 work in neurons

Bidirectional regulation forgetting by Rac1 in MB





Active forgetting



- The importance of Rac1 in active forgetting
- Neural circuits involved in forgetting
- How does Rac1 work in neurons

Dopamine and Rac1 have similar effects on memory

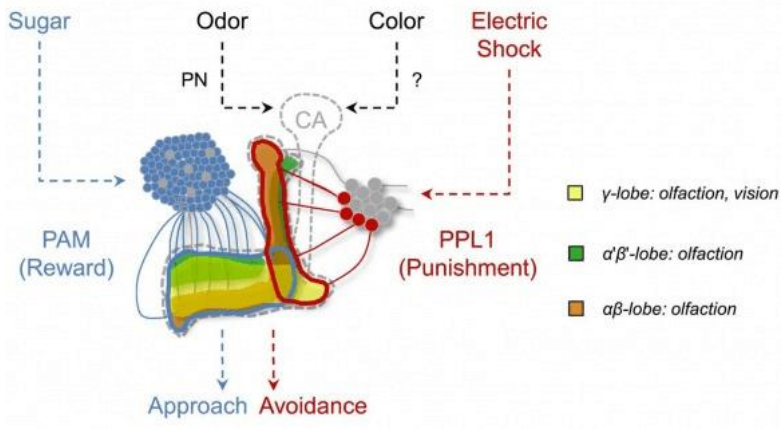
Neuron

Volume 74, Issue 3, 10 May 2012, Pages 530-542

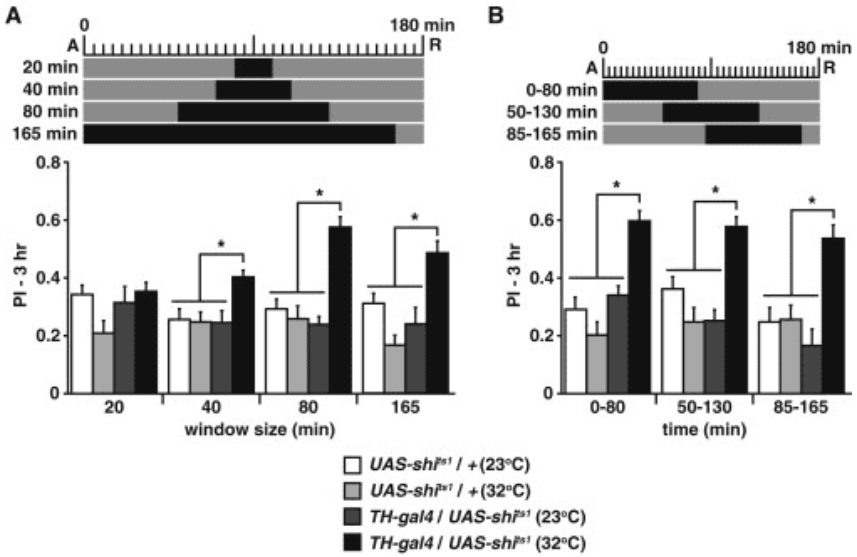
Article

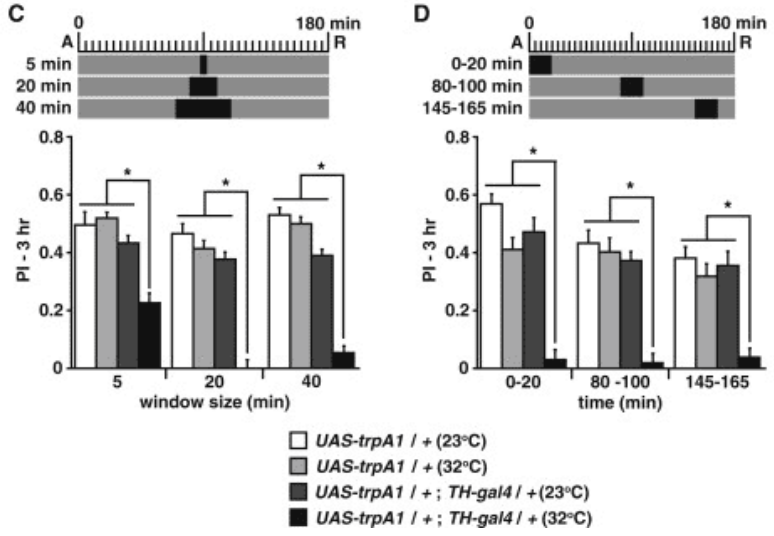
Dopamine Is Required for Learning and Forgetting in *Drosophila*

Jacob A. Berry [1 2], Isaac Cervantes-Sandoval [1], Eric P. Nicholas [1], Ronald L. Davis [1]

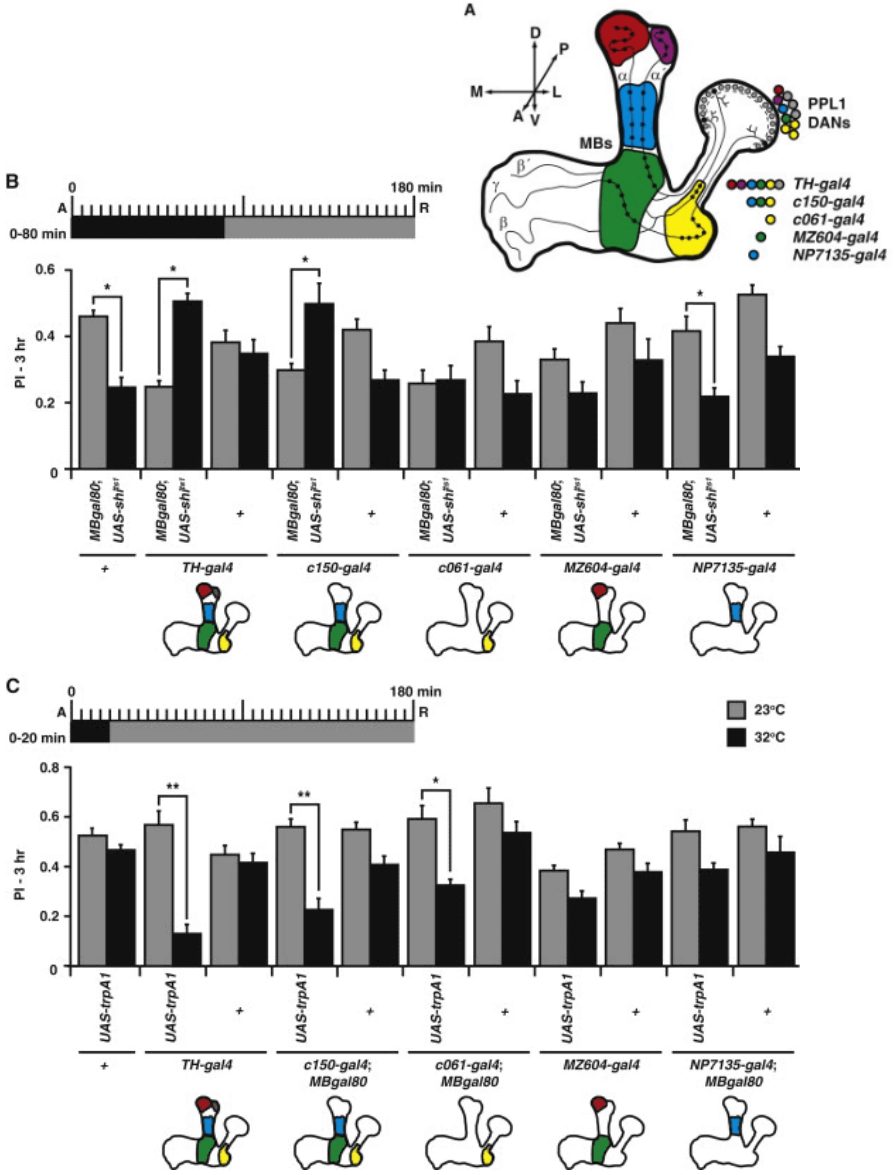


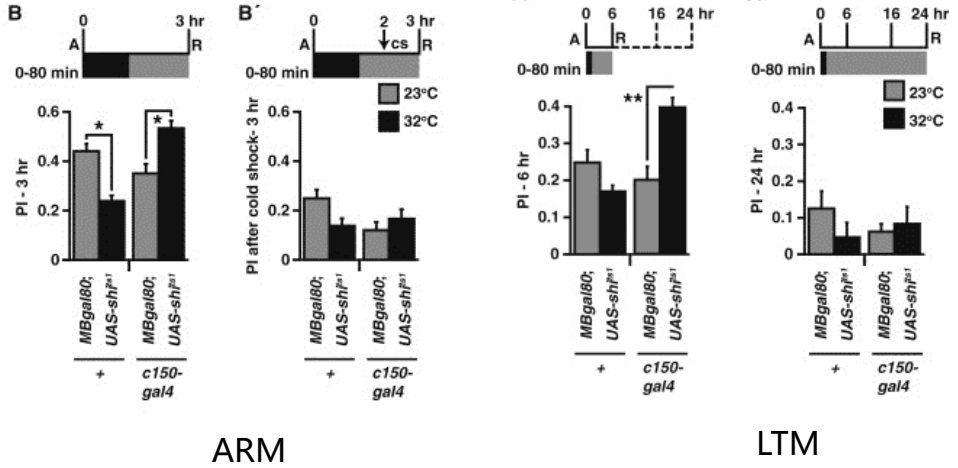
Vogt, Katrin *et al.*, *eLife*, 2014.





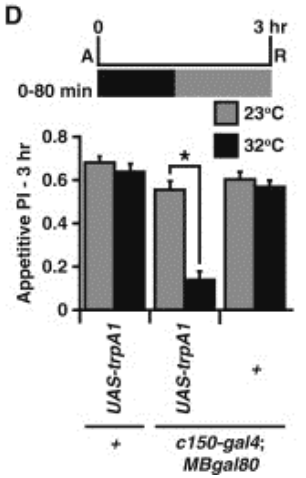
PPL1 DAN promotes the regression of ASM



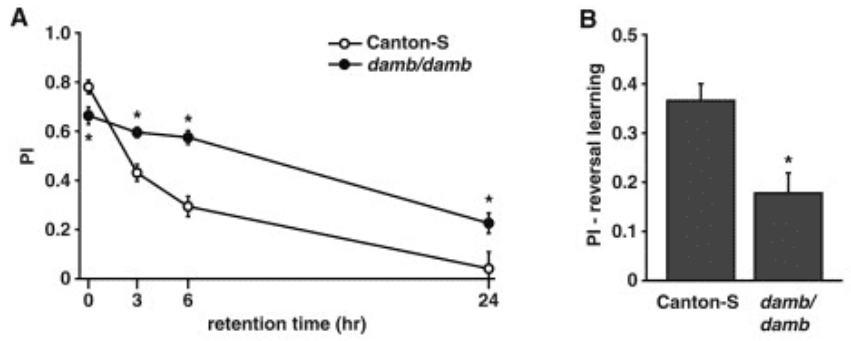


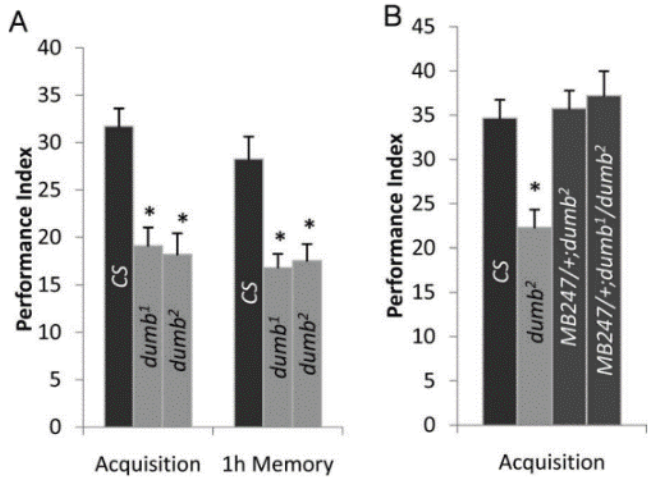
ARM

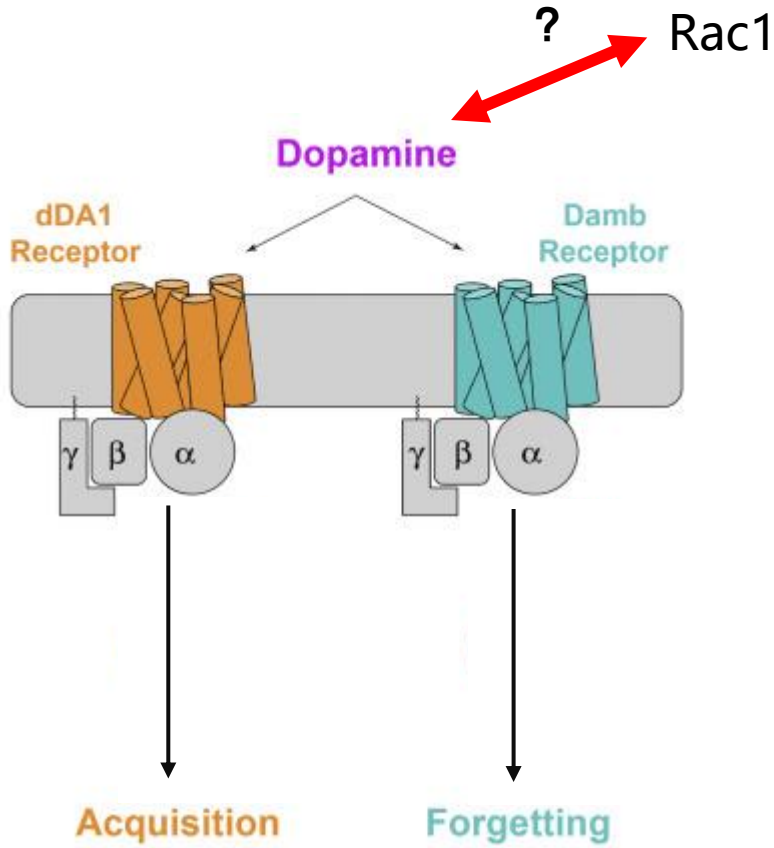
LTM



Two types of dopamine receptors are involved in memory and forgetting respectively

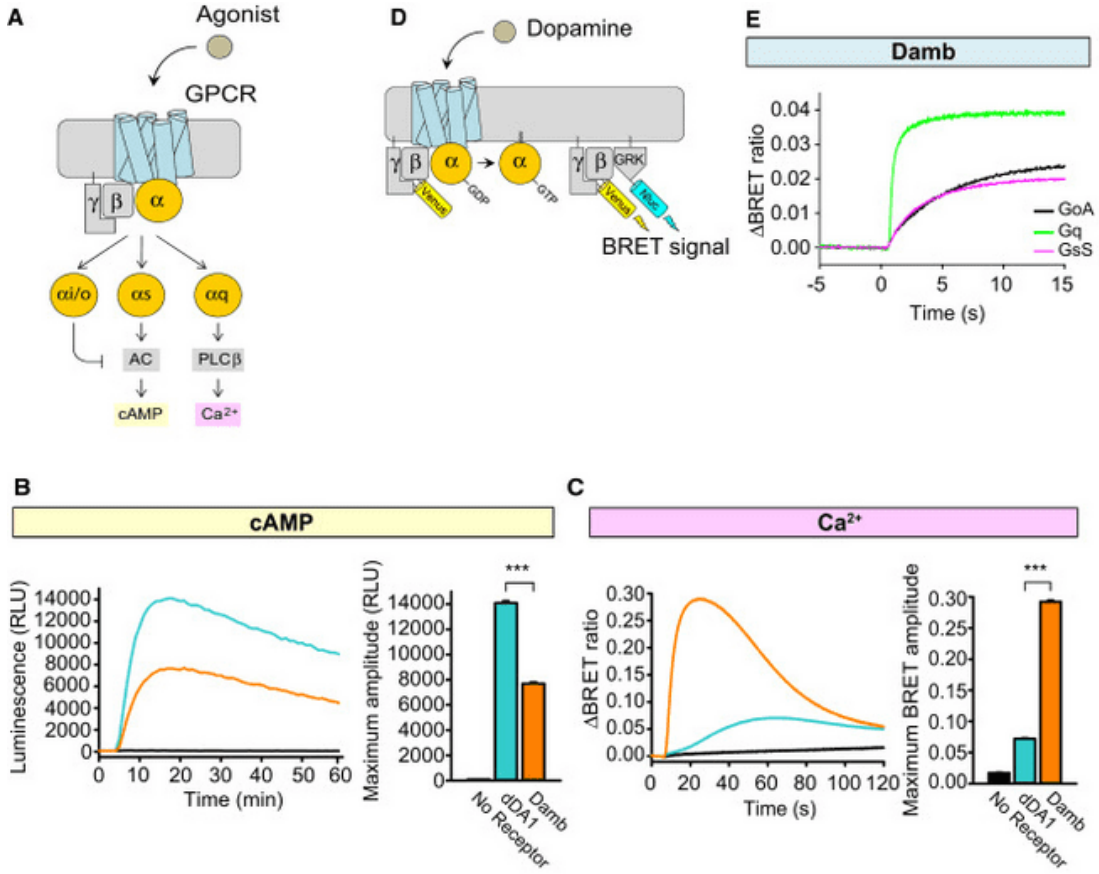






Kim, Young-cho, *et.al.*, *The Journal of Neuroscience*, 2007.

Damb has high affinity with small G protein Gαq



Cell Reports

CellPress

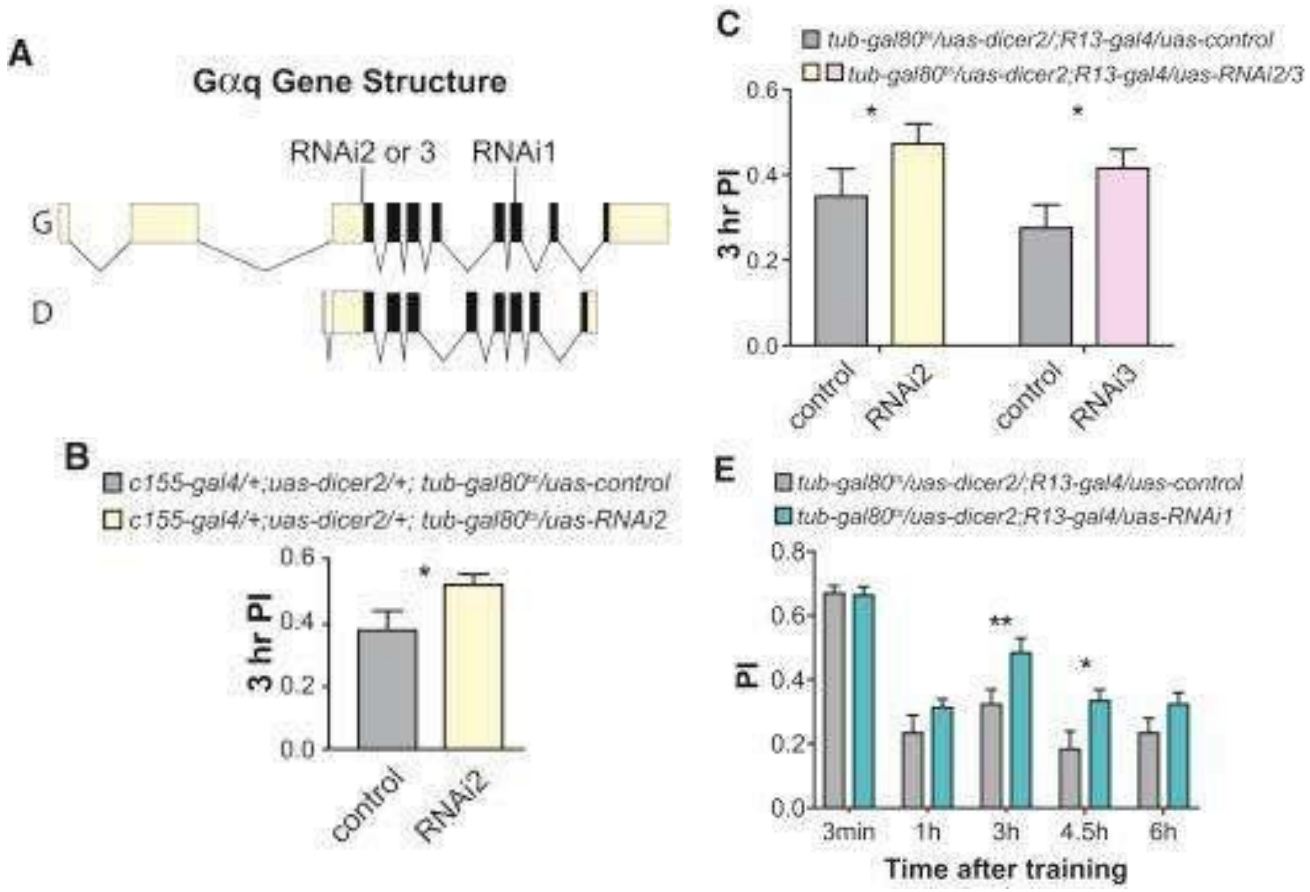
Volume 21, Issue 8, 21 November 2017, Pages 2074-2081

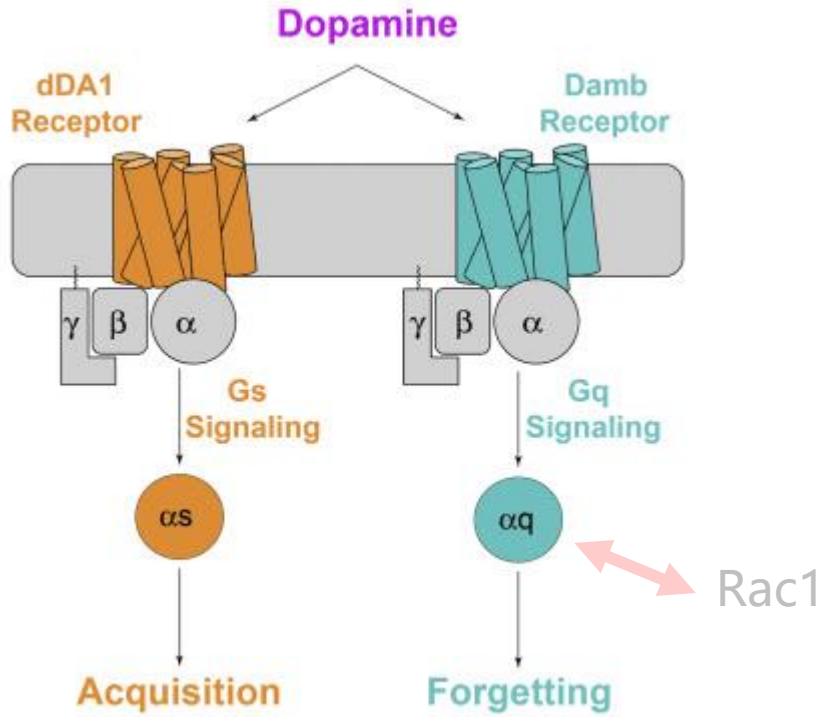
Report

Dopamine Receptor DAMB Signals via Gq to Mediate Forgetting in *Drosophila*

Sophie Himmelreich [1 2], Ikuo Masuho [1 2], Jacob A. Berry [1], Courtney MacMullen [1], Nickolas K. Skamangas [1], Kirill A. Martemyanov [1 3], Ronald L. Davis [1 3 4]

MBNs regulates forgetting through GPCR





- The importance of Rac1 in active forgetting
- Neural circuits involved in forgetting
- How does Rac1 work in neurons

Scaffolding protein promotes forgetting

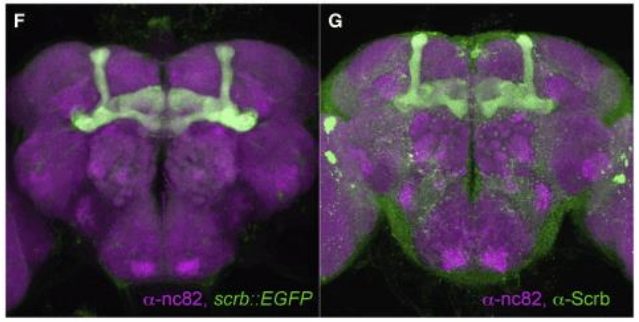
Neuron

Volume 90, Issue 6, 15 June 2016, Pages 1230-1242

Article

Scribble Scaffolds a Signalosome for Active Forgetting

Isaac Cervantes-Sandoval [1], Molee Chakraborty [1], Courtney MacMullen [1], Ronald L. Davis [1]

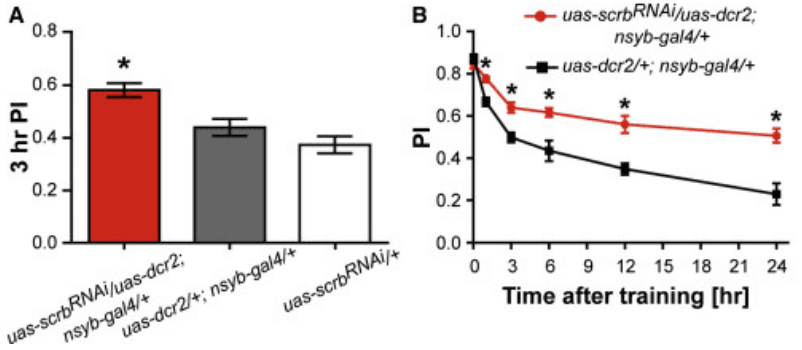


Rac1

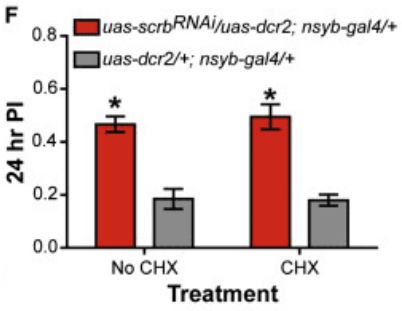
?

Damb

scrb

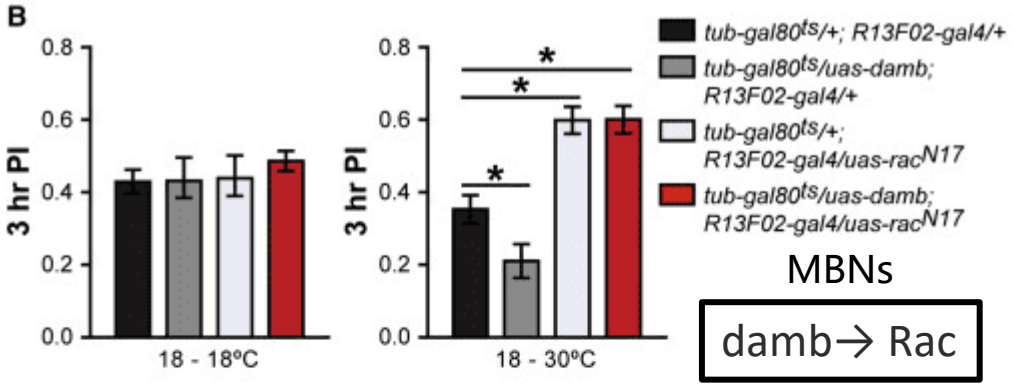


acquisition



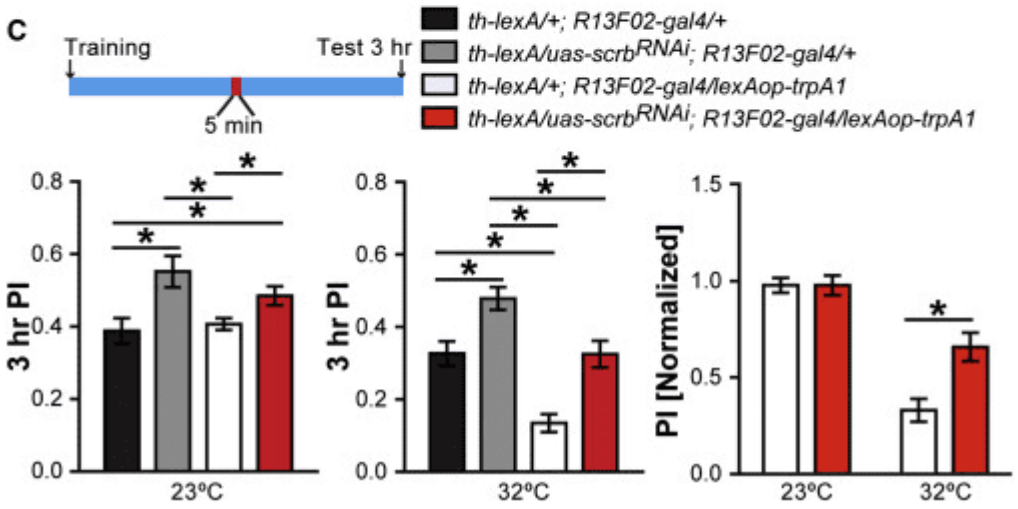
consolidation

The neural circuitry of forgetting

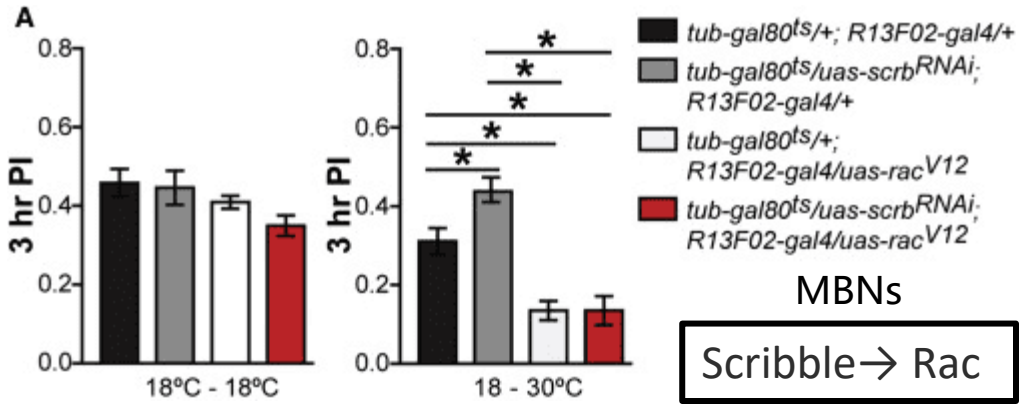


MBNs

damb→ Rac



TH → Dopa → MBNs: Scribble

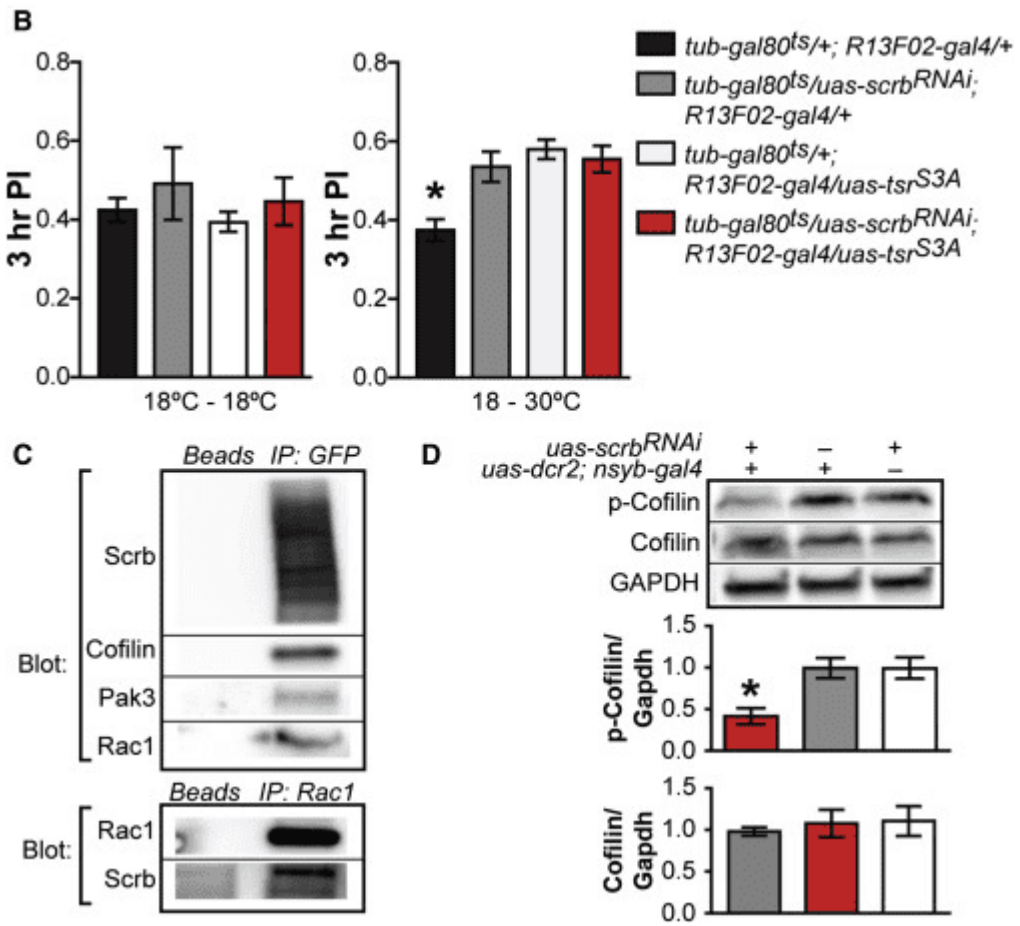


MBNs

Scribble→ Rac

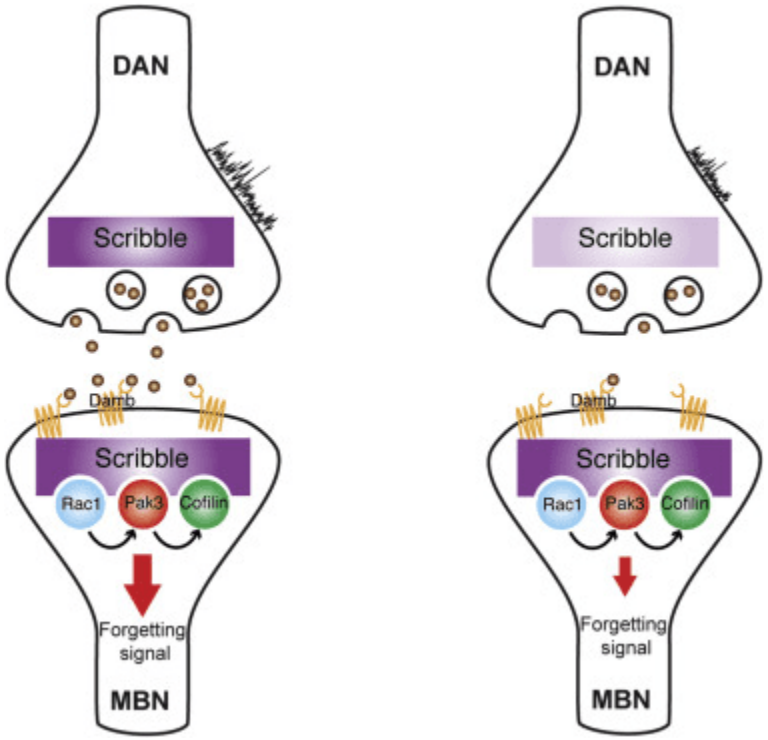
TH → Dopa → MBNs: Damb→Scribble→Rac

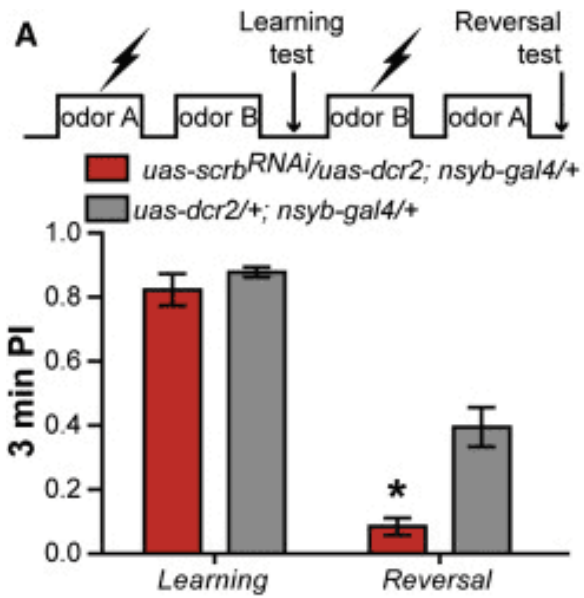
Scribble interacts with Rac1, Pak3, and Cofilin, scaffolding a signalosome for active forgetting



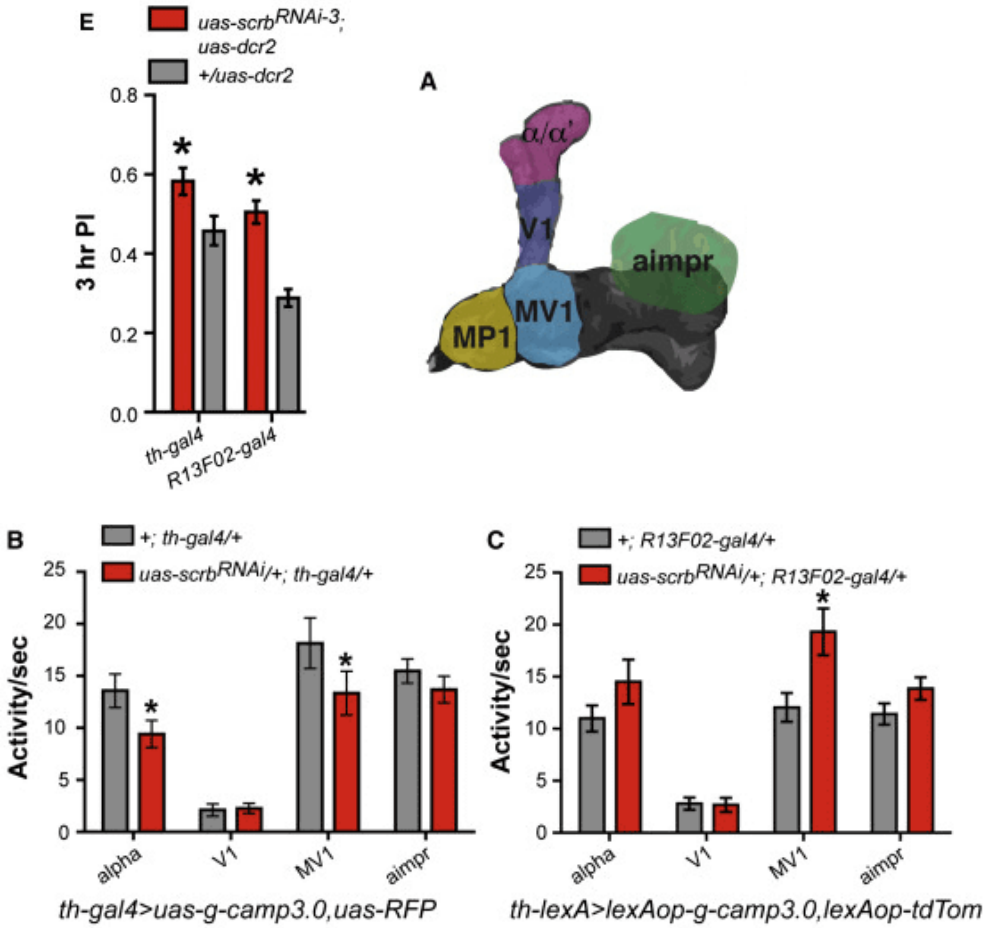
Scribble-Cofilin-Pak3-Rac1

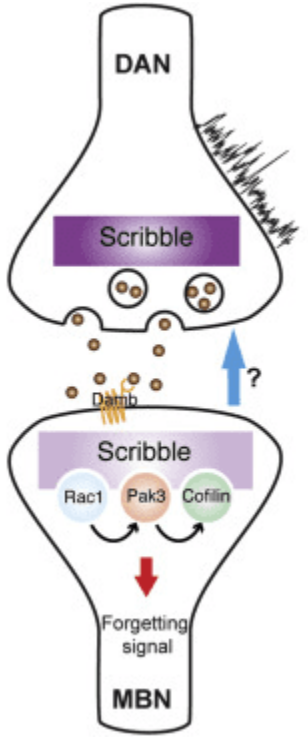
Summary


DAN
Scribble
Damb
Scribble
Rac1
Pak3
Cofilin
Forgetting signal
MBN
DAN
Scribble
Damb
Scribble
Rac1
Pak3
Cofilin
Forgetting signal
MBN


A
Learning test
Reversal test
odor A
odor B
odor B
odor A
uas-scrb^RNAi/uas-dcr2; nsyb-gal4/+
uas-dcr2/+; nsyb-gal4/+
3 min PI
1.0
0.8
0.6
0.4
0.2
0.0
*
Learning
Reversal

Scribble has different functions in DANs and MBNs





Take home messages

- Forgetting is important for the formation of new memories.
- Dopamine can not only mediate olfactory learning and memory, but also participate in active forgetting.
- Recombination of skeletal proteins in MBNs may cause cell imprinting disappear.

Factors affecting forgetting

The potential factors associated forgetting processing

Li Ziqi

2023.03.30

Content

1. How does sleep improve memory?

Dopamine neurons promote active forgetting in the fruit fly's olfactory memory, and sleep suppresses the activity of dopamine neurons, a key factor in the control of forgetting

2. How does social isolation accelerate forgetting of social memory?

Social isolation activated Rac1 protein in the hippocampus of mice, and re-socializing reversed Rac1 protein activation and social memory impairment caused by isolation.

3. How can labile memory be actively protected?

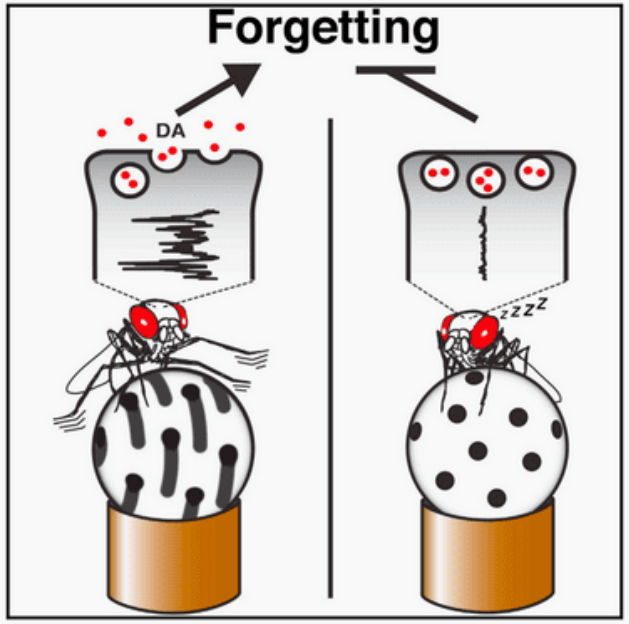
Learning/training itself can actively protect newly formed short-term memory by activating Raf/MAPK signaling pathway to prevent memory from being quickly forgotten due to unexpected interference.

Article

Cell

Sleep Facilitates Memory by Blocking Dopamine Neuron-Mediated Forgetting

Graphical Abstract



Authors

Jacob A. Berry, Isaac Cervantes-Sandoval, Molee Chakraborty, Ronald L. Davis

Correspondence

rdavis@scripps.edu

In Brief

Sleep is generally thought to stabilize new memories, but early psychology studies suggest that it prevents new learning from interfering with old memories. This study shows that sleep suppresses the activity of dopamine neurons that promote active forgetting of olfactory memories in flies, providing integration between neuroscience and psychology research.

睡眠如何促进记忆?

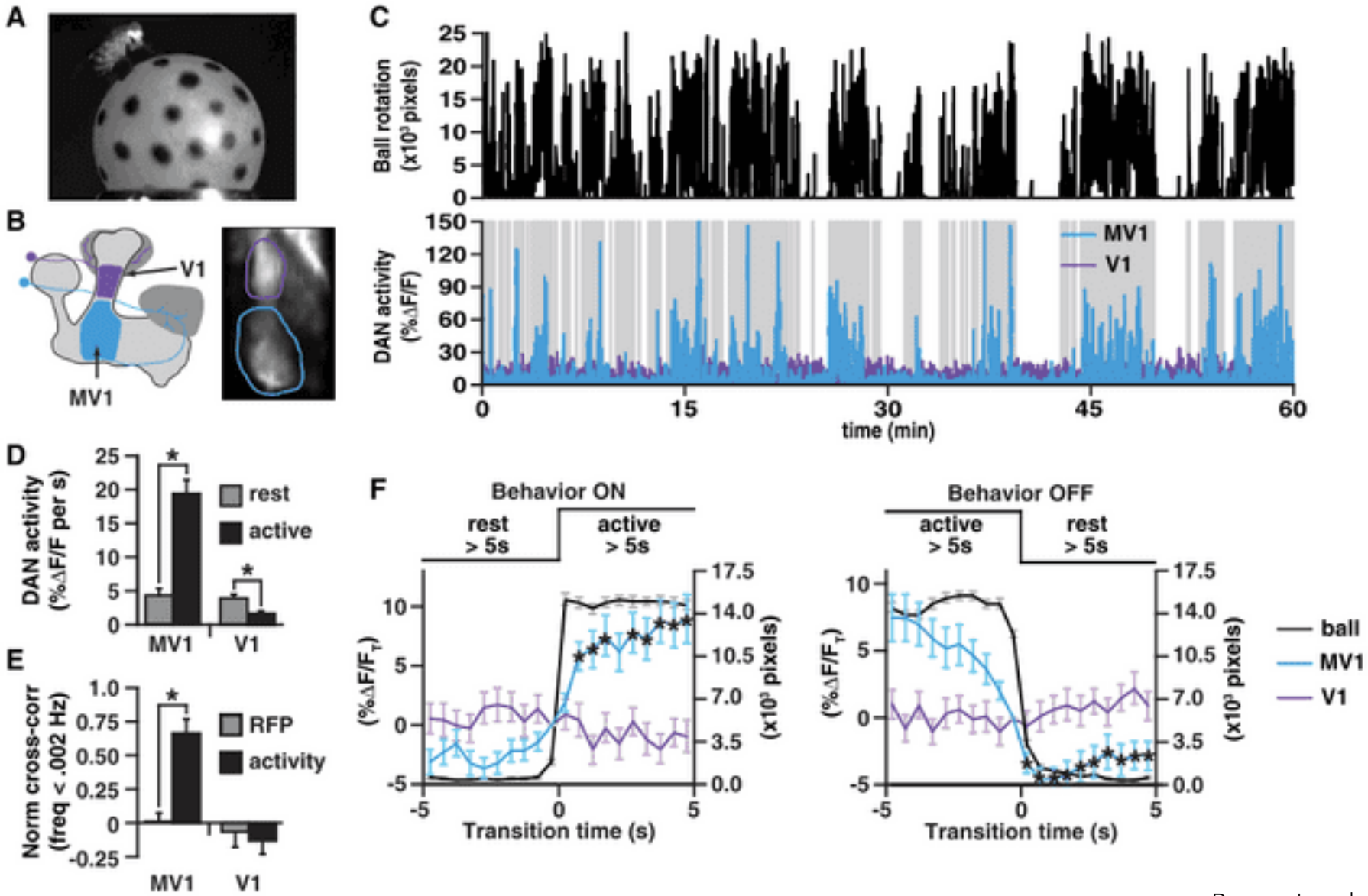
1.Develop mitochondrial therapeutics.Mitochondrial dysfunction is a major hallmark of most neurodegenerative disorders, including Alzheimer's disease (AD) and amyotrophic lateral sclerosis (ALS).

2.Uncover the brain mechanisms that lead to forgetting.

Berry, Jacob A et al. Cell.(2015)

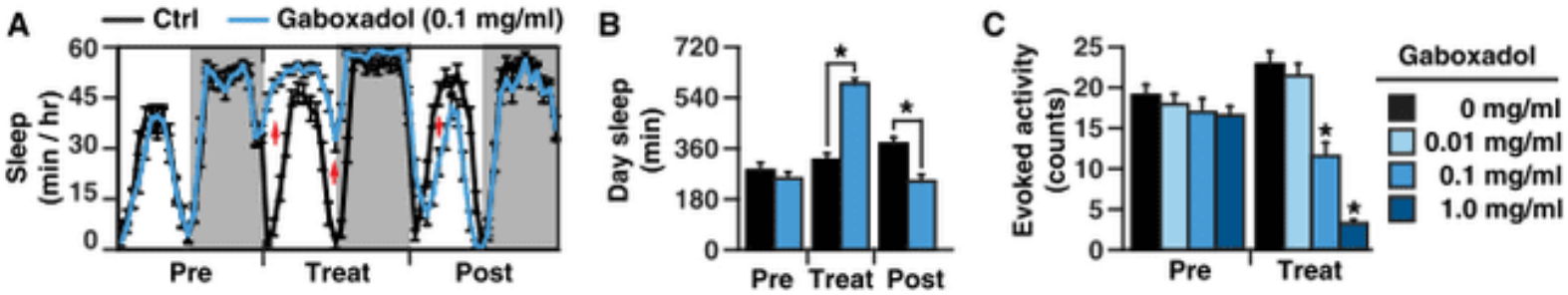
1.Ongoing Dopamine Neuron Activity Is Regulated by Behavioral State

two regions of the DAN processes that form synaptic connections to the MBs



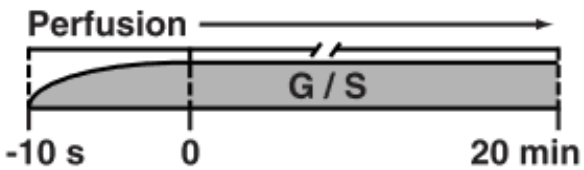
Berry, Jacob A et al. Cell.(2015)

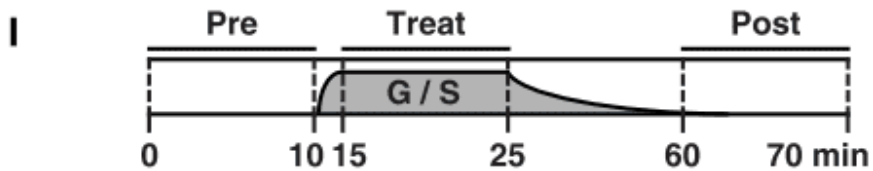
2.Sleep Reduces Ongoing DAN Activity——Gaboxadol

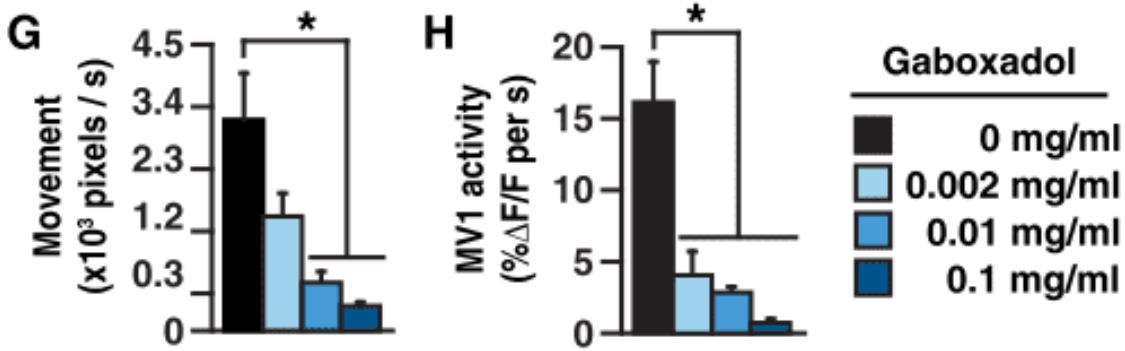


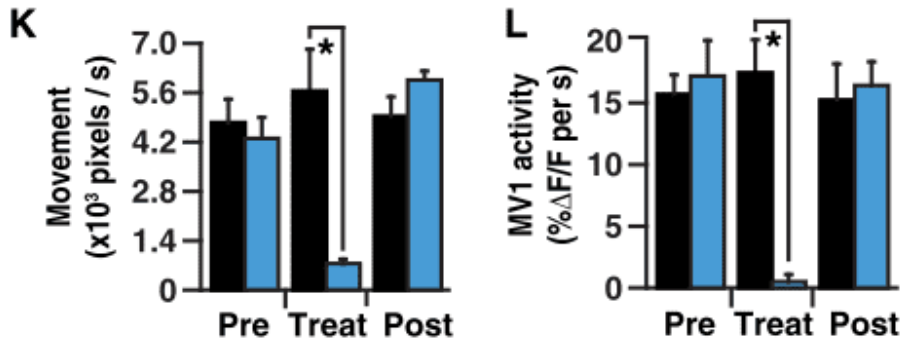
可逆静止
稳态调节
唤醒阈值增加

加博沙多诱导果蝇真正的睡眠



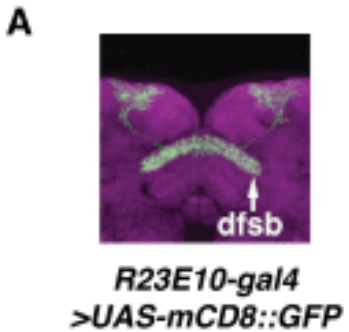




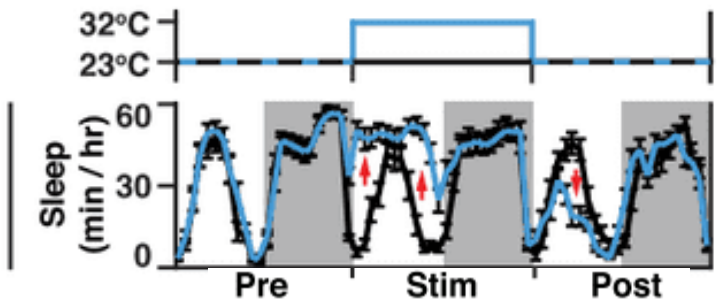


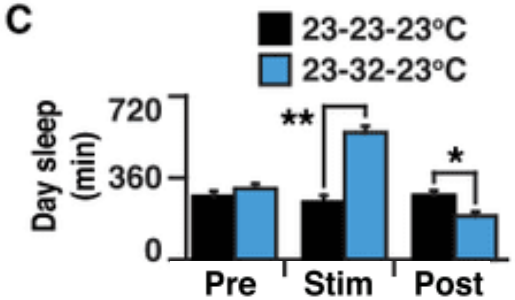
Berry, Jacob A et al. Cell.(2015)

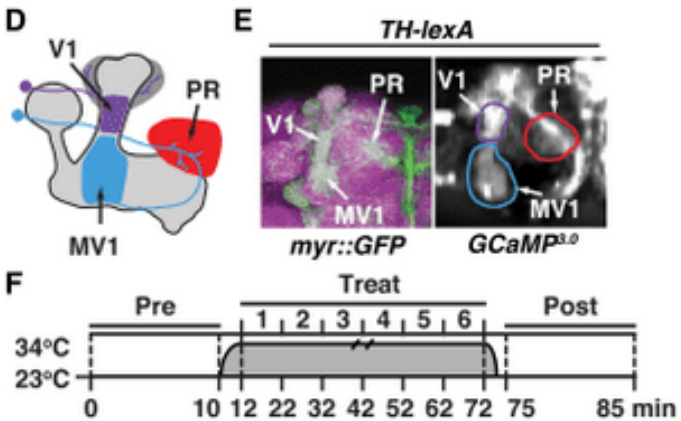
2.Sleep Reduces Ongoing DAN Activity——Stimulate the sleep neural circuit

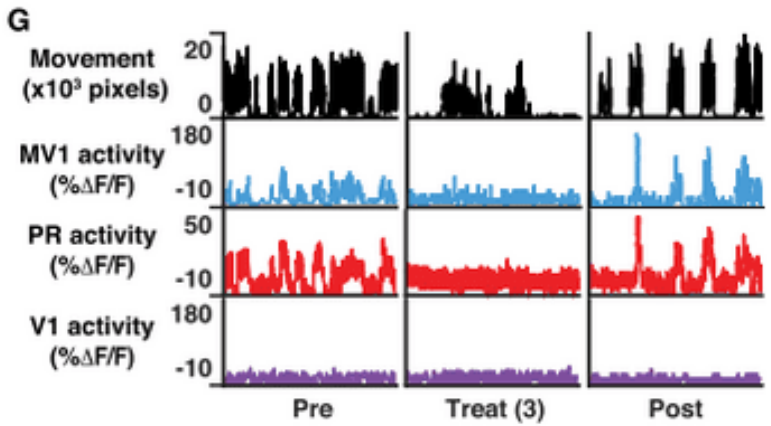


中央大脑中的背扇状体(DFSB)回路

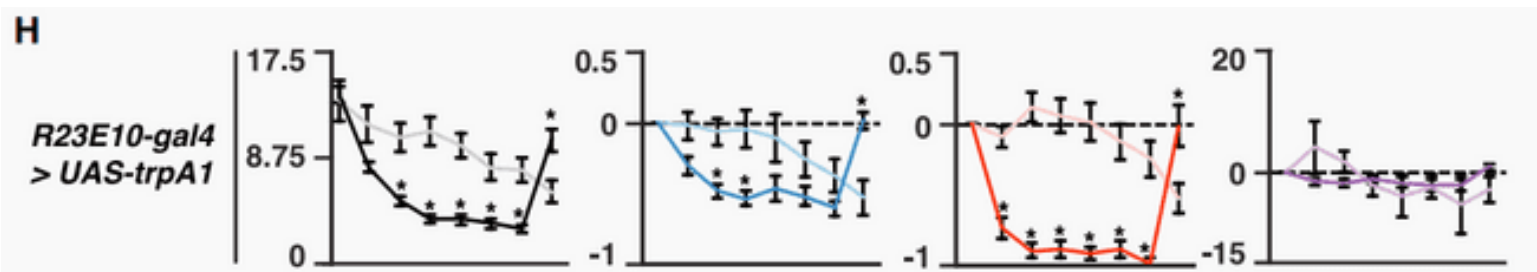








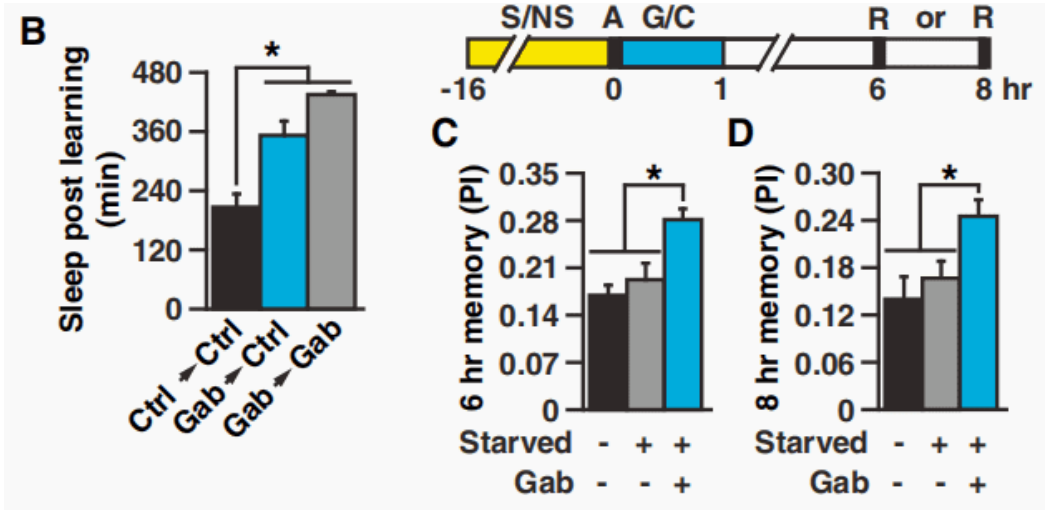
Sleep Circuit Stimulation Induces Sleep and Rapidly and Reversibly Eliminates Movement and Ongoing DAN Activity



(—or—) Movement (x10³ pixels/s)
(—or—) MV1 activity (Relative change to pre)
(—or—) PR activity (Relative change to pre)
(—or—) V1 activity (Relative change to pre)

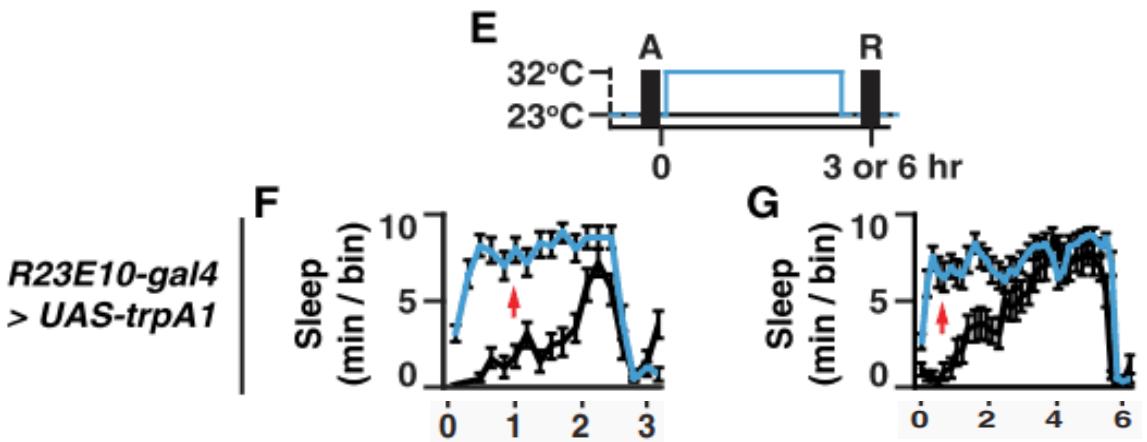
Berry, Jacob A et al. Cell.(2015)

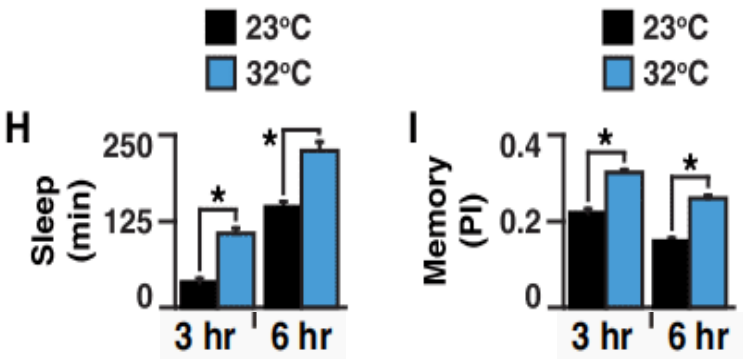
3.Increased Sleep after Learning Impairs Forgetting



Gaboxadol

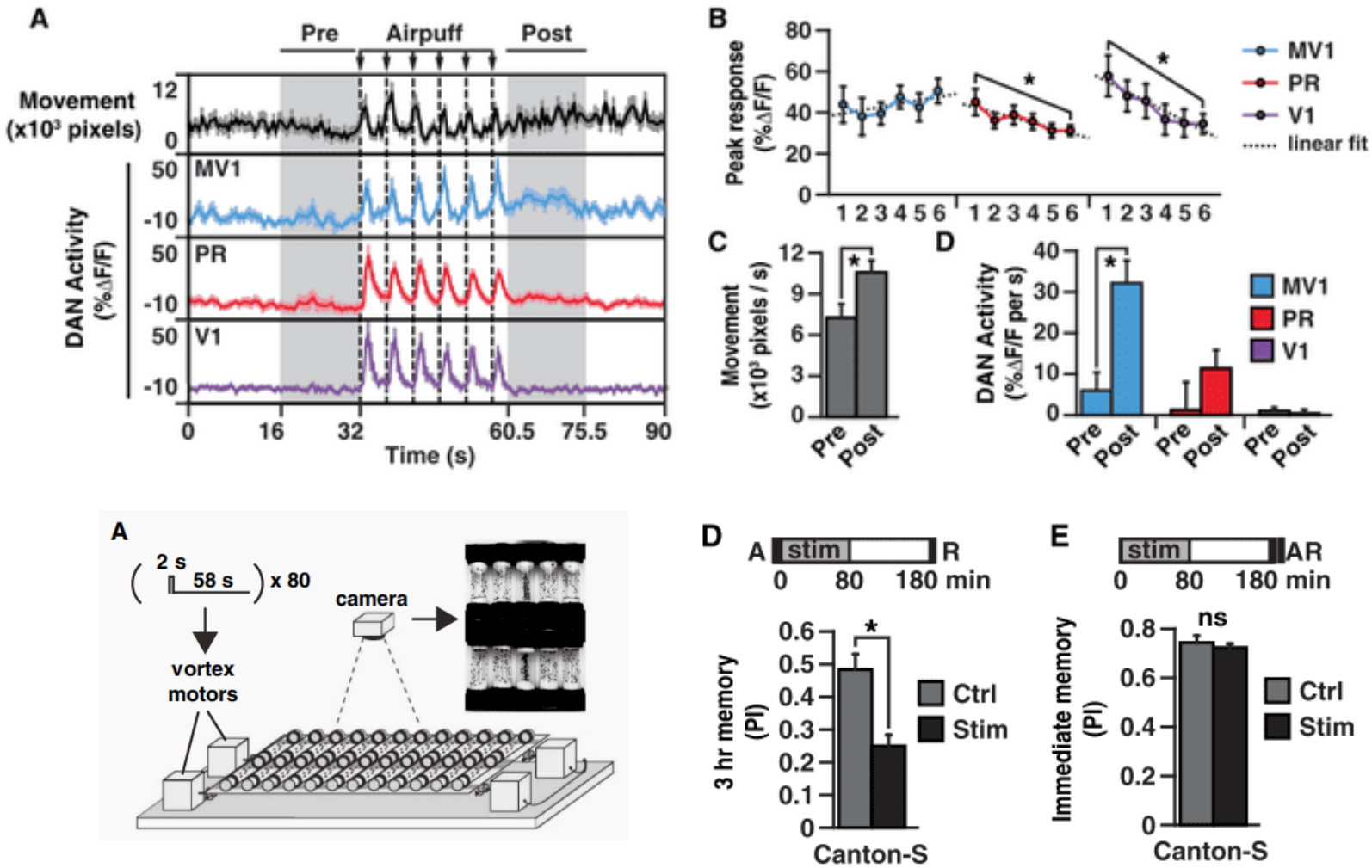
Stimulate the sleep neural circuit





Berry, Jacob A et al. Cell.(2015)

4.Increased Arousal after Learning Increases Forgetting

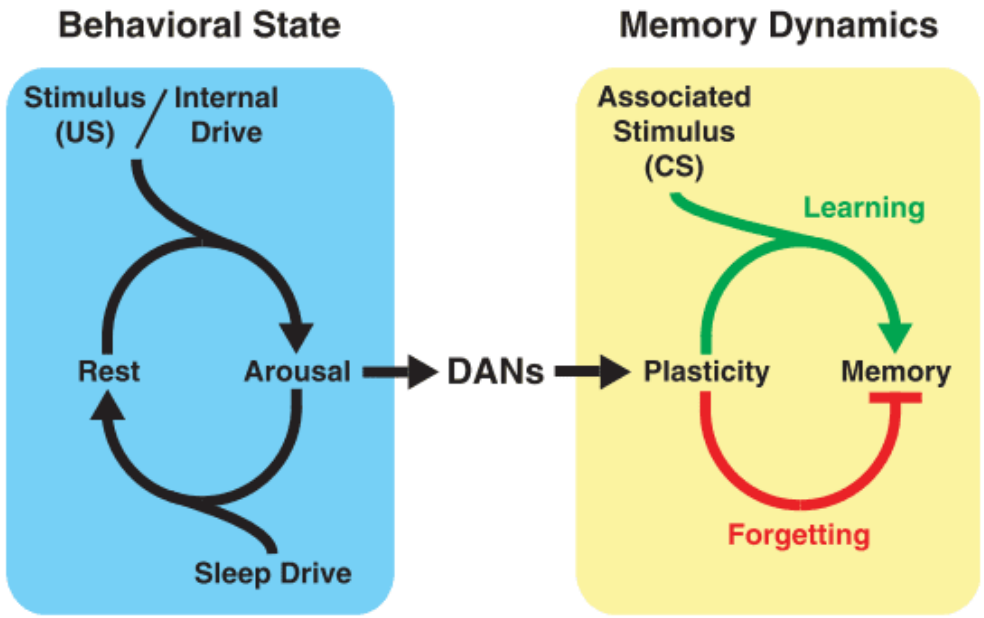


Berry, Jacob A et al. Cell.(2015)

Summary

1.睡眠如何促进记忆?

多巴胺神经元促进果蝇嗅觉记忆的主动遗忘，睡眠抑制了多巴胺神经元的活动，多巴胺是控制遗忘的关键因子



动物不断地在休息和觉醒之间转换行为状态。 内驱力或外部刺激，使动物进入被唤醒和活跃的状态，进而激活支配MB记忆中心的DAN。

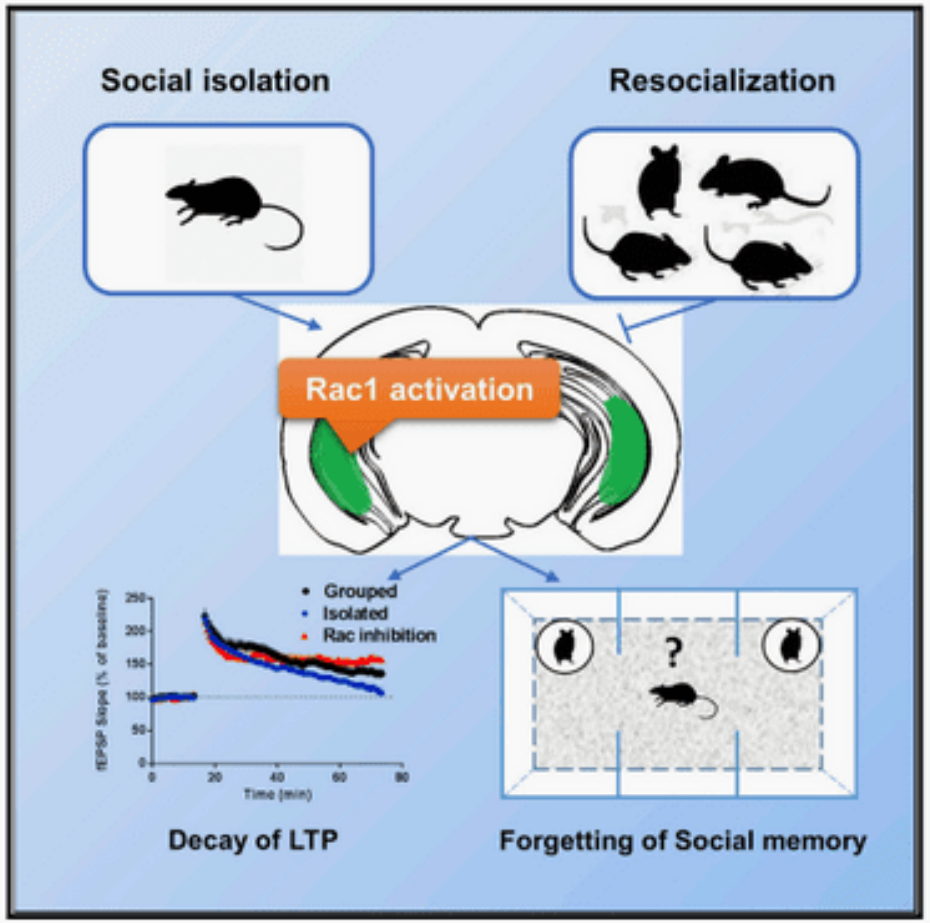
认为唤醒诱导的DAN激活增加了MB神经元的可塑性，以促进记忆更新。

睡眠通过将行为状态从唤醒中转移出来，从而减少DAN介导的可塑性和更新，从而有利于记忆的保持。

Cell Reports

Social Isolation Induces Rac1-Dependent Forgetting of Social Memory

Graphical Abstract



Authors

Yunlong Liu, Li Lv, Lianzhang Wang, Yi Zhong

Correspondence

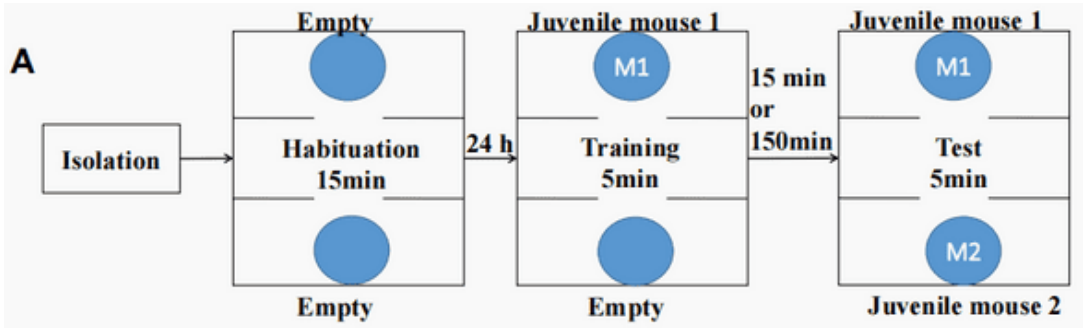
zhongyi@tsinghua.edu.cn

In Brief

Liu et al. identify a Rac1-dependent forgetting pathway that mediates isolation-induced memory impairment. Such findings underscore the importance of maintained social interactions on cognitive function, which may have implications for autism and Alzheimer's disease.

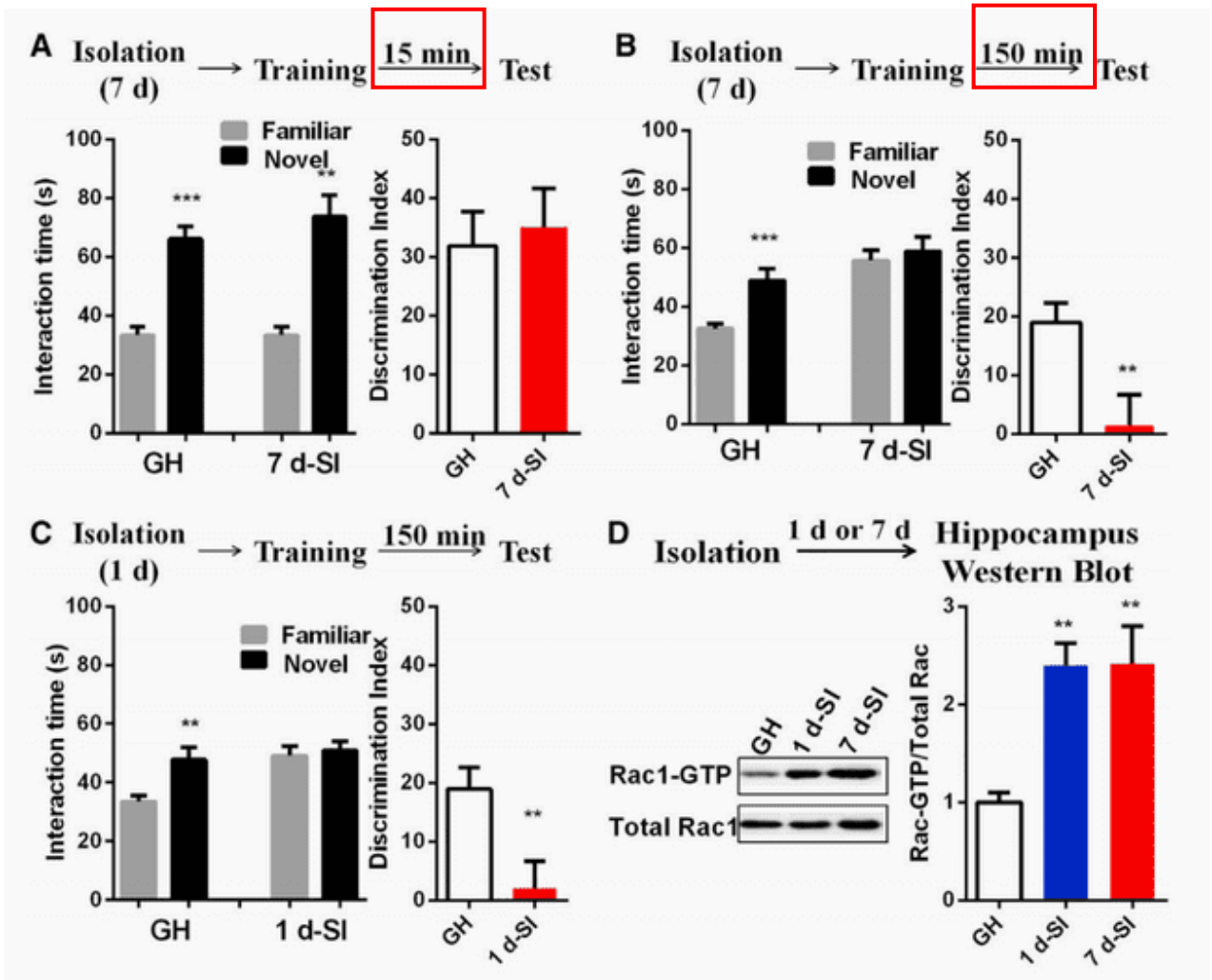
社交隔离如何导致社交记忆加速遗忘?

1.Social Isolation Induced a Rapid Decay of SRM through Increased Hippocampal Rac1 Activity



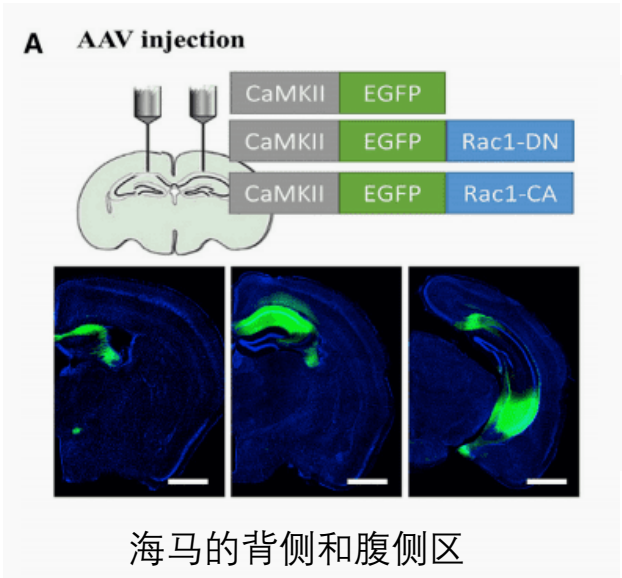
social recognition memory (SRM)

social discrimination paradigm

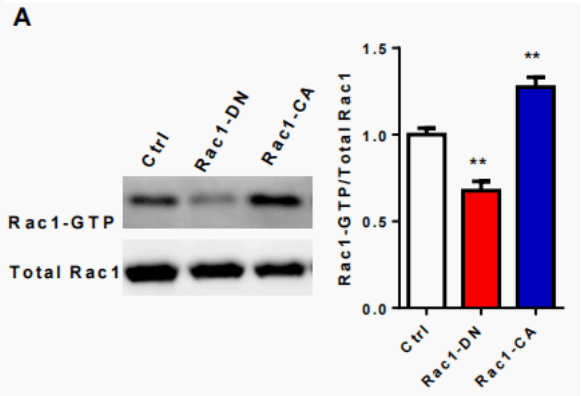


Liu, Yunlong et al. Cell reports.(2018)

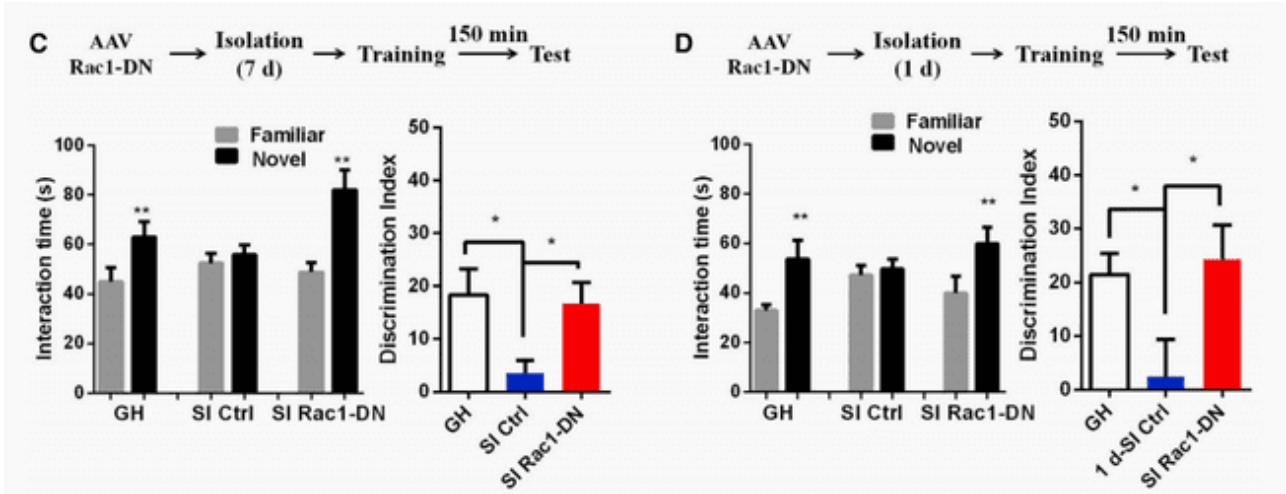
Manipulation of Rac1 Activity Affects the Retention, but Not the Acquisition, of Social Memory



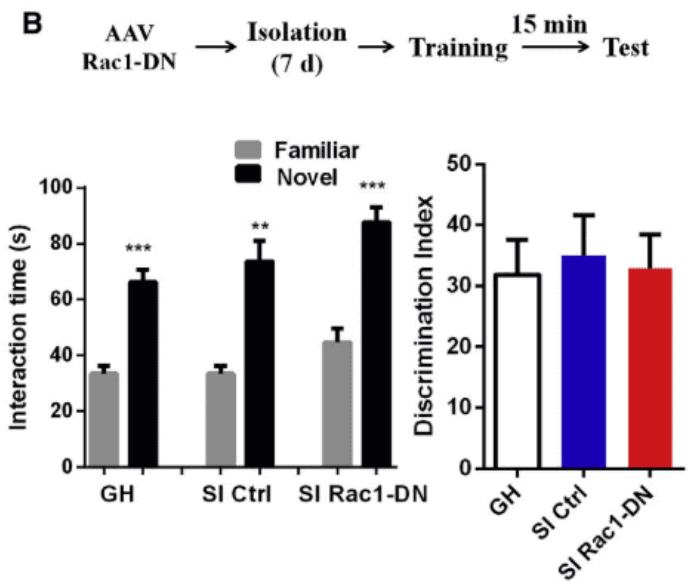
海马的背侧和腹侧区



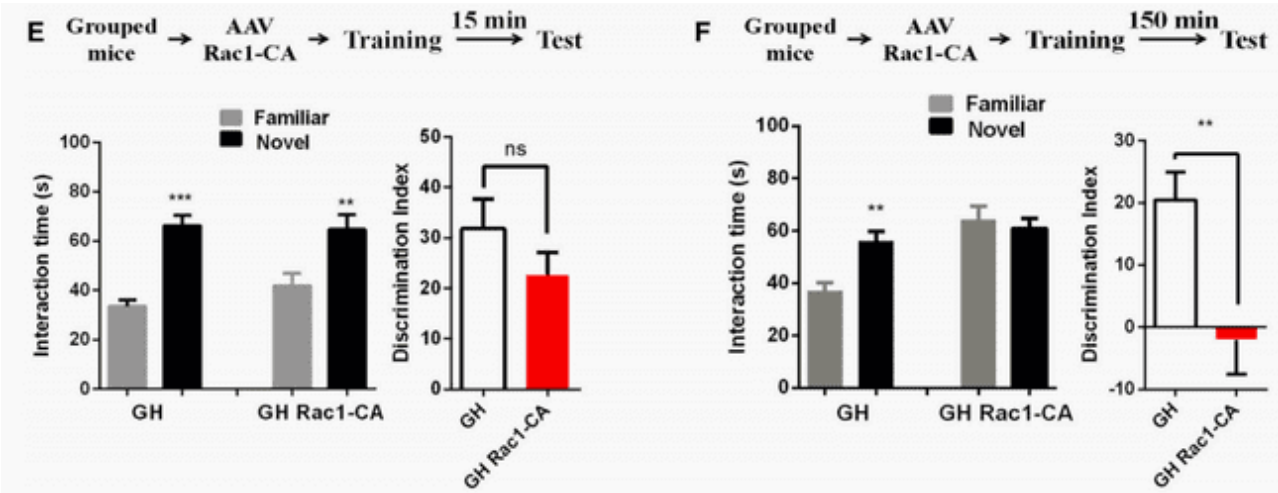
Rac1-DN：抑制Rac1活性
Rac1-CA：增加Rac1活性



Rac1-DN注射的小鼠修复了150分钟社交记忆受损



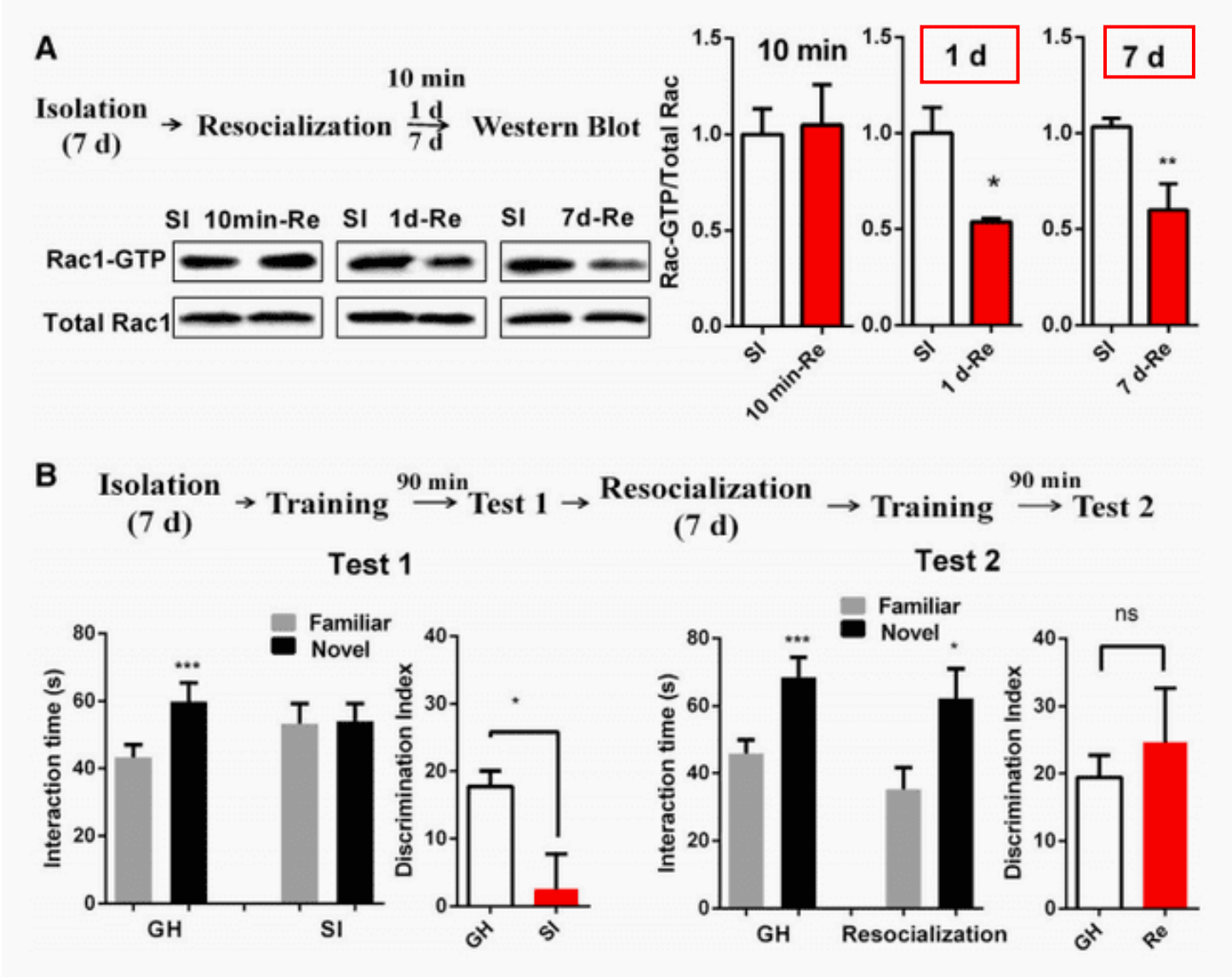
抑制 Rac1 活性对孤立小鼠的社交记忆形成没有影响



AAV-RAC1-CA注射对群养小鼠社会记忆的形成没有影响.
但导致了群养小鼠社交记忆的快速遗忘

Liu, Yunlong et al. Cell reports.(2018)

2.Resocialization Reversed Isolation-Induced Memory Defects and Elevated Rac1-Activity



Liu, Yunlong et al. Cell reports.(2018)

Summary

社交隔离如何导致社交记忆加速遗忘?

1.社交隔离激活了小鼠海马区Rac1蛋白。

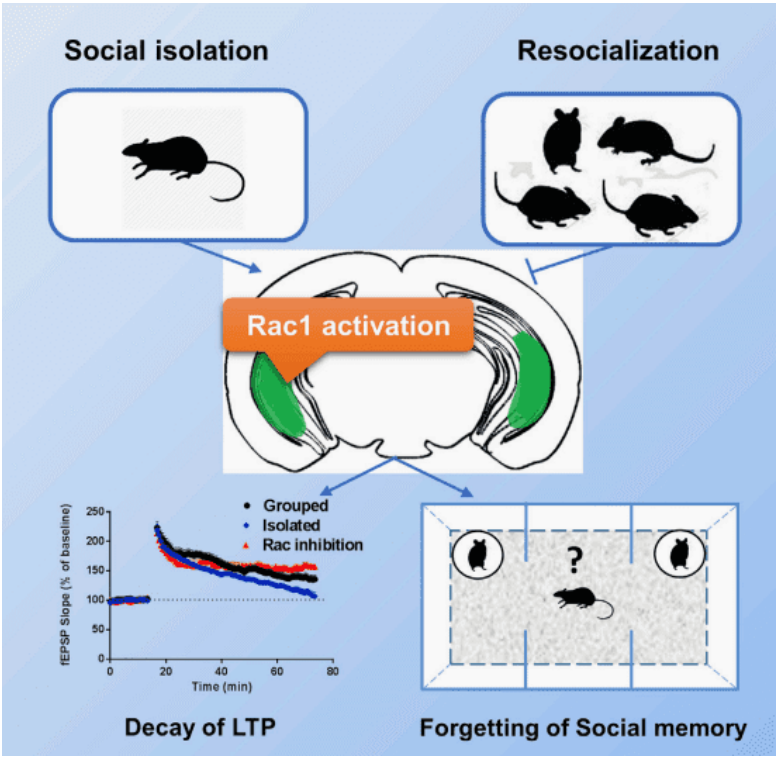
2.通过抑制小鼠海马区Rac1蛋白活性可以阻止隔离导致的记忆障碍，而激活Rac1蛋白则加速小鼠社交识别记忆的遗忘。

3.重新社交活动可以逆转隔离导致的Rac1蛋白激活以及社交记忆障碍。

4.抑制Rac1蛋白活性挽救社交隔离导致的长时程突触增强（LTP）缺陷。

该研究提示社交活动可以保护记忆，

为社交隔离导致的人类认知障碍提供了一定线索。

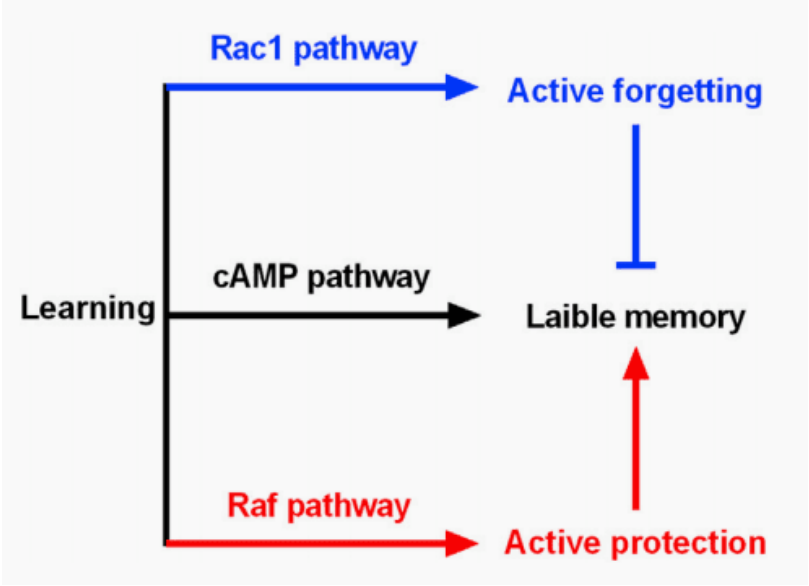


CellPress

Neuron
Article

Active Protection: Learning-Activated Raf/MAPK Activity Protects Labile Memory from Rac1-Independent Forgetting

Xuchen Zhang,[1,4] Qian Li,[1,4,*] Lianzhang Wang,[1] Zhong-Jian Liu,[2,3] and Yi Zhong[1,5,*]
[1]Tsinghua-Peking Center for Life Sciences, IDG/McGovern Institute for Brain Research, MOE Key Laboratory of Protein Sciences, School of Life Sciences, Tsinghua University, Beijing 100084, China
[2]Shenzhen Key Laboratory for Orchid Conservation and Utilization, The National Orchid Conservation Center of China and The Orchid Conservation and Research Center of Shenzhen, Shenzhen 518114, China
[3]The Center for Biotechnology and Biomedicine, Graduate School at Shenzhen, Tsinghua University, Shenzhen 518055, China
[4]These authors contributed equally
[5]Lead Contact
*Correspondence: lqnirvana@gmail.com (Q.L.), zhongyi@tsinghua.edu.cn (Y.Z.)
https://doi.org/10.1016/j.neuron.2018.02.025

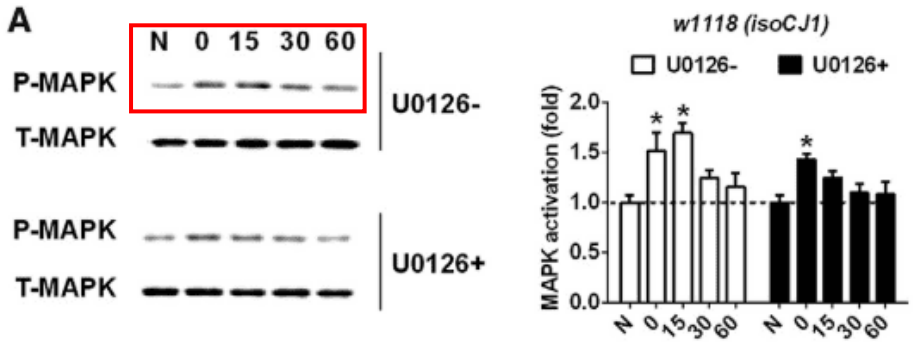


不稳定记忆如何被主动保护?

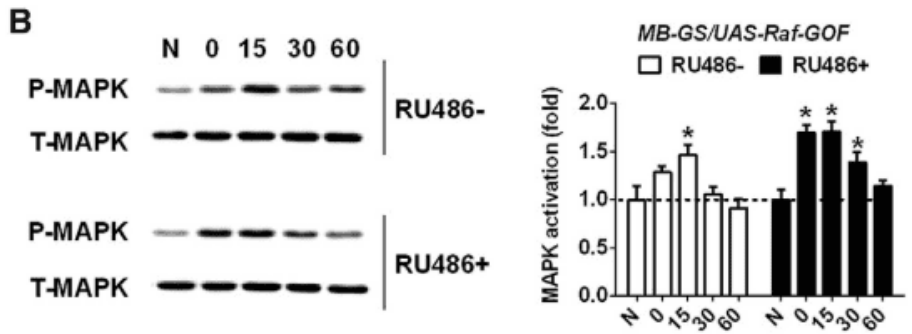
Zhang, Xuchen et al.Neuron.(2018)

1.Learning Activates Raf/MAPK Activity in a Brain Region Where Labile Memory Is Formed

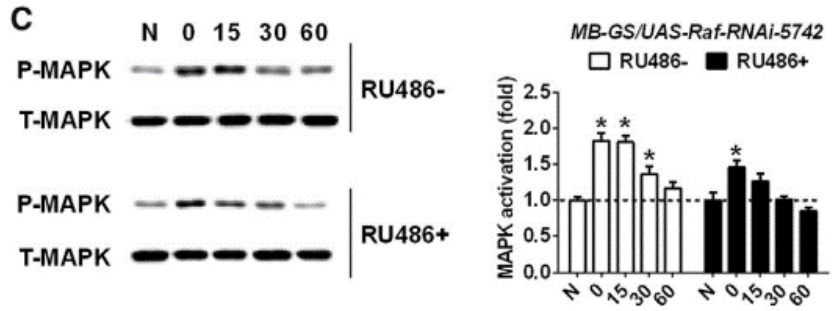
pharmacological and genetic manipulations of Raf/MAPK activity

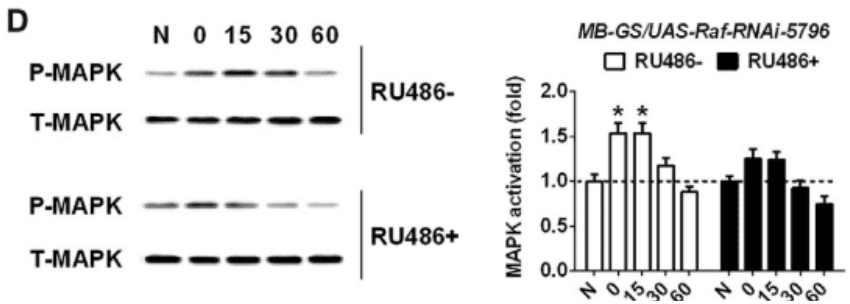


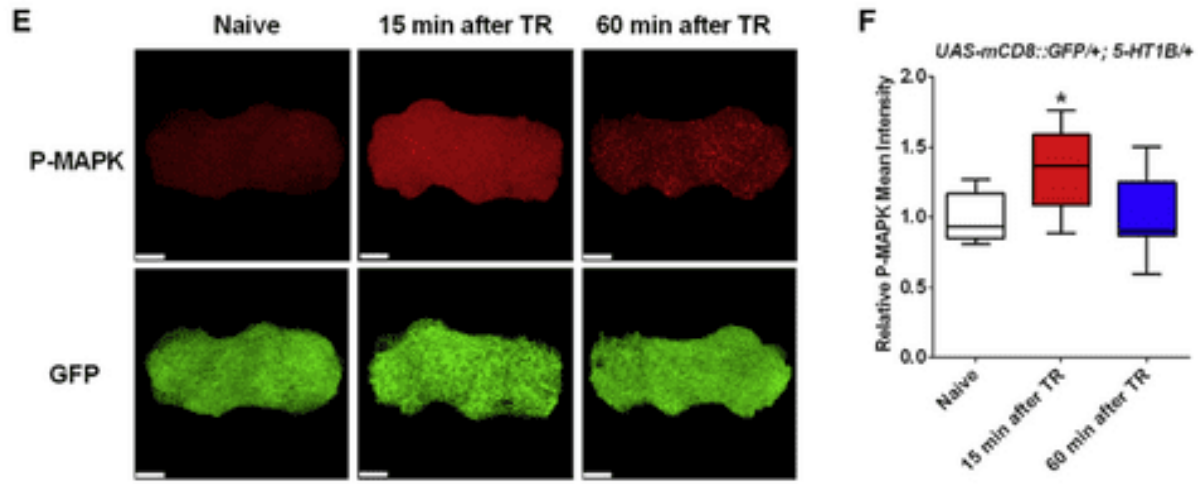
U0126： MAPK 通路抑制剂，损害长期记忆



表达转基因 UAS-RafGOF，编码活性 Raf 激酶。通过喂食 RU486 来实现



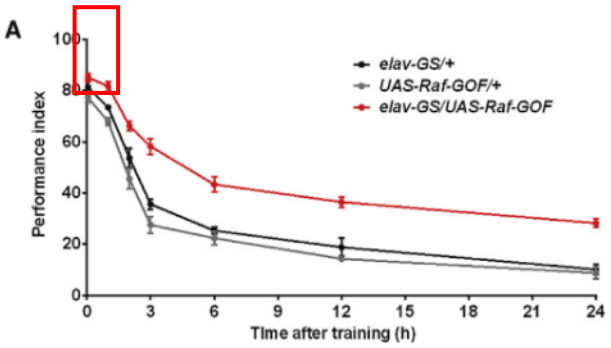




MB γ叶是获得厌恶性嗅觉不稳定记忆的关键部位

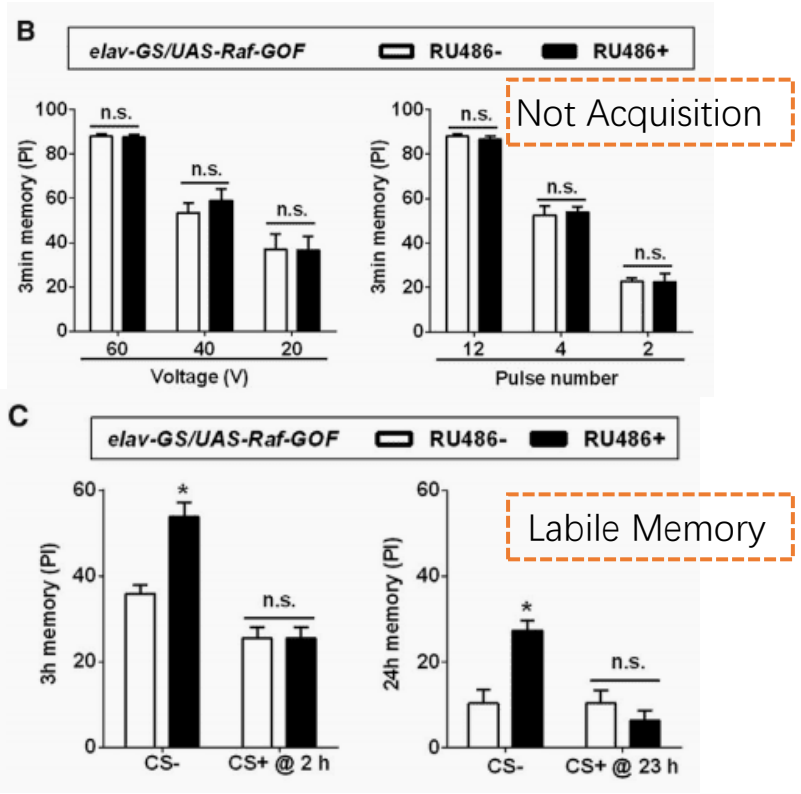
Zhang, Xuchen et al.Neuron.(2018)

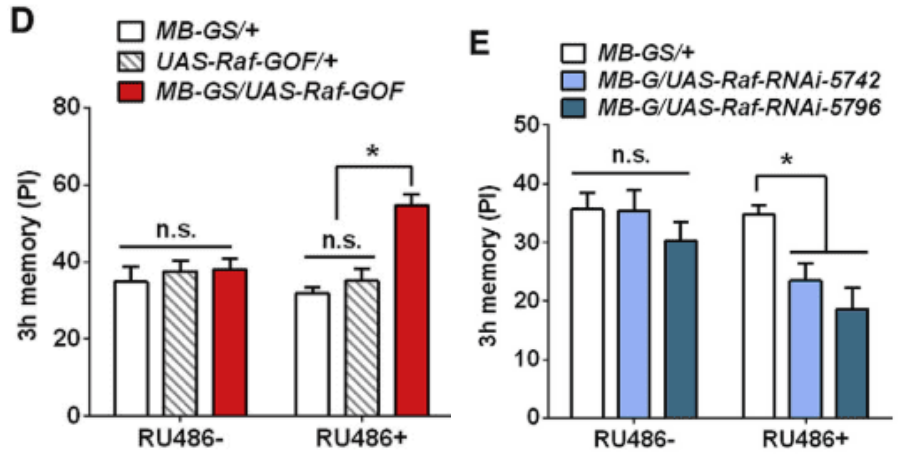
2.Labile Memory Retention, but Not Acquisition, Is Bidirectionally Regulated by Raf/MAPK Activity in γ MB Neurons

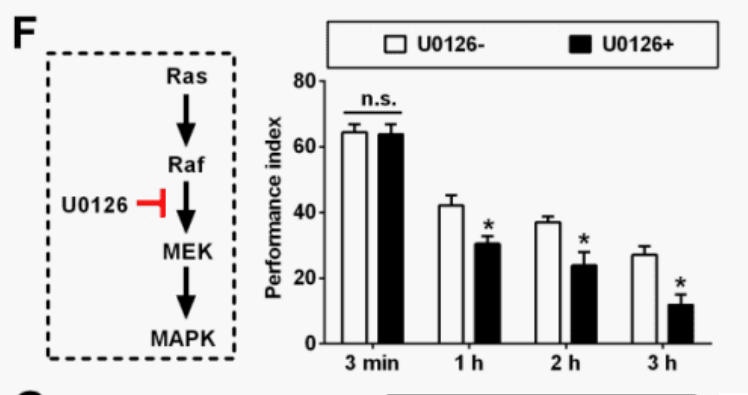


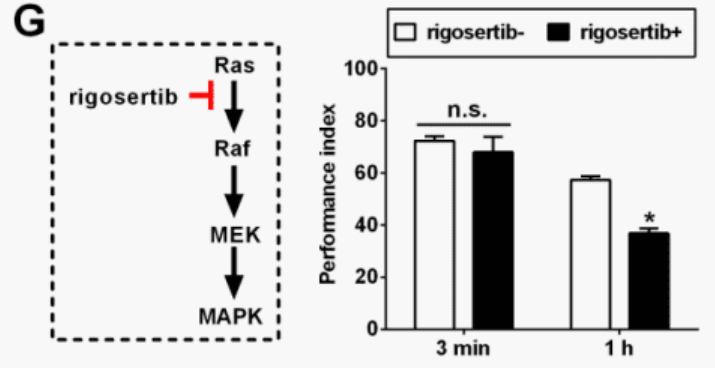
表达RAF-GOF表现出较慢的记忆衰减和相似的记忆获得

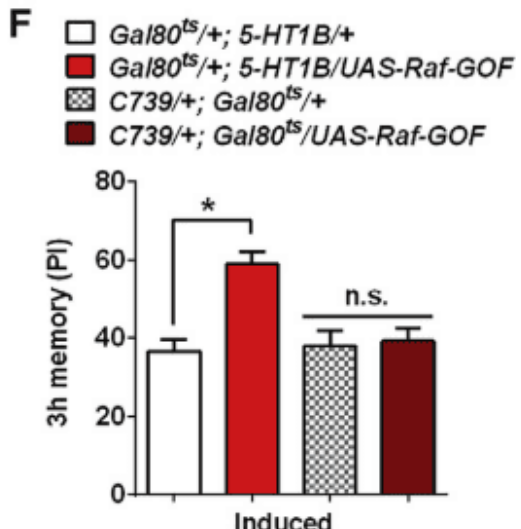
γ MB Neurons

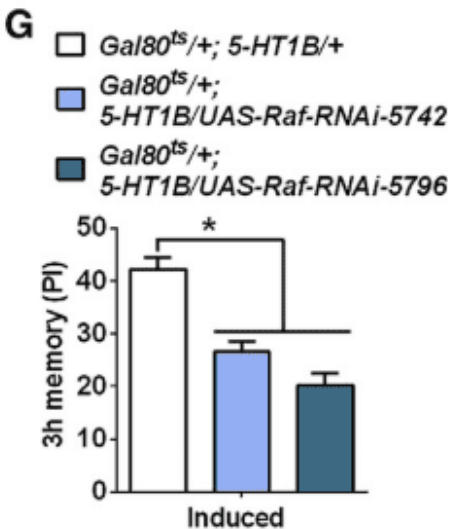


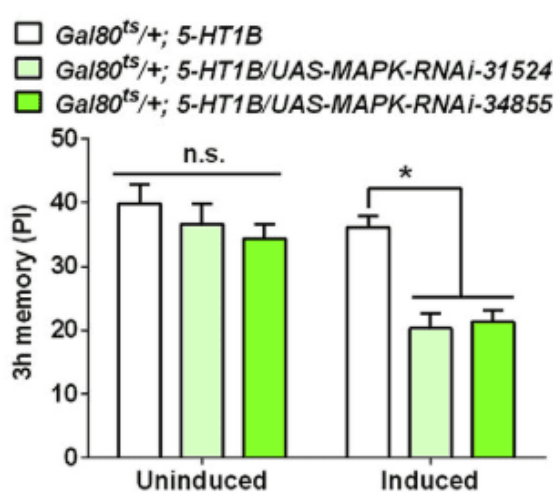






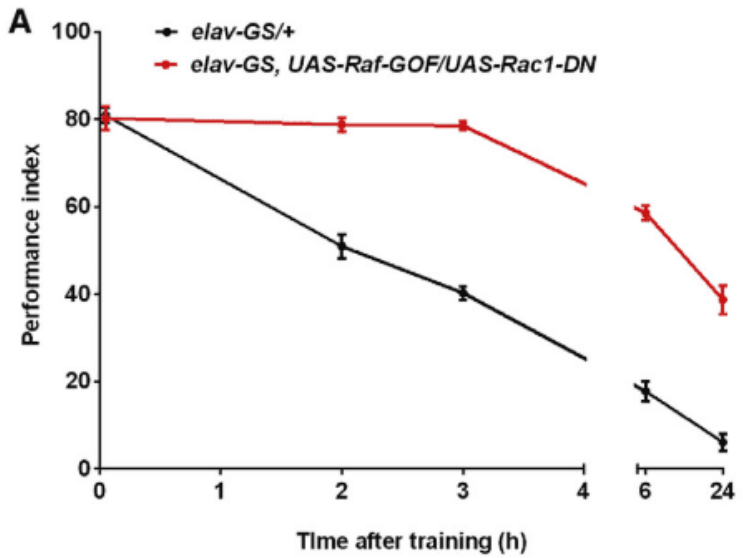


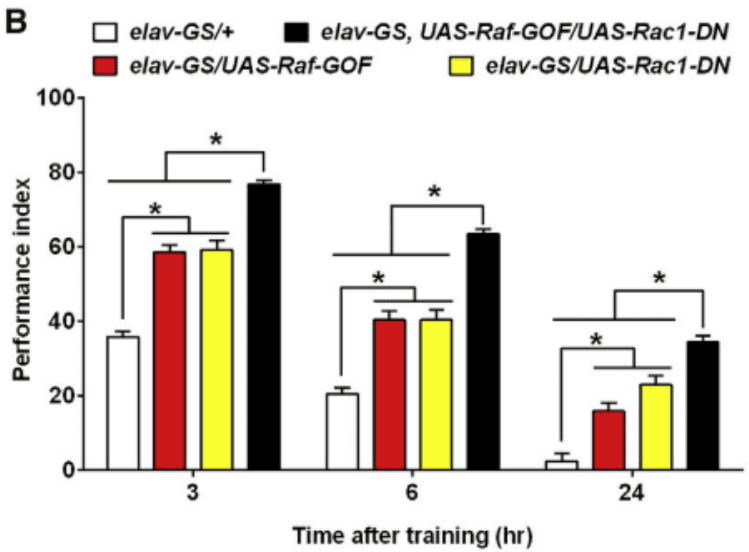


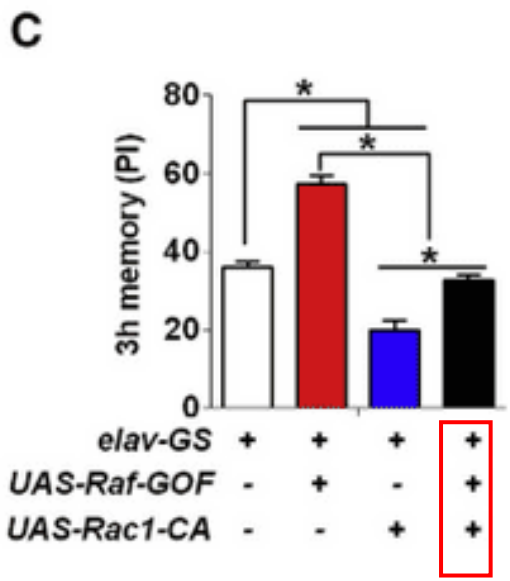


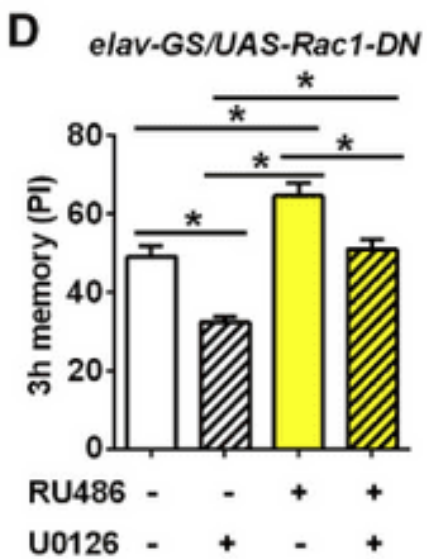
Zhang, Xuchen et al.Neuron.(2018)

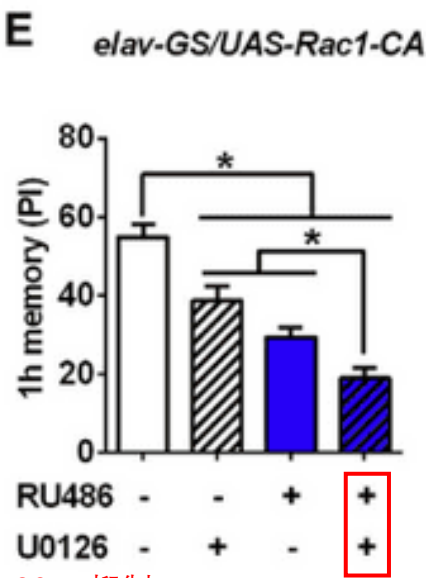
3.Raf-Suppressed Forgetting Is Distinct from Rac1-Mediated Active Forgetting

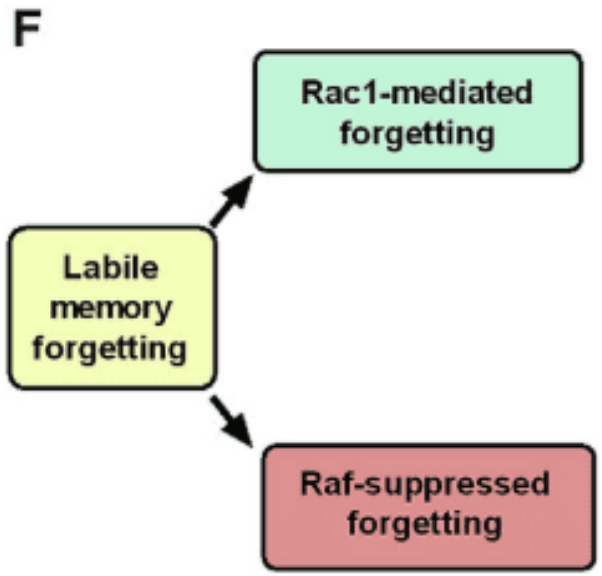








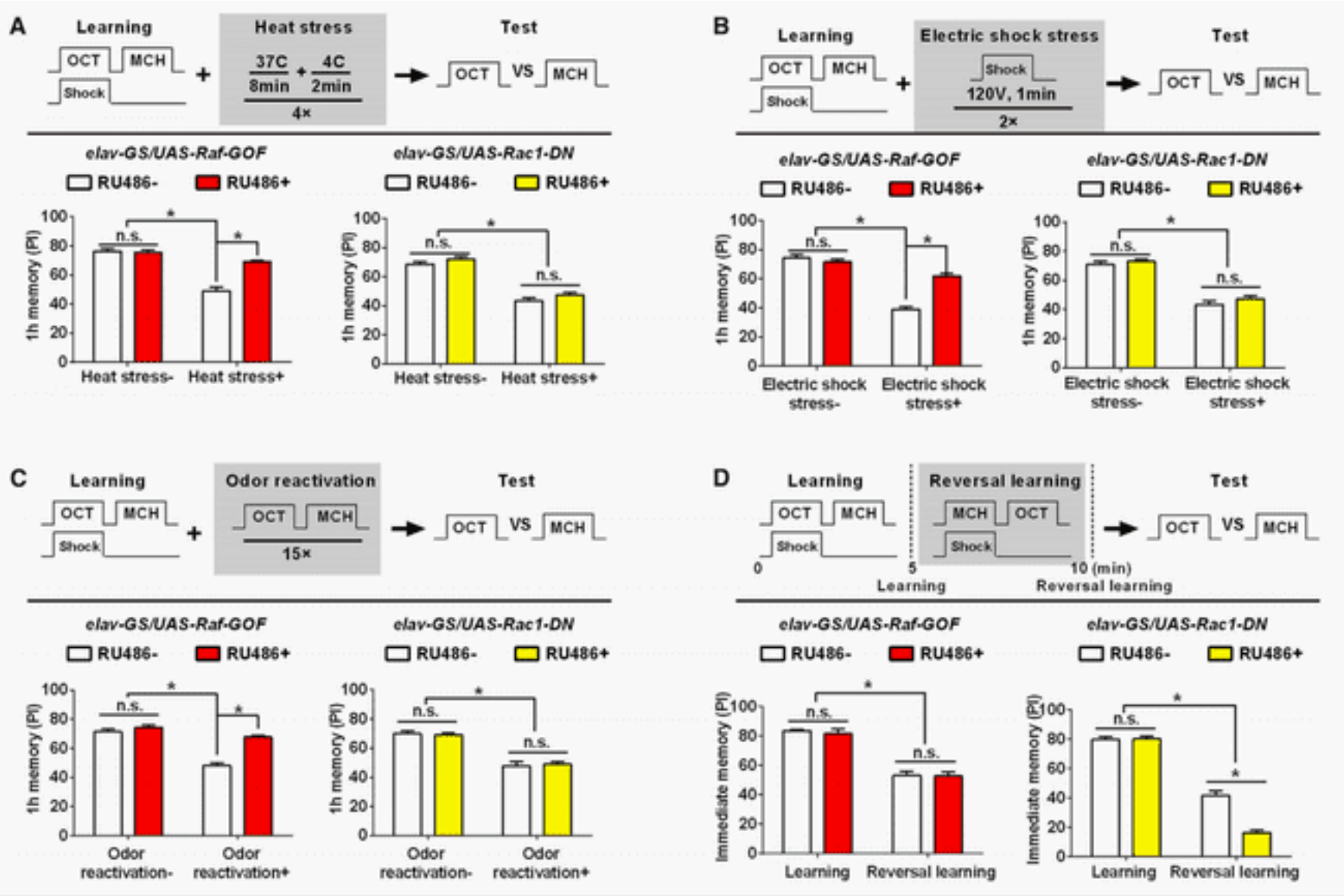




U0126： 抑制MAPK

Zhang, Xuchen et al.Neuron.(2018)

3.Raf-Suppressed Forgetting Is Distinct from Rac1-Mediated Active Forgetting



非联想学习干扰：如电刺激、环境温度剧变和学习相关的嗅觉刺激

联想学习干扰：如相似学习干扰和逆转学习干扰（Rac1）

Zhang, Xuchen et al.Neuron.(2018)

Summary

不稳定记忆如何被主动保护?

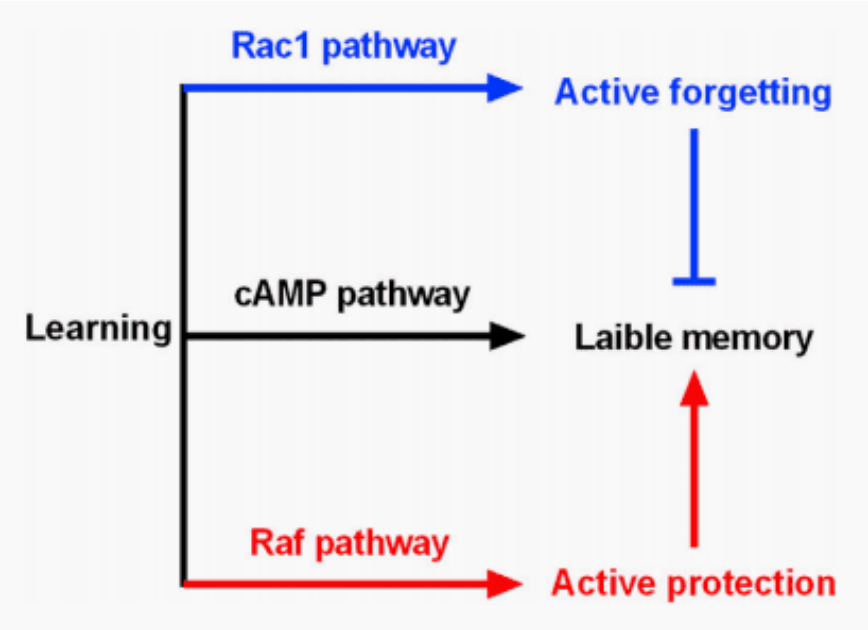
1.学习过程会主动激活Raf/MAPK信号通路。

2.延长这一信号通路激活时间后，观察到短期记忆也被显著的延长，而不影响记忆的形成过程。

缩短这一信号通路激活时间后，短期记忆会发生更快速的遗忘，而且同样不影响记忆的形成。

所以，Raf/MAPK通路会抑制一种遗忘机制。

3.Raf/MAPK所抑制的遗忘可能是独立于Rac1通路存在的另外一种遗忘机制。



Rac1-mediated active forgetting
Raf-mediated active protection

Tips for delaying forgetting:

Repeated Learning

The best for the last

Go sleeping after learning something new

Have a fantastic social life

Thank you !