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Effects of sleep deprivation on retrieval and reconsolidation of morphine reward memory in rats

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ABSTRACT

Relapse induced by exposure to cues associated with drugs of abuse is a major challenge to the treatment of drug addiction. Drug seeking can be inhibited by manipulation of the reconsolidation of drug-related memory. Sleep has been proposed to be involved in various memory processes. However, the role of sleep in drug reward memory is not clear. The present study used conditioned place preference to examine the effects of total sleep deprivation on retrieval and reconsolidation of morphine reward memory in rats. Six-hour total sleep deprivation had no effect on the retrieval of morphine reward memory. However, sleep deprivation from 0–6 h, but not 6–12 h, after re-exposure disrupted the reconsolidation of morphine reward memory. This impairment was not attributable to the formation of an aversive associative memory between the drug-paired context and sleep deprivation. Our findings suggest that sleep plays a critical role in morphine reward memory reconsolidation, and sleep deprivation may be a potential non-pharmacotherapy for the management of relapse associated with drug-related memory.

1. Introduction

Drug addiction is a chronic, relapsing brain disorder (Leshner, 1997). Repeated drug administration can develop an intense associative memory between drug-paired cues and the rewarding effects of the drug (Nestler, 2001; Hyman, 2006; Robbins et al., 2008). Relapse can occur when addicts encounter cues associated with their prior drug use (Childress et al., 1988). However, similar to other types of memories, a consolidated drug reward memory is not permanently resistant to change, and reactivation can return it to a labile, sensitive state, during which it can be modified or erased (Nader, 2003). Using pharmacological techniques, a growing number of studies has disrupted the drug reward memory by manipulating the reconsolidation process (Debiec and Ledoux, 2004; Lee et al., 2005; Lee et al., 2006; Boccia et al., 2007; Robinson and Franklin, 2007; Wang et al., 2008; Bustos et al., 2009; Yu et al., 2009).

Sleep has been proposed to be involved in a variety of physiological process, ranging from thermoregulation to the maintenance of immune function (Rechtschaffen, 1998). Recently, accumulating evidence has elucidated a critical role of sleep in memory processes. Human and animal studies have shown that both declarative and procedural learning can alter subsequent sleep stages, and selective sleep deprivation can impair memory consolidation and extinction (Aly and Moscovitch, 2010; Brawn et al., 2010; Diekelmann and Born, 2010; Ellenbogen et al., 2006; Stickgold and Walker, 2007; Rauchs et al., 2008; Debarnot et al., 2009; Hussaini et al., 2009; Landsness et al., 2009; Li et al., 2009a, 2009b; Sterpenich et al., 2009). However, few studies have explored the relationship between sleep and reconsolidation (Stickgold and Walker, 2005; Tian et al., 2009). In the present study, we used an animal model of conditioned place preference (CPP) to study the effects of pre- and post-retrieval sleep deprivation on the retrieval and reconsolidation of morphine reward memory in rats.

2. Materials and methods

2.1. Animals

Ninety-nine male Sprague–Dawley rats weighing 220–240 g were obtained from the Laboratory Animal Center, Peking University Health Science Center. Rats weighed approximately 300–320 g when experiments began. They were housed individually in a temperature-(23 ± 2 °C) and humidity-(50 ± 5%)controlled room with food and water available *ad libitum* and were kept on a reverse 12 h/12 h light/dark cycle. All animal procedures were performed in accordance with the National Institutes of Health Guide for the Care and Use of Laboratory Animals and were approved by the local Committee of Animal Use and Protection.

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2.2. Behavioral procedures

2.2.1. Conditioned place preference

The CPP procedure was conducted according to our previous study (Li et al., 2008; Wang et al., 2008). The apparatus for CPP training and testing consisted of five identical three-chamber polyvinyl chloride boxes. Two large side chambers (27.9 cm long × 21.0 cm wide × 20.9 cm high), with differences in floor texture (bar or grid, respectively), were separated by a smaller chamber (12.1 cm long × 21.0 cm wide × 20.9 cm high with a smooth polyvinyl chloride floor). These three distinct chambers were separated by manual guillotine doors.

To determine baseline preference, rats were initially placed in the middle chamber with the doors removed for 15 min (pre-conditioning test). A computer measured the time spent in the designated saline- or morphine-paired chambers during the 15 min session through interruptions of infrared beams. Most rats spent approximately one-third of the time in each chamber (data not shown). About 2% of the total rats were discarded because of a strong unconditioned preference toward one chamber (>540 s). Conditioning was performed using an unbiased, balanced protocol.

On the subsequent conditioning days, each rat was trained for 8 consecutive days with alternate injections of drug (morphine, 5 mg/kg, s.c.) and saline (1 ml/kg, s.c.) (Bardo et al., 1995; Zhai et al., 2007; Wang et al., 2008; Li et al., 2009a, 2009b). After each injection, the rats were confined to the corresponding conditioning chambers for 45 min before being returned to their home cages. The next day after conditioning, the rats were tested for the expression of morphine-induced CPP (post-conditioning test) under conditions identical to those described in the pre-conditioning test. The CPP score was defined as the time spent in the morphine-paired chamber minus the time spent in the morphine-unpaired (saline-paired) chamber (Harris et al., 2005).

2.2.2. Retrieval and reconsolidation of reward memory

Rats were confined to the morphine-paired chamber for 10 min to selectively reactivate morphine reward memory (Milekic et al., 2006; Li et al., 2009a, 2009b) and were then subjected to sleep deprivation. Rats were tested for the expression of morphine-induced CPP (post-treatment test) 24 h after sleep deprivation for the reconsolidation test. If rats did not demonstrated morphine CPP after the post-conditioning test (Re-post test), 24 h later they were administered a priming dose (3 mg/kg, s.c.) of morphine and immediately tested again (priming test). For the retrieval test, 18 h after the post-conditioning test, rats were subjected to sleep deprivation immediately followed by re-exposure to the training condition for 15 min.

2.3. Total sleep deprivation

Total sleep deprivation was conducted according to previous studies (Ledoux et al., 1996; Graves et al., 2003; Debiec and Ledoux, 2004; Ramanathan et al., 2010). Briefly, the gentle handling procedure included brushing their fur with a cotton tip applicator, introducing new objects into their cages, disturbing their cage bedding, tapping on their cages. Sleep-deprived rats were kept awake in their home cages by gentle handling to arouse them from sleep for 6 h, from either 0 h to 6 h before or after retrieval or from 6 h to 12 h after retrieval, while Non-sleep-deprived rats were left undisturbed in their home cages under the same feeding environment as the sleep-deprived rats. All rats were habituated for the handling for at least 3 days before experiments and all experiments were balanced between sleep-deprived and non-sleep-deprived rats.

2.4. Experimental design

2.4.1. Experiment 1: effects of total sleep deprivation on reward memory retrieval

Experiment 1 used two groups of rats ($n = 8$–10 per group) to determine the effect of total sleep deprivation on reward memory retrieval. The rats were trained for morphine-induced CPP for 8 days and tested for the expression of CPP on day 9. One day later, the rats were randomly divided into different groups that were matched for CPP scores and subjected either to sleep deprivation or no sleep deprivation. All rats were then again tested for CPP (Fig. 1A).

2.4.2. Experiment 2: effects of total sleep deprivation on reward memory reconsolidation

Experiment 2 investigated the role of total sleep deprivation in the reconsolidation of morphine reward memory. Rats were randomly divided into different groups (n = 9–12 per group) that were matched for CPP scores and subjected to treatments (exposure to the morphine-paired chamber and sleep deprivation) 24 h after post-conditioning: (*i*) rats were not given any treatments (No exposure + No sleep deprivation,), (*ii*) rats were re-exposed to the morphine-paired chamber for 10 min without sleep deprivation (exposure + No sleep deprivation), (*iii*) rats were not re-exposed to any chamber prior to sleep deprivation (No exposure + 0–6 h sleep deprivation), and (*iv*) rats were subjected to sleep deprivation immediately after re-exposure to the previously morphine-paired chamber (exposure + 0–6 h sleep deprivation). To investigate the time-window of the effect of sleep deprivation on morphine reward memory reconsolidation, another two groups (n = 9–10 per group) of rats were used: (*i*) rats were subjected to sleep deprivation 6 h after re-exposure to the previously morphine-paired chamber (exposure + 6–12 h sleep deprivation) and (*ii*) rats were subjected to sleep deprivation 6 h after no re-exposure to the previously morphine-paired chamber (No exposure + 6–12 h sleep deprivation). One day later, all rats were subjected to another CPP test according to the same behavioral procedure (Fig. 2A).

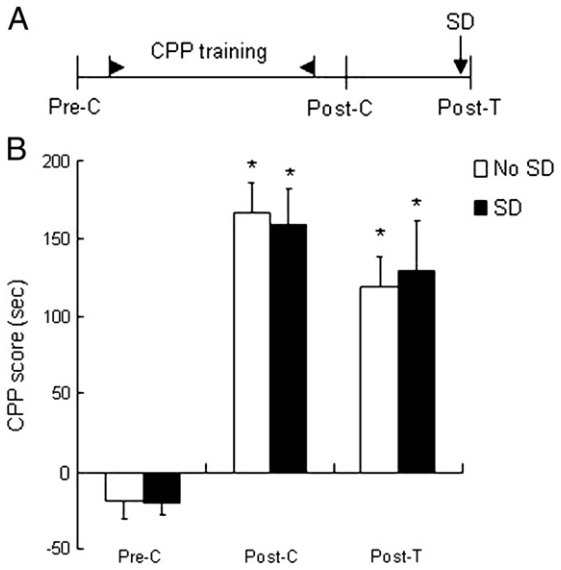

Fig. 1. Effects of total sleep deprivation on the retrieval of morphine reward memory. Data are expressed as mean ± SEM. (A) behavioral procedure. (B) 6 h sleep-deprivation before reactivation of CPP did not affect on morphine reward memory retrieval. *$p<0.01$, compared with pre-conditioning within each group ($n = 8$ or 10 per group). CPP, conditioned place preference; pre-C, pre-conditioning; post-C, post-conditioning; post-T, post-treatment; SD, sleep deprivation.

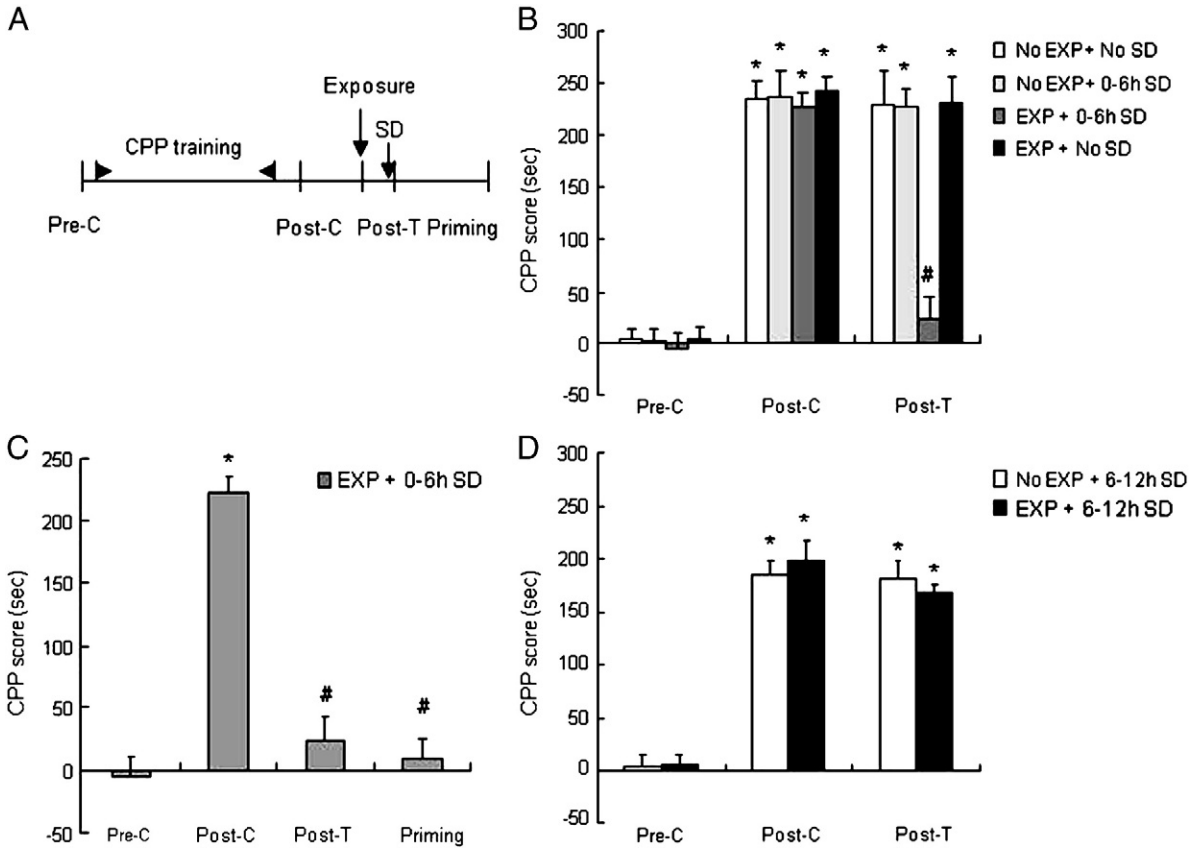

Fig. 2. Effect of sleep deprivation on reconsolidation of morphine reward memory.] Data are expressed as mean ± SEM. (A) behavioral procedure. (B) 6 h total sleep deprivation immediately after reactivation of CPP impaired morphine reward memory reconsolidation. (C) 6 h total sleep deprivation immediately after reactivation of CPP impaired priming of morphine reward memory; (D) total sleep deprivation 6 h after reactivation of CPP did not impair morphine reward memory reconsolidation. $n = 9$–12 per group. $^{*}p < 0.01$, compared with pre-conditioning within group; $^{\#}p < 0.01$, compared with any other group during post-treatment. CPP, conditioned place preference; pre-C, pre-conditioning; post-C, post-conditioning; post-T, post-treatment; SD, sleep deprivation; EXP, exposure.

2.4.3. Experiment 3: effects of total sleep deprivation on saline conditioned place preference

In Experiment 3, to exclude the possibility that post-reactivation sleep deprivation produced an aversive associative memory between the drug-paired context and sleep deprivation, which would counteract the morphine reward memory, we used another two groups of rats. Rats were trained and tested for CPP as described above, with the exception that the rats always received the saline injection before being placed in either side chamber during the 8 day CPP training. The next day after post-conditioning, rats were subjected to sleep deprivation (0–6 h) after re-exposure to one of the two side chambers (side A and side B). Twenty-four hours after sleep deprivation, the rats were re-tested for the expression of CPP according to the same behavioral procedure (Fig. 3A).

2.5. Statistical analyses

Data are expressed as mean ± SEM. Two-way or three-way mixed factorial analyses of variance (ANOVAs) were performed on the data, with the between-subjects factors exposure (exposure, no exposure), sleep deprivation (sleep deprivation, no sleep deprivation), and exposure chamber (side A, side B) and the within-subjects factor test condition (pre-conditioning, post-conditioning, post-treatment). All *post hoc* comparisons were made using the Tukey test. Values of $p < 0.05$ were considered statistically significant.

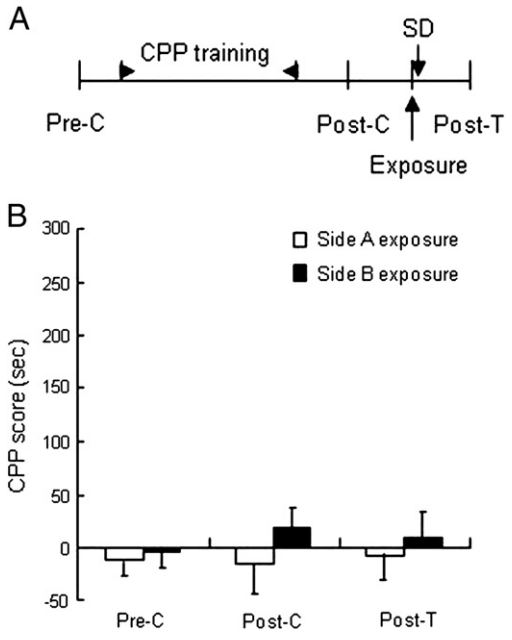

Fig. 3. Total sleep deprivation had no effect on CPP in rats. Data are expressed as mean ± SEM. (A) behavioral procedure. (B) total sleep deprivation did not produce either a preference or aversion toward either side chamber during saline-induced CPP training. $n = 9$ or 10 per group. CPP, conditioned place preference; pre-C, pre-conditioning; post-C, post-conditioning; post-t, post-treatment; SD, total sleep deprivation.

3. Results

3.1. Effects of total sleep deprivation on retrieval of morphine reward memory

Fig. 1A shows the behavioral procedure. There were no differences for time (sec) spent by rats among different groups during pre-conditioning test in drug-paired side (283.7 ± 60.5, 297.5 ± 58.0 for no sleep deprivation and sleep deprivation group respectively) or saline-paired side (309.7 ± 68.5, 298.9 ± 67.2 for no sleep deprivation and sleep deprivation group respectively). The ANOVA with between-subjects factor sleep deprivation (6 h sleep deprivation, no sleep deprivation) and within-subjects factor test condition (pre-conditioning, post-conditioning, post-treatment) revealed a significant effect of test condition ($F_{2,40} = 26.94$, $p < 0.01$) but no effect of sleep deprivation ($p > 0.05$) and no test condition × sleep deprivation interaction ($p > 0.05$). *Post hoc* analyses showed that rats in both groups acquired morphine-induced CPP after 8 days of training ($p < 0.05$), and the expression of morphine-induced CPP was not impaired by 6 h sleep deprivation before the post-treatment test (Fig. 1B). These results indicated that sleep deprivation had no effect on morphine reward memory retrieval.

3.2. Effects of total sleep deprivation on reconsolidation of morphine reward memory

Fig. 2A shows the behavioral procedure. There were no differences for time (sec) spent by rats among different groups during pre-conditioning test in drug-paired side (311.4 ± 27.0, 314.2 ± 40.8, 284.7 ± 63.4, 306.4 ± 27.7, 288.9 ± 40.2 and 323.8 ± 45.2, for No EXP + No SD, No EXP + 0–6 h SD, EXP + No SD, EXP + 0–6 h SD, No EXP + 6–12 h SD and EXP + 6–12 h SD groups respectively) or saline-paired side (307.2 ± 43.3, 311.6 ± 38.7, 294.0 ± 47.5, 302.3 ± 33.8, 286.1 ± 45.1, 318.0 ± 38.8 for those groups above respectively). The ANOVA with between-subjects factor sleep deprivation (0–6 h sleep deprivation, no sleep deprivation) and exposure (exposure, no exposure) and within-subjects factor test condition (pre-conditioning, post-conditioning, post-treatment) revealed significant effects of test condition ($F_{2,66} = 170.18$, $p < 0.01$), exposure ($F_{1,33} = 10.80$, $p < 0.01$), and sleep deprivation ($F_{1,33} = 13.66$, $p < 0.01$) and a significant test condition × exposure × sleep deprivation interaction ($F_{2,66} = 9.70$, $p < 0.01$). The interaction was attributable to the fact that only rats in the exposure + 0–6 h sleep deprivation group exhibited impaired morphine-induced CPP during the post-treatment test (Fig. 2B). *Post hoc* analyses showed that all four groups acquired morphine-induced CPP after conditioning ($p < 0.01$), with no significant differences between any two groups during the post-conditioning test ($p > 0.05$). Moreover, the impaired effect of sleep deprivation on memory is persistent, because the impaired memory did not recover during the priming test following a challenge injection of 5 mg/kg morphine during the day after post-treatment test (Fig. 2C). A one way ANOVA showed that there was no significant difference in CPP score between priming test and Re-Post test or pre-conditioning test ($P > 0.05$). These results indicated that 6 h sleep deprivation immediately following memory reactivation sufficiently impaired morphine reward memory reconsolidation.

In the next experiment, to determine the specific time window of the effects of sleep deprivation on reconsolidation, the ANOVA with between-subjects factor exposure (exposure, no exposure) and within-subjects factor test condition (pre-conditioning, post-conditioning, post-treatment) revealed no significant test condition × exposure interaction ($F_{2,32} = 0.51$, $p > 0.05$). These results indicated that the delayed sleep deprivation had no effect on drug memory reconsolidation (Fig. 2D). *Post hoc* analyses also showed that both groups acquired morphine-induced CPP after conditioning ($p > 0.05$), with no significant differences between groups during either the post-conditioning or post-treatment tests ($p > 0.05$). Altogether, 6 h total sleep deprivation immediately following re-exposure impaired the reconsolidation of the unstable morphine reward memory.

3.3. Effects of total sleep deprivation on saline conditioned place preference

Fig. 3 shows that post-reactivation sleep-deprivation did not produce an aversive associative memory between the drug-paired context and sleep deprivation (Fig. 3B). There were no differences for time (sec) spent by rats among different groups during pre-conditioning test in A-paired side (312.5 ± 31.5, 292.5 ± 39.0 for No SD, SD group respectively) or the B-paired side (325.3 ± 25.3, 287.8 ± 30.8 for no SD, SD group respectively). The two-way ANOVA with between-subjects factor exposure chamber (side A, side B) and within-subjects factor test condition (pre-conditioning, post-conditioning, post-treatment) revealed no significant effect of test condition ($F_{2,34} = 0.09$, $p > 0.05$) or exposure chamber ($F_{1,17} = 1.25$, $p > 0.05$) and no test condition × exposure chamber interaction ($F_{2,34} = 0.11$, $p > 0.05$). Additionally, *post hoc* analyses showed no significant differences between the post-conditioning and post-treatment tests for both the side A and side B groups ($p > 0.05$).

4. Discussion

The present study investigated the role of sleep in the retrieval and reconsolidation of morphine reward memory. Six hour total sleep deprivation before the CPP test had no effect on morphine reward memory retrieval, but 0–6 h (not 6–12 h) total sleep deprivation after exposure to the morphine-paired context disrupted the subsequent expression of morphine-induced CPP, indicating a critical role of sleep in morphine reward memory reconsolidation.

The role of sleep in the reconsolidation of morphine reward memory was specific and not attributable to possible methodological issues. First, the effect of sleep deprivation on morphine reward memory reconsolidation occurred within a specific time window. Sleep deprivation from 0 to 6 h, but not 6 to 12 h, impaired memory, which is consistent with previous memory reconsolidation studies (Nader et al., 2000). Second, 6 h total sleep deprivation immediately after exposure to the saline CPP compartment had no effect on saline CPP, thus excluding the possibility that the effect of sleep deprivation on reconsolidation was attributable to an aversive associative memory between the drug-paired context and sleep deprivation. Third, one explanation for our findings may be that post-reactivation 0–6 h sleep deprivation may facilitate the extinction of morphine reward memory but not disrupt memory reconsolidation. Memory retrieval elicited by re-exposure to the conditioned stimulus without the unconditioned stimulus presentation may initiate two competitive processes: reconsolidation and extinction. However, our findings exclude this possibility because a priming dose of morphine failed to reinstate morphine-induced CPP. Finally, one may argue that the impairment effect of sleep deprivation may be confounded by the effect of stress produced by gentle stroking. Indeed, both acute stress and an increase in the levels of stress hormones can affect memory formation and processing during retrieval, depending on the stressor and the magnitude of hormone increase (Kim and Diamond, 2002; Wolf, 2003). However, gentle handling, which was used for the total sleep deprivation procedure in the present study, has been a common method for investigating the effects of total sleep deprivation and memory in previous studies (Ramanathan et al., 2010; Cai et al., 2009). Moreover, sleep deprivation induced by gentle handling has no effect on plasma levels of corticosterone and adrenocorticotropic hormone (Palchykova et al., 2006). This evidence supports our findings that sleep deprivation impaired morphine reward memory reconsolidation.

Memory is often considered a multi-component process that includes acquisition, consolidation, retrieval, reconsolidation, and

extinction. Many studies have shown that sleep may play an important role in various memory processes, such as acquisition, consolidation, and extinction. Previous results showed that pre-training rapid-eye-movement (REM) sleep deprivation impaired both rewarding and contextual fear learning and inhibitory avoidance conditioning in rats (Dametto et al., 2002). Post-acquisition total sleep deprivation impaired contextual but not cued fear conditioning in C57BL/6 mice (Graves et al., 2003). Silvestri (2005) found that rats subjected to REM sleep deprivation exhibited impaired post-fear conditioning extinction in a cued but not contextual conditioning task, although the retention of both conditioning tasks was normal (Silvestri, 2005). A potential role of sleep in reconsolidation has also been suggested (Stickgold and Walker, 2005; 2007). Our findings support this hypothesis. But the present study appears to contrast with a previous study that post-reactivation REM sleep deprivation, whether from 0 to 6 h or 6 to 12 h, had no effect on the reconsolidation of either cued or contextual fear memory (Tian et al., 2009). The animal model and the methodological manipulation may explain this discrepancy. The present study used morphine-induced CPP, whereas Tian et al. (2009) used contextual and cued fear memory. More importantly, in the present study, rats were subjected to total sleep deprivation rather than specifically REM sleep deprivation. Moreover, the Tian et al. (2009) study cannot exclude the potential role of non-REM sleep in memory reconsolidation.

In summary, the present results demonstrated that established morphine-related reward memories are disrupted by total sleep deprivation via inhibition of its reconsolidation. Furthermore, this impairment effect of sleep deprivation occurred within a specific time window. *Although there is limitation for using sleep deprivation in clinical treatment, the role of sleep deprivation as the adjuvant non-pharmacotherapeutic method to manage relapse associated with drug reward-related memory merits further investigation.*

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