CIRCADIAN RHYTHMS

Remembering night and day

an intact SCN is required for circadian dysrhythmia to cause disruption of memory formation

Disruption to circadian rhythms caused by, for example, shift work or travel across time zones, is associated with impaired memory. Age-related circadian dysrhythmia can also contribute to dementia and mild cognitive impairment. Surprisingly, however, animals in which circadian arrhythmia is induced by disruption to the suprachiasmatic nucleus (SCN, the main control centre of circadian rhythms in the brain) display only minor memory deficits. The reason for this

is not known, but Fernandez *et al.* now show that intact SCN circuitry is crucial for circadian arrhythmia to affect memory.

Fernandez *et al.* used Siberian hamsters, in which, as in other nocturnal rodents, exposure to a single light pulse during the dark period causes phase resetting. However, exposure to such a phase-advancing light stimulus on the first night, followed by a phase-delaying light stimulus on the following night (termed disruptive phase shift protocol, DPS), results in a complete loss of circadian fluctuations in clock gene expression in the SCN and a loss of normal behavioural circadian variation. This dysrhythmia persists even when the animals are subsequently kept under normal light–dark cycle conditions.

The DPS protocol is useful because it allows comparison of the effect of intact versus ablated SCN circuitry on the behavioural effects of circadian arrhythmia. The authors compared novel object recognition memory (NOR) and spatial memory in intact hamsters exposed to DPS and SCN-ablated hamsters.

In both these memory tests, hamsters with bilateral SCN ablation performed as well as controls

in both memory tests, but the performance of DPS hamsters was markedly reduced. These findings suggest that an intact SCN is required for circadian dysrhythmia to cause disruption of memory formation.

To further test the nature of the link between circadian dysrhythmia and memory formation, the authors trained hamsters in both the NOR and spatial memory tasks to establish a baseline response. Animals were then exposed to the DPS protocol and tested a second time. Then, after recovery from SCN ablation or sham surgery, these animals were tested a third time. Before DPS, hamsters showed normal spatial memory and NOR performance, which was lost following DPS. Interestingly, however, SCN ablation reinstated both recognition and spatial memory to pre-DPS levels. Together, these findings suggest that circadian arrhythmia is not sufficient to cause memory disruption, but that expression of this cognitive impairment requires intact SCN circuitry.

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ORIGINAL RESEARCH PAPER Fernandez, F. *et al.* Dysrhythmia in the suprachiasmatic nucleus inhibits memory processing. *Science* **346**, 854–857 (2014)

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