

## Supplementary Information

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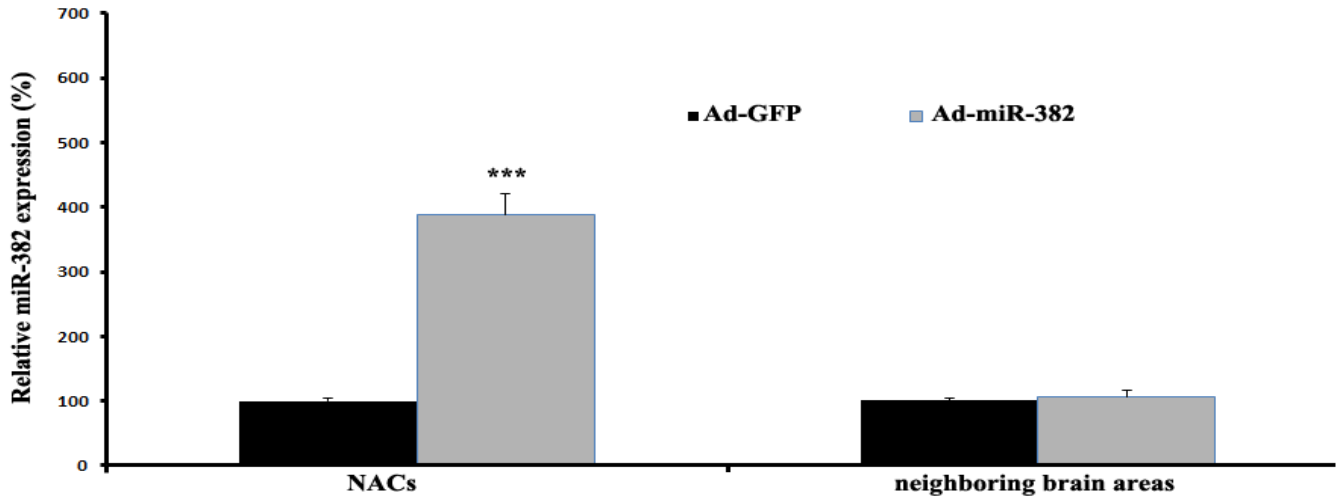
**Table SI:** Comparison of membrane properties of NAc MSNs in acute brain slices of rats received NAc injection of saline or Ad-miR-382.

Group	Saline or virus only	Ad miR-382
Number of neurons	29	16
Passive membrane properties		
RMP (mV)	-67.2 ± 3.0	-62.2 ± 1.8 (P=0.16)
R <sub>in</sub> (MΩ)	344±67	533±133 (P=0.29)
Active membrane properties		
Current to generate AP (pA)	66.00 ± 17.13	87.50 ± 11.97 (P=0.20)
AP threshold (mV)	-31.7 ± 2.03	-35.5 ± 2.3 (P=0.27)
AP amplitude (mV)	62.9 ± 3.4	61.0 ± 3.2 (P=0.71)
½ AP duration (ms)	2.50 ± 0.18	2.3 ± 0.13 (P=0.65)
AHP amplitude (mV)	6.6 ± 1.4	5.5 ± 0.6 (P=0.40)

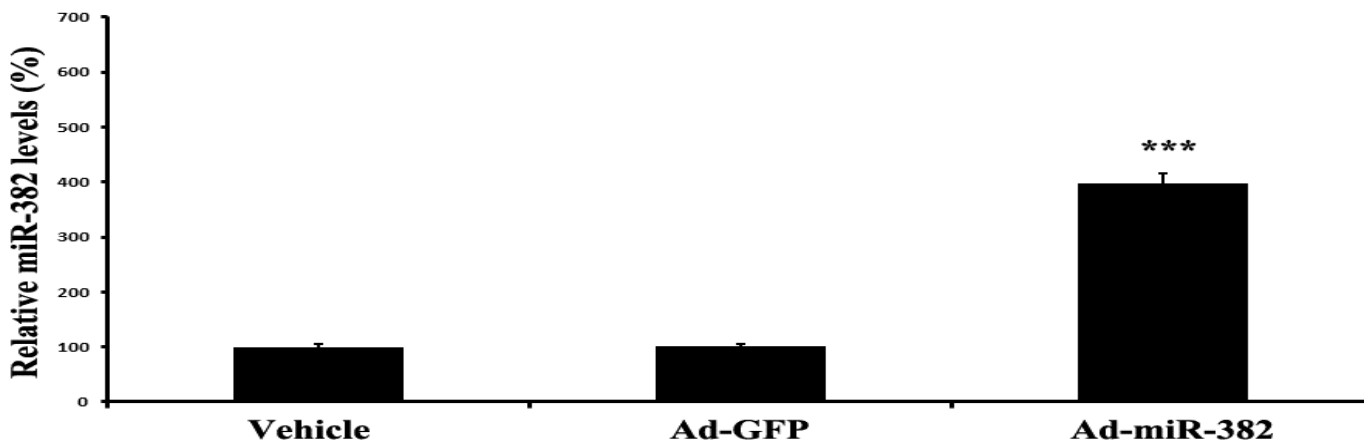
Note: The passive and active membrane properties of medium spiny NAc neurons in acute brain slice were measured by current clamp recordings from rat injecting either Ad-miR-382 or saline, or virus only. Since the results of saline injected and virus only were not difference they were pooled together. Values represent the mean ± SEM for the number of neurons indicated. RMP: resting membrane potential; AP: action potential; AHP: after-hyperpolarization. R<sub>in</sub>, input resistance. The P values are obtained by t-test the difference between the two groups of neurons.

**Table SI: PCR Primer Sequences**

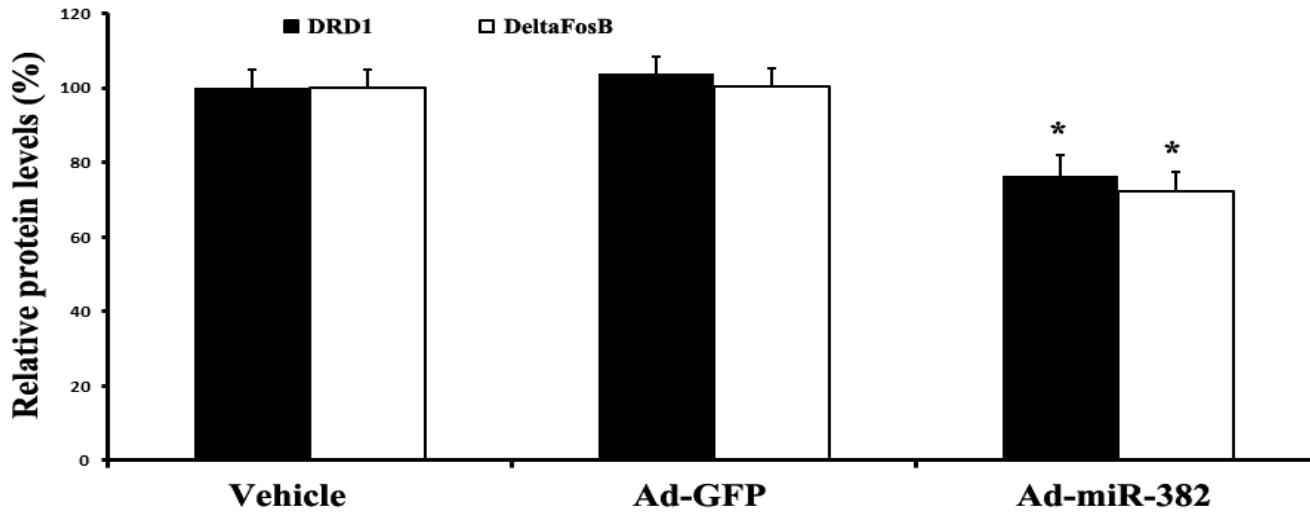
Primer Names	Sequences (5' → 3')
rat ΔFosB real-time forward primer	GAGGAAAAGGCAGAGCTGGA
rat ΔFosB real-time reverse primer	TGGGCCACCAGGACAAACT
rat DRD1 real-time forward primer	ACTTCGGCTCTGAAATCAGTTTGG
rat DRD1 real-time reverse primer	TCAACTCCTACCCTTCCTTTCTGG
mouse DRD1 real-time forward primer	GATGGCTCCTAACACTTCTACC
mouse DRD1 real-time reverse primer	GGCTGTGAGGATGCGAAAG
rat GAPDH real-time forward primer	AAGCTCACTGGCATGGCCTT
rat GAPDH real-time reverse primer	CGGCATGTCAGATCCACAAC



**Figure S1. Delivery of Ad-miR-382 to the rat NAc increases the expression of miR-382 in NAc, but not in the neighboring brain areas.** \*\*\* $P < 0.0001$ , Student's t-test. Ad-miR-382 ( $4\mu\text{l}$ ,  $1 \times 10^9$  pfu/ml) or the control virus ( $4\mu\text{l}$ , Ad-GFP) was injected bilaterally into NAc core using a Kopf stereotaxic frame. Injections were performed with two  $10\mu\text{l}$  Hamilton syringe fitted with a micropipette. Four  $\mu\text{l}$  per side were injected into the NAc core at a rate of  $0.1\mu\text{l}/\text{min}$ . At 5 days after injection, the rat NACs and neighboring brain areas ( $0.3\text{ cm}$  far from NAc) were isolated to determine the miR-382 levels by qRT-PCR. MiR-382 was increased via Ad-miR-382 in NACs ( $p = 1.22306\text{E-}5$ ), but not in the neighboring brain areas. Values are mean  $\pm$  SEM from 3 independent experiments ( $n=3$ ), compared with that in Ad-GFP control.



**Figure SII. The successful upregulation of miR-382 via Ad-miR-382 in NAc of rats at 7 days after drinking of alcohol.** \*\*\* $P < 0.0001$ , Student's t-test. The animals under the intermittent access two-bottle choice drinking paradigm were randomly divided into three groups which received infusion of Ad-miR-382, control adenovirus Ad-GFP, or vehicle (saline) respectively into the NAc. Seven days later, NAc were isolated to determine the levels of miR-382 via qRT-PCR. MiR-382 was overexpressed via Ad-miR-382 ( $p = 8.42076215208\text{E-}5$ ). Values are mean  $\pm$  SEM from 3 independent experiments ( $n=3$ ), compared with that in Ad-GFP control.



**Figure S111. The successful modulation of DRD1 and DeltaFosB via Ad-miR-382 in NAc of rats at 7 days after drinking of alcohol.** \* $P < 0.05$ , Student's t-test. The animals under the intermittent access two-bottle choice drinking paradigm were randomly divided into three groups which received infusion of Ad-miR-382, control adenovirus Ad-GFP, or vehicle (saline) respectively into the NAc. Seven days later, NAc were isolated to determine the levels of DRD1 and DeltaFosB by Western blot analysis. Both DRD1 ( $p = 0.01966$ ) and DeltaFosB ( $p = 0.01604$ ) were downregulated by Ad-miR-382. Values are mean  $\pm$  SEM from 3 independent experiments ( $n = 3$ ), compared with that in Ad-GFP control.