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Data Article

Dataset of eyeblink conditioning in mice treated with the selective mGluR1 antagonist JNJ16259685

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ABSTRACT

Eyeblink conditioning is associated with motor learning, which requires the cerebellum and the brainstem. This article provides behavioral data on whether JNJ16259685, a selective metabotropic glutamate receptor type 1 (mGluR1) antagonist, affects eyeblink conditioning in wild-type mice (C57BL/6 J strain). The dataset contains four types of behavioral outputs pertinent to eyeblink conditioning. We used a *t*-test and an analysis of variance (ANOVA) to analyze the percentage of conditioned responses (CR%), peak CR latencies, onset CR latencies, and electromyography (EMG) amplitudes. The information obtained in this dataset adds to our knowledge of the molecular mechanisms underlying eyeblink conditioning and can prove beneficial for investigators studying the pharmacological effects of mGluR1 on motor learning. Future research might use this dataset as a basis for conducting experiments with different JNJ16259685 doses, administration methods, and durations than the ones used in the described procedures.

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Specifications Table

Subject	Neuroscience
Specific subject area	Behavioral pharmacology
Type of data	Graph Figure
How the data were acquired	Eyeblink conditioning analyzing system (A-M Systems, Sequim, WA, USA; Nihon Kohden, Tokyo, Japan).
Data format	Raw Analyzed Graph
Description of data collection	Data for all mice and all days used in the experiment were evaluated; there were no missing or excluded data. A comprehensive dataset was deposited in Mendeley repository data [1].
Data source location	• Institution: Teikyo University • City/Town/Region: Itabashi City/ Tokyo • Country: Japan • Latitude and longitude (and GPS coordinates, if possible) for collected samples/data: 35° 75′ 90.05″ N and 139° 71′ 38.99″ E
Data accessibility	Data together with the original publication is available on Mendeley with the 10.17632/nnmg5v5rjt.3 Repository name: Mendeley Data Data identification number: https://10.17632/nnmg5v5rjt.3 Direct URL to data: https://data.mendeley.com/datasets/nnmg5v5rjt/3

Value of the Data

- Numerous studies, primarily using genetically engineered mice, have shown that mGluR1 plays an important role in motor learning. Still, only a few studies have examined its pharmacological importance. Thus, this dataset is useful for examining the contribution of mGluR1 to motor learning from a pharmacological perspective.
- This dataset can prove useful for researchers studying the pharmacological effects of mGluR1 on cognitive function, particularly motor learning.
- In the future, researchers can use this dataset as a basis for conducting similar experiments with different JNJ16259685 doses, administration methods, and durations than the ones described here. Additionally, researchers interested in setting up experimental paradigms for eyeblink conditioning in mice can also find it helpful, as there are only a few studies that provide specific raw data regarding eyeblink conditioning in wild-type mice.

1. Objective

Eyeblink conditioning is a type of Pavlovian conditioning that has been extensively used in experimental psychology and neurophysiology. It is used as a behavioral task to study the neural structures and mechanisms underlying learning and memory [2,3]. Various studies over several decades have provided evidence that metabotropic glutamate receptor type 1 (mGluR1) is required for this type of motor learning in rodents [4–8]. These findings have been mainly observed in gene knockout studies or theoretical modeling research. On the other hand, there has been lack of evidence provided by pharmacological studies. Thus, the objective of the present dataset was to present behavioral outputs of the effects of JNJ16259685, a selective mGluR1 antagonist, on eyeblink conditioning in wild-type mice [9].

2. Data Description

This dataset contains four types of behavioral outputs pertinent to eyeblink conditioning in wild-type mice treated with an mGluR1 antagonist. Fig. 1a indicates the percentage acquisi-

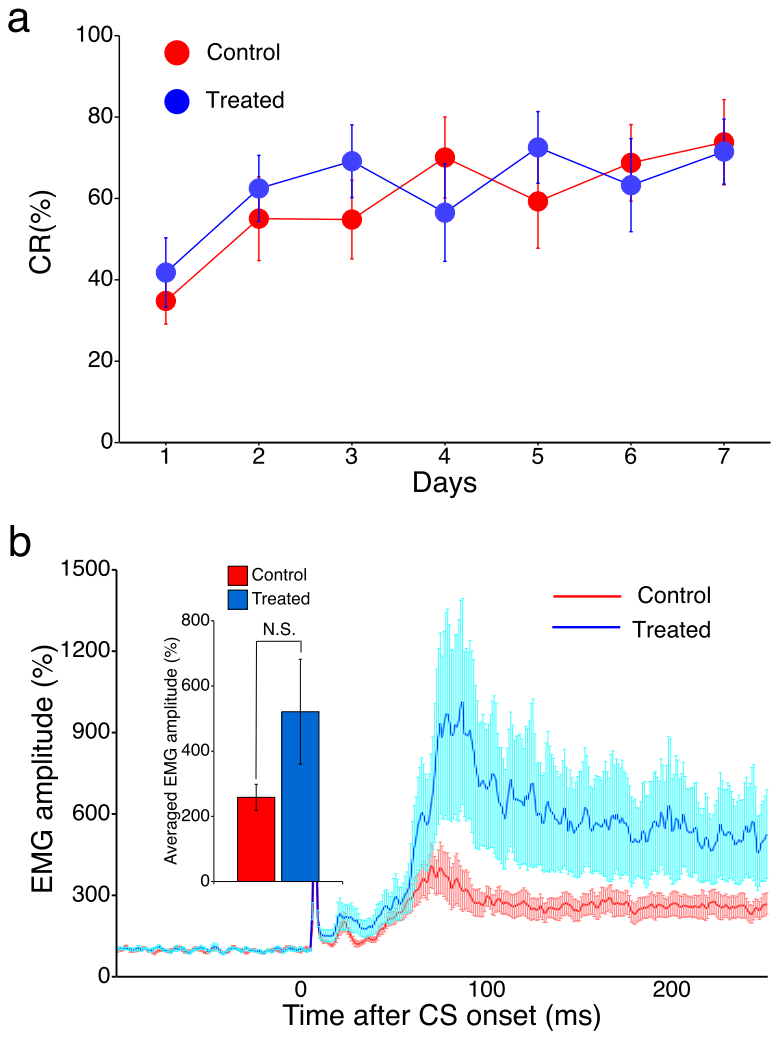

Fig. 1. Conditioned eyeblink responses in the mice treated with JNJ16259685.
(A) Developing of eyeblink CR% in the control (red circle, $n = 11$) and JNJ16259685-treated mice (blue circle, $n = 11$). (B) Average EMG amplitudes on day 7. On the horizontal axis, the time of CS onset is set to zero. EMG amplitudes obtained in 100 trials were averaged to represent the overall blink activity response pattern. Inset: Quantitative comparison of EMG amplitude just before US onset between control (red column) and JNJ16259685-treated (blue column) mice. The amplitude represents the magnitude of CR. The amplitude was evaluated based on the 50 ms period immediately prior to the unconditioned stimuli (US). All data are represented as mean ± standard error of the mean (S.E.M.). N.S. indicates not significant ($p > 0.05$).

tion of conditioned responses (CR%) of the control and JNJ16259685-treated groups. A two-way ANOVA did not detect significant differences in either the interaction effect between sessions and groups ($F(6, 120) = 1.325$, $p = 0.251$) nor any groups effect ($F(1, 20) = 0.076$, $p = 0.786$). Fig. 1b shows the average EMG amplitudes on day 7, the last day of the session. Although the EMG amplitude in the JNJ16259685-treated groups was larger compared to the controls, quantitative analysis did not detect significant differences between the two groups (Fig. 1b inset; $p = 0.129$).

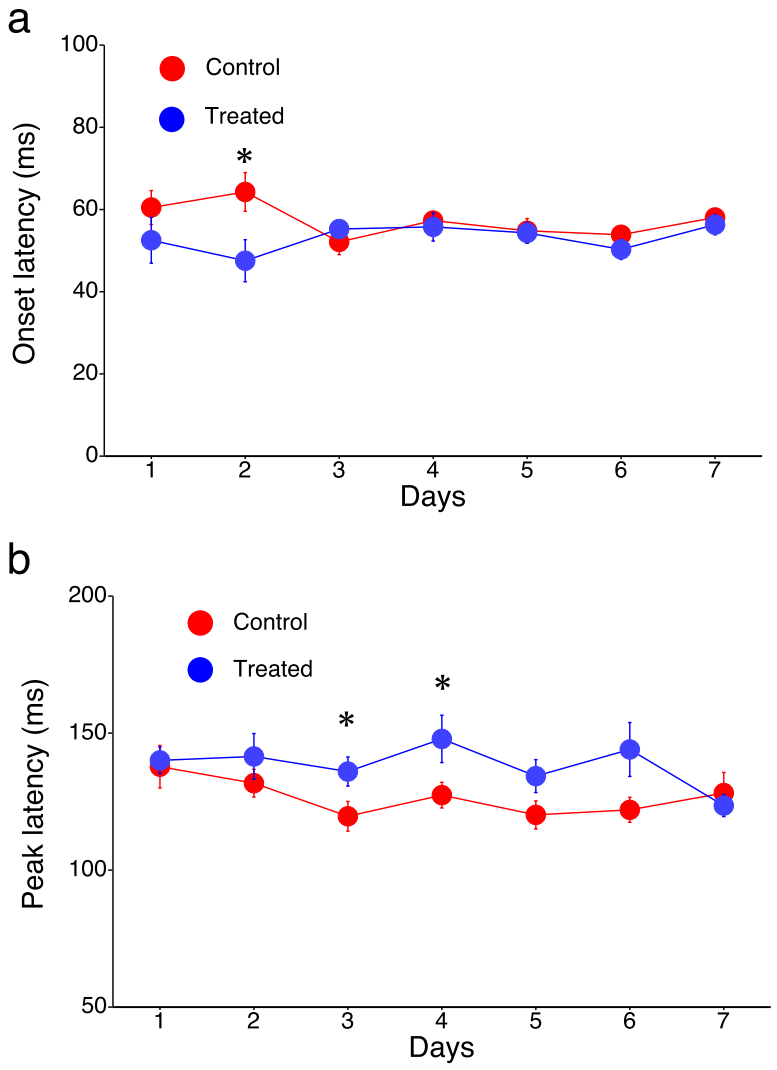

Fig. 2. Timing of conditioned eyeblink responses in the mice treated with JNJ16259685. (A) The onset latency of CR during 7-days session were evaluated in control (red circle, $n = 11$) and JNJ16259685-treated mice (blue circle, $n = 11$). (B) The course of peak latency of CR during eyeblink conditioning in control (red circle, $n = 11$) and JNJ16259685-treated mice (blue circle, $n = 11$). Data are represented as mean ± standard error of the mean (S.E.M.). * indicates $p < 0.05$ for the post-hoc test.

Fig. 2 shows the latency behavior of the CR throughout the 7-day conditioning session. Regarding the onset latency (Fig. 2a), a two-way ANOVA revealed no significant interaction effect between session and group ($F(6, 120) = 1.83$, $p = 0.099$). On the other hand, it revealed a significant group effect ($F(1, 20) = 5.75$, $p = 0.026$). Similar results were observed for peak latency (Fig. 2b). There was no significant interaction effect ($F(6, 120) = 1.198$, $p = 0.312$), however there was a significant group effect ($F(1, 20) = 8.38$, $p = 0.009$).

3. Experimental Design, Materials and Methods

3.1. Animals

We used male C57BL/6 J wild-type mice (Sankyo Labo Service Corporation, Tokyo, Japan) with body weights 22–28 g (8–9 weeks old). Mice were maintained in standard cages under a 12 h light/dark cycle. Food and water were provided ad libitum.

3.2. Drugs

We followed the protocol described by Steckler et al. [10] where JNJ16259685 (Tocris Bioscience, UK) was dissolved in saline containing 10% hydroxypropyl-β-cyclodextrin acidified with tartaric acid. The solutions were administered subcutaneously (s.c.) 30 min before the start of a daily training session at a dose of 10 mg/kg. Steckler et al. [10] reported that JNJ16259685 was administered at doses between 0.63–10 mg/kg and that even the lowest dose of 0.63 mg/kg inhibited spatial learning in mice; hence, in the present experiment, we chose the highest concentration (10 mg/kg) used. The control group was administered a drug-free solvent.

3.3. Eyeblink Conditioning

The classical conditioning procedure was the same as previously reported [11,12]. Four stainless-steel electrodes (100 μm diameter; A-M Systems, Sequim, WA, USA) were implanted s.c. under the left eyelid of anesthetized mice. Two of the electrodes were used to deliver unconditioned stimuli (US), while the remaining two were used to receive EMG signals from the orbicularis oculi muscle. The conditioned stimulus (CS) was a tone (352 ms, 1 kHz, 80 dB), and the US was an electric shock (100 ms, 0.2–0.5 mA, 100 Hz). The CS and US overlapped in time and were completed simultaneously. The mice completed these acquisition sessions over a 7-day period. The EMG criteria used for determining CR, as well as the definitions and evaluation methods for EMG amplitudes and latency, were the same as those described in previous reports [11,12]. The CR amplitude was calculated as the average amplitude over the 50 ms period immediately prior to the US [13].

3.4. Statistical Analysis

Data were analyzed with a *t*-test and a two-way (session $\times$ group) ANOVA, followed by a post-hoc Bonferroni test. All statistical analyses were performed using GraphPad Prism version 6.0.0 (GraphPad Software, San Diego, California USA). All data are presented as the mean (M) $\pm$ standard error of the mean (S.E.M.). The significance threshold was set at $p < 0.05$.

Ethics Statement

All animal procedures were approved by the Teikyo University Animal Ethics Committee (approval number: 21–029) and were carried out in accordance with the National Institutes of Health guide for the care and use of laboratory animals (NIH Publications No. 8023, revised 1978).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data regarding classical eyeblink conditioning in mouse treated with metabotropic glutamate receptor 1 antagonist JNJ16259685. (Original data) (Mendeley Data).

CRediT Author Statement

Shoichi Tohyama: Investigation, Data curation, Writing – original draft; **Yasushi Kishimoto:** Supervision, Methodology, Software, Writing – original draft, Writing – review & editing.

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References

[1] Y. Kishimoto, S. Tohyama (2022), "Data regarding classical eyeblink conditioning in mouse treated with metabotropic glutamate receptor 1 antagonist JNJ16259685", Mendeley Data, V3, Available at: https://data.mendeley.com/datasets/nnmg5v5rjt/3. doi:10.17632/nnmg5v5rjt.3.

[2] D.A. McCormick, R.F. Thompson, Cerebellum: essential involvement in the classically conditioned eyelid response, Science 223 (1984) 296–299, doi:10.1126/science.670151.

[3] R.F. Thompson, D.J. Krupa, Organization of memory traces in the mammalian brain, Annu. Rev. Neurosci. 17 (1994) 519–549, doi:10.1146/annurev.ne.17.030194.002511.

[4] A. Aiba, M. Kano, C. Chen, M.E. Stanton, G.D. Fox, K. Herrup, T.A. Zwingman, S. Tonegawa, Deficient cerebellar long-term depression and impaired motor learning in mGluR1 mutant mice, Cell 21 (1994) 377–388, doi:10.1016/0092-8674(94)90205-4.

[5] Y. Kishimoto, R. Fujimichi, K. Araishi, S. Kawahara, M. Kano, A. Aiba, Y. Kirino, mGluR1 in cerebellar Purkinje cells is required for normal association of temporally contiguous stimuli in classical conditioning, Eur. J. Neurosci. 16 (2002) 2416–2424, doi:10.1046/j.1460-9568.2002.02407.x.

[6] H. Nakao, Y. Kishimoto, K. Hashimoto, K. Kitamura, M. Yamasaki, K. Nakao, M. Watanabe, M. Kano, Y. Kirino, A. Aiba, mGluR1 in cerebellar Purkinje cells is essential for the formation but not expression of associative eyeblink memory, Sci. Rep. 9 (2019) 1–10, doi:10.1038/s41598-019-43744-z.

[7] M. Harbers, H. Nakao, T. Watanabe, K. Matsuyama, S. Tohyam, K. Nakao, Y. Kishimoto, M. Kano, A. Aiba, mGluR5 is substitutable for mGluR1 in cerebellar Purkinje cells for motor coordination, developmental synapse elimination, and motor learning, Cells 11 (2022) 2004, doi:10.3390/cells11132004.

[8] J.C. Fiala, S. Grossberg, D. Bullock, Metabotropic glutamate receptor activation in cerebellar Purkinje cells as substrate for adaptive timing of the classically conditioned eye-blink response, J. Neurosci. 16 (1996) 3760–3774, doi:10.1523/JNEUROSCI.16-11-03760.1996.

[9] H. Lavreysen, R. Wouters, F. Bischoff, S. Nóbrega Pereira, X. Langlois, S. Blokland, M. Somers, L. Dillen, A.S.J Lesage, JNJ16259685, a highly potent, selective and systemically active mGlu1 receptor antagonist, Neuropharmacology 47 (2004) 961–972, doi:10.1016/j.neuropharm.2004.08.007.

[10] T. Steckler, A. Oliveira, C. Van Dyck, H. Van Craenendonck, A. Mateus, X. Langlois, A. Lesage, J. Prickaerts, Metabotropic glutamate receptor 1 blockade impairs acquisition and retention in a spatial water maze task, Behav. Brain Res. 16 (2005) 52–60, doi:10.1016/j.bbr.2005.05.010.

[11] Y. Kishimoto, I. Oku, A. Nishigawa, A. Nishimoto, Y. Kirino, Impaired long-trace eyeblink conditioning in a Tg2576 mouse model of Alzheimer's disease, Neurosci. Lett. 506 (2012) 155–159, doi:10.1016/j.neulet.2011.10.071.

[12] Y. Kishimoto, M. Hirono, R. Atarashi, S. Sakaguchi, T. Yoshioka, S. Katamine, Y. Kirino, Age-dependent impairment of eyeblink conditioning in prion protein-deficient mice, PLoS One 8 (2013) e60627, doi:10.1371/journal.pone.0060627.

[13] Y. Kishimoto, M. Hirono, R. Atarashi, S. Sakaguchi, T. Yoshioka, S. Katamine, Y. Kirino, Impairment of cerebellar long-term depression and GABAergic transmission in prion protein deficient mice ectopically expressing PrPLP/Dpl, Sci. Rep. 10 (2020) 15900, doi:10.1038/s41598-020-72753-6.