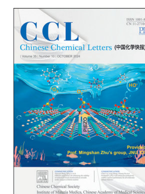
Chinese Chemical Letters 35 (2024) 109572

Contents lists available at ScienceDirect

Chinese Chemical Letters

journal homepage: www.elsevier.com/locate/cclet



“Three-in-one” strategy of trifluoromethyl regulated blood-brain barrier permeable fluorescent probe for peroxynitrite and antiepileptic evaluation of edaravone

Lei Shen[a], Hongmei Liu[a], Ming Jin[a], Jinchao Zhang[a], Caixia Yin[b,*], Shuxiang Wang[a,*], Yutao Yang[a,*]

[a] *Key Laboratory of Medicinal Chemistry and Molecular Diagnosis of the Ministry of Education, Chemical Biology Key Laboratory of Hebei Province, State Key Laboratory of New Pharmaceutical Preparations and Excipients, College of Chemistry and Materials Science, Hebei University, Baoding 071002, China*
[b] *Key Laboratory of Chemical Biology and Molecular Engineering of Ministry of Education, Institute of Molecular Science, Shanxi University, Taiyuan 030006, China*

ARTICLE INFO

Article history:
Received 13 December 2023
Revised 24 January 2024
Accepted 25 January 2024
Available online 2 February 2024

Keywords:
Fluorescent probe
Blood-brain barrier
Peroxynitrite
Epilepsy
Drug repurposing

ABSTRACT

Epilepsy, as a chronic neurological disease of the brain, is closely related to oxidative stress, and the peroxynitrite ($ONOO^-$) significantly rise up in this event. Therefore, $ONOO^-$ is considered as a potential biomarker for early prediction of epilepsy. However, some potential diagnostic reagents for epilepsy are hindered by the blood-brain barrier (BBB). Meanwhile, “drug repurposing” is attracting a growing interest. Edaravone (EDA), as a first-line drug in the clinical treatment of cerebral ischemia, plays antioxidant roles in scavenging free radicals, promising potential antiepileptic activity. Thus, it is imperative to develop fluorescent probes for monitoring $ONOO^-$ fluctuations in the epileptic brain. Hence, we proposed a novel fluorescent probe with the thiocarbonate as the promising recognition unit for $ONOO^-$ and dicyanoisophorone derivative as the fluorophore. Moreover, by the “three-in-one” strategy, the introduction of trifluoromethyl into **DCI-ONOO-3** can extend the emission wavelength of the fluorophore, shorten the response and increase lipophilicity. Consequently, **DCI-ONOO-3** was used for monitoring $ONOO^-$ fluxes in brain of epileptic mice and evaluating the antiepileptic efficacy of EDA. It opens up a new way for the design of BBB permeable fluorescent probes, and provides a convincing new method for the diagnosis and treatment of epilepsy.

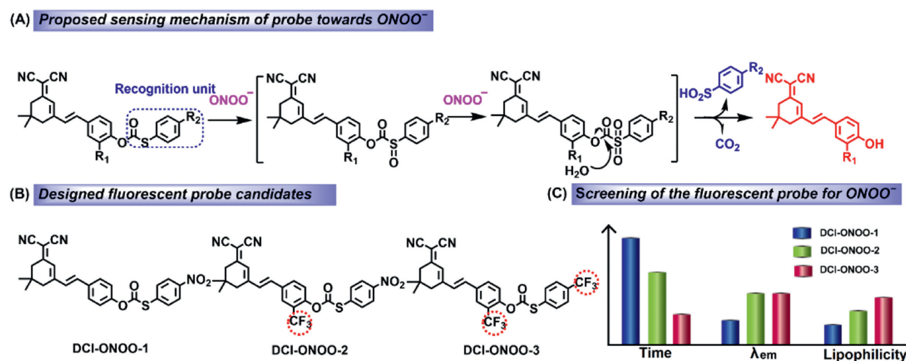
© 2024 Published by Elsevier B.V. on behalf of Chinese Chemical Society and Institute of Materia Medica, Chinese Academy of Medical Sciences.

Epilepsy is a neurodegenerative disease occurring at all ages with high disability and fatality rate. It is a kind of serious disease which threatens human’s health and life [1–3]. Rapid and accurate diagnosis of epilepsy and effective treatment are essential to control seizures. Recently, as numerous studies have shown, oxidizing stress is one of the precipitating factors, and a large number of reactive oxygen species (ROS) are produced during epileptic seizure [4–7]. The superoxide anion ($^{\bullet}O_2^-$) produced in the body rapidly combines with nitric oxide (NO) to generate excessive $ONOO^-$, which causes irreversible damage to biomacromolecules for example DNA, protein and so on, leading in turn to neuron cells death [8–15]. Therefore, oxidative stress should be considered in the treatment of patients with epilepsy [16,17]. Abnormal levels of $ONOO^-$ lead to the redox imbalance in cells, and it may serve as the potential diagnostic biomarker and therapeutic target for epilepsy.

Since 2019, due to the impact of corona virus disease 2019 (COVID-19), the strategy of “drug repurposing” is attracting a growing interest recently, especially for the research of marketed drugs for new indications. As we know, edaravone (EDA) is a first-line drug for the clinical treatment of cerebral ischemia. It was first developed by Mitsubishi Tanabe Pharma Corporation and approved for market in Japan in 2001 [18]. EDA has good effects in preventing and treating cerebral ischemia, and the mechanism of treatment may be related to protect the oxidative injury of nerve cell through antioxidative action of scavenging free radicals and inhibiting lipid peroxidation [19]. Hence, we hypothesize that EDA might alleviate epilepsy, since the onset of epilepsy is closely related to oxidative nerve damage. Thus, it is imperative to develop novel methods for monitoring the $ONOO^-$ fluctuations during seizures for evaluating the efficacy of EDA in the brains of epilepsy.

* Corresponding authors.
E-mail addresses: yincx@sxu.edu.cn (C. Yin), wsx@hbu.edu.cn (S. Wang), yutaoyang2016@163.com (Y. Yang).

https://doi.org/10.1016/j.cclet.2024.109572
1001-8417/© 2024 Published by Elsevier B.V. on behalf of Chinese Chemical Society and Institute of Materia Medica, Chinese Academy of Medical Sciences.



Scheme 1. Design strategy of the fluorescent probes for ONOO⁻.

Fluorescent probes, as the ideal tool for mapping bioactive molecules in biological systems because of the superior performance of simple operation, high sensitivity, high selectivity, and non-invasive visualization, have shown excellent effects in diagnosing diseases and evaluating pharmacodynamics in recent years [20–25]. To date, several fluorescent probes have been developed for real-time detection of enzymes, ROS, active nitrogen (RNS), and other active molecules for pharmacodynamic evaluation in mice [26–28]. However, evaluation of EDA for the antiepileptic pharmacodynamics using fluorescent probes has not been reported. Therefore, it is necessary to develop a novel near-infrared (NIR) fluorescent probe to monitor ONOO⁻ fluctuations in order to evaluate the pharmacodynamics of EDA in epileptic mice brain. However, few probes have been able to detect ONOO⁻ in the brain, which limit the efficacy evaluation of antiepileptic drugs *in vivo*. Thus, development of fluorescent probes that can effectively track endogenous ONOO⁻ fluctuations in kainic acid (KA) induced seizures is necessary. There are several challenges of probes to achieve the goal, possessing the characteristic feature of crossing the BBB, high sensitivity, high selectivity and fast response for ONOO⁻ in NIR region.

In view of the above considerations, we are committed to developing fluorescent probes to visualize ONOO⁻ fluctuations in the epileptic brain. Hence, we proposed a promising recognition unit for ONOO⁻, thiocarbonate derivatives, due to the susceptibility to oxidation and decomposition under mild conditions [29]. ONOO⁻ induced probes to remove thiocarbonate derivatives, and significantly restored the fluorescence emission of fluorophores (Scheme 1A). In this study, we firstly designed the probe **DCI-ONOO-1** for detection of ONOO⁻. However, ONOO⁻ induced **DCI-ONOO-1** to release **DCI-OH** with emission wavelength at 598 nm, which was not suitable for *in vivo* imaging. As we know, to construct an ideal fluorescent probe for brain imaging, several factors must be considered, such as emission wavelength, molecular weight, signal intensity, stability, lipophilicity. Therefore, the **DCI-CF$_3$-OH** was developed by introduction of trifluoromethyl into **DCI-OH** to adjust the acidity of the phenol hydroxyl group, prolonging the emission wavelength at 652 nm (Figs. S1 and S2 in Supporting information). Then, **DCI-ONOO-2** and **DCI-ONOO-3** were following closely synthesized by introducing trifluoromethyl due to its high lipidsolubility, good metabolic stability, high electronegativity and bioavailability (Scheme 1B and Scheme S1 in Supporting information). Subsequently, the physicochemical properties of the **DCI-ONOO-1**, **DCI-ONOO-2** and **DCI-ONOO-3** such as the oil-water partition coefficient (Log*P*) and BBB permeability (Pe) of were investigated, and the corresponding results were 0.71, 1.03, 1.27 for Log*P* and 1.22, 2.36, 3.27 for Pe, respectively (Figs. S3 and S4, Table S1 in Supporting information). Also, the kinetics of **DCI-ONOO-1**, **DCI-ONOO-2** and **DCI-ONOO-3** with ONOO⁻ were also studied with response times of 730, 520 and 210 s, respectively (Fig. S5 and Table S1 in Supporting information).

To sum up, by the "three-in-one" strategy, the introduction of trifluoromethyl in the **DCI-ONOO-3** can (1) improve the reaction activity and shorten the reaction time of **DCI-ONOO-3** with ONOO⁻ by facilitating the hydrolysis of oxidation products, (2) extend the emission wavelength of the fluorophore into the NIR region by adjusting the acidity of the phenol hydroxyl group, (3) increase lipophilicity (Scheme 1C). Taking of these advantages, using the probe **DCI-ONOO-3**, we found that the concentration of ONOO⁻ in the epileptic mice was increased. Importantly, the antagonism of EDA on epilepsy at different concentrations was explored by observing ONOO⁻ levels in the brains of epileptic mice treated with EDA. This provides a compelling method for the accurate diagnosis of epilepsy and the antiepileptic evaluation of EDA.

Dicyanoisophorone derivatives are used extensively in the design of fluorescence probes on account of the advantages of typical D-π-A structure, easy synthesis modification. Thus, we used dicyanoisophorone as a fluorophore to synthesize the probe **DCI-ONOO-1** for the detection of ONOO⁻ (Scheme S1 in Supporting information). The presence of ONOO⁻ induced **DCI-ONOO-1** to remove (4-nitrophenylsulfide)carbonyl and restored the fluorescence emission of fluorophore (Scheme 1A). By introducing a strong electron-withdrawing group (trifluoromethyl) at the site of the phenol hydroxyl group, a new fluorophore is expected to emit NIR fluorescence (Scheme 1C and Fig. S2 in Supporting information). As far as we know, trifluoromethyl is widely used in the design of drugs for the treatment of brain diseases to improve lipophilicity and biocompatibility [30,31]. Therefore, we then developed the probes **DCI-ONOO-2** and **DCI-ONOO-3** for ONOO⁻ (Scheme S1). By screening, **DCI-ONOO-3** with the outstanding physicochemical and optical properties was used as the target probe to detect ONOO⁻. The sensing mechanism of the **DCI-ONOO-3** to ONOO⁻ was confirmed by HRMS and HPLC analysis. As shown in Fig. S6B (Supporting information), the peaks of 208.9831 ([M-H]⁻), 357.1221 ([M-H]⁻), 561.1082 ([M-H]⁻) were to be the compound **A**, **DCI-CF$_3$-OH**, and the probe **DCI-ONOO-3**, respectively, demonstrating that successful release of fluorophore **DCI-CF$_3$-OH** and compound **A** caused by the reaction of **DCI-ONOO-3** and ONOO⁻. Moreover, the results of HPLC analysis showed that the retention times of **DCI-ONOO-3**, **DCI-CF$_3$-OH** and compound **A** were 49.5, 11.6 and 34.4 min, respectively. Then, with addition of ONOO⁻ (0.5 equiv.) to the solution of **DCI-ONOO-3**, the peak at 49.5 min decreased, the peaks at 11.6 and 34.4 min were increased, corresponding to **DCI-ONOO-3**, **DCI-CF$_3$-OH** and compound **A**. Upon adding excessive ONOO⁻, the peak at 49.5 min (**DCI-ONOO-3**) disappeared, meanwhile, the peaks at 11.6 min (**DCI-CF$_3$-OH**) and 34.4 min (**A**) increased. The above results indicated that **DCI-ONOO-3** was triggered by ONOO⁻, restoring the fluorescence of **DCI-CF$_3$-OH** (Fig. S6C in Supporting information).

The spectral properties of the **DCI-ONOO-3** for ONOO⁻ were systematically performed in the CH_3CN/PBS buffer (10 mmol/L,

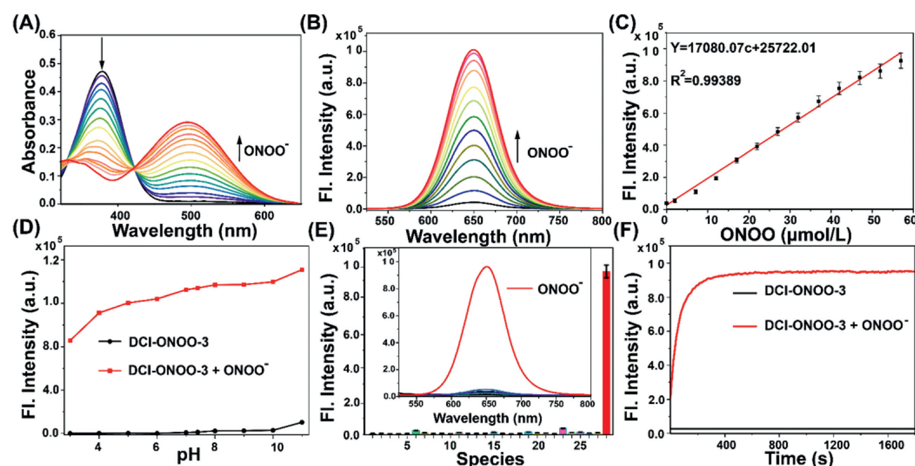


Fig. 1. (A) Absorption and (B) fluorescence titration of **DCI-ONOO-3** (10 μmol/L) to $ONOO^-$ (60 μmol/L) in phosphate buffered saline (PBS) (10 mmol/L, pH 7.4, containing 40% MeCN) solution. (C) Fluorescence intensity *versus* $ONOO^-$ concentration at 652 nm. (D) Fluorescence spectral response of **DCI-ONOO-3** (10 μmol/L) to $ONOO^-$ (10 equiv.) in pH 3–11. (E) Fluorescence response of **DCI-ONOO-3** (10 μmol/L) with addition of $ONOO^-$ (60 μmol/L) and other species (1 mmol/L). (F) The effect of reaction time between **DCI-ONOO-3** (10 μmol/L) and $ONOO^-$ (100 μmol/L). $\lambda_{ex} = 500$ nm, slit/slit = 2/2 nm.

pH 7.4, 2/5, v/v). The optical titration experiments of **DCI-ONOO-3** (10 μmol/L) toward $ONOO^-$ (0–60 μmol/L) were carried out. As shown in Fig. 1A, the absorption peak decreased at 380 nm and increased at 500 nm after adding $ONOO^-$. Correspondingly, 61-fold fluorescence enhancement at 652 nm can be observed with an increase of $ONOO^-$ concentration (Fig. 1B), and a good linearity was obtained between the fluorescence intensity and the $ONOO^-$ concentration (0–60 μmol/L) with the limit of detection calculated to be around 73 nmol/L (Fig. 1C), illustrating the high sensitivity of **DCI-ONOO-3** for $ONOO^-$. Besides, The **DCI-ONOO-3** can detect $ONOO^-$ over a wide range of pH 5–10 (Fig. 1D), indicating that **DCI-ONOO-3** was suitable for $ONOO^-$ detection under complex physiological and biological condition. Moreover, the selectivity of **DCI-ONOO-3** for $ONOO^-$ was investigated, as shown in Fig. 1E, only addition with $ONOO^-$ generated obvious fluorescence enhancement, whereas addition of other species (Na^+, K^+, Fe^{2+}, Cu^{2+}, Ca^{2+}, Fe^{3+}, Mg^{2+}, Zn^{2+}, Cl^-, NO_3^-, CO_3^{2-}, SO_4^{2-}, HSO_3^-, Na_2S, SO_3^{2-}, NO, NO_2^-, H_2O_2, ClO^-, TBHP, $^1O_2$, $^•OH$, Cys, GSH, Hcy, NE) showed a negligible response. Finally, as shown in Fig. 1F, the time-dependent was performed, indicating fast response of **DCI-ONOO-3** to $ONOO^-$ in 210 s. All the above results illustrated that **DCI-ONOO-3** has high selectivity, sensitivity and fast response of $ONOO^-$, showing the possibility of **DCI-ONOO-3** for monitoring $ONOO^-$ in biosystem.

Encouraged by the excellent performance of **DCI-ONOO-3** for the detection of exogenous $ONOO^-$, we further explored the application of **DCI-ONOO-3** in the detection of intracellular $ONOO^-$. The cytotoxicity of **DCI-ONOO-3** for PC12 cells, SH-SY5Y cells and HT-22 cells was firstly performed by cell counting kit-8 (CCK8) method. As shown in Fig. S7 (Supporting information), the cells survival rate remained at about 82%, indicating that the **DCI-ONOO-3** presented low cytotoxicity when the concentration of **DCI-ONOO-3** up to 20 μmol/L. Then, **DCI-ONOO-3** was used to fluorescence image of $ONOO^-$ in HT22 cells. As shown in Figs. S8A and D (Supporting information), the fluorescence intensity was concentration-dependent with **DCI-ONOO-3**. Meanwhile, Fig. S8C (Supporting information) indicated the high sensitivity of **DCI-ONOO-3** for endogenous $ONOO^-$ by flow cytometry. Subsequently, we conducted the kinetic study of **DCI-ONOO-3** towards $ONOO^-$ within 300 s in HT22 cells, and the fluorescence intensity gradually enhanced and stabilized in 200 s, indicating a fast response of **DCI-ONOO-3** for $ONOO^-$ (Figs. S8B and E in Supporting information).

Furthermore, the levels of endogenous $ONOO^-$ under oxidative stress mediated by lipopolysaccharide (LPS) and 3-morpholinosydnonimine (SIN-1) were investigated, and the $ONOO^-$ fluxes were also visualized by adding *N*-acetylcysteine (NAC, a ROS inhibitor), Fe(III)tetrakis(1-methyl-4-pyridyl) (FeTMpyP, peroxynitrite deconstructor) and EDA, respectively. As shown in Fig. 2A, in contrast with the **DCI-ONOO-3** (10 μmol/L) incubated group, HT22 cells were pretreated with LPS (1 μg/mL) or SIN-1 (100 μmol/L), followed by incubation of **DCI-ONOO-3** (10 μmol/L), the increased intensity was observed. However, the cells were pretreated with LPS or SIN-1, followed by incubation of NAC (1 mmol/L), FeTMPyP (50 μmol/L) or EDA (150 μmol/L), the fluorescence intensity decreased, indicating that EDA has the potential of scavenging $ONOO^-$ in cells and alleviating oxidative stress.

Moreover, we established the cell model of epilepsy by incubating HT22 cells in Mg^{2+}-free media (ACSF, Artificial cerebro-spinal fluid (-Mg) [32] for studying antiepileptic efficacy of EDA. The HT22 cells of epilepsy were incubated with carbamazepine (CBZ, a clinical drug for epilepsy) and EDA respectively. Fig. 2B showed that the fluorescence intensity of the HT22 cells of epilepsy incubated with **DCI-ONOO-3** increased compared to the control group. Whereas, after pretreating the epilepsy model groups with CAZ and EDA, the fluorescence increased compared to the epilepsy model groups. The above results proved that EDA has the potential of anti-epilepsy.

Having verified the selectivity and sensitivity of the **DCI-ONOO-3** for $ONOO^-$ in living cells, as well as the good BBB permeability of the **DCI-ONOO-3** by establishing a PAMPA model [33], we aimed to establish a mouse model of epilepsy [34] suitable for the study of antiepileptic activity of EDA. In this study, all animal experiments were conducted in accordance with the protocol approved by the Declaration of Animal Welfare and Ethical Approval of Hebei University, using 5-week-old BALB/c purchased from the SPF (Beijing) BIO-TECHNOLOGY Co., Ltd. The appropriate time for KA (20 mg/kg) induction to establish epileptic mice was firstly selected as 0.5 h by Racine grading and fluorescence imaging of $ONOO^-$ levels in the mouse brain (Fig. S9 in Supporting information). Immediately, the mice were divided into four groups: (a) Control group (injection of **DCI-ONOO-3** for 0.5 h), (b) Epileptic group (KA-induced for 0.5 h, following injection of **DCI-ONOO-3** for 0.5 h), (c, d) CBZ or EDA treated epileptic mice groups (KA-induced for 0.5 h, then treated with CBZ or EDA for 0.5 h, following injection of **DCI-ONOO-3** for 0.5 h). As shown in Fig. 3, the fluorescence in-

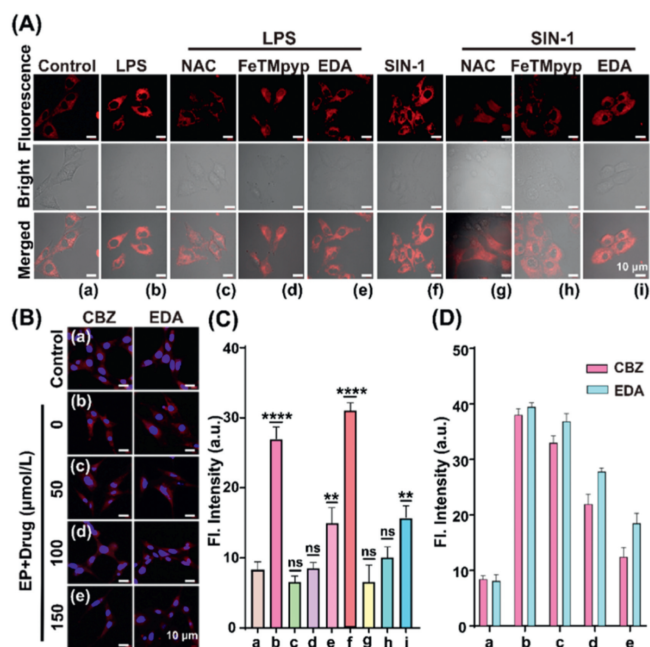


Fig. 2. (A) Fluorescence images of **DCI-ONOO-3** in HT22 cells. (a) HT22 cells were incubated with **DCI-ONOO-3** (10 μmol/L) for 20 min. (b–e) HT22 cells were pretreated with LPS (1 μg/mL) for 30 min, following incubation of none, or NAC (1 mmol/L), or FeTMpyp (50 μmol/L), or EDA (150 μmol/L) for 30 min, then incubated with **DCI-ONOO-3** (10 μmol/L) for 20 min. (f–i) HT22 cells were pretreated with SIN-1 (100 μmol/L) for 30 min, following incubation of none, or NAC (1 mmol/L), or FeTMpyp (50 μmol/L), or EDA (150 μmol/L) for 30 min, then incubated with **DCI-ONOO-3** (10 μmol/L) for 20 min, respectively. (B) Fluorescence images of HT22 cells of epilepsy. (a) Control group, HT22 cells were treated with **DCI-ONOO-3** (10 μmol/L). (b–e) The HT22 cells of epilepsy pretreated with EDA or CBZ (0, 50, 100, 150 μmol/L), followed by incubation of **DCI-ONOO-3** (10 μmol/L). (C) The fluorescence intensity of (A). (D) The relative fluorescence intensity of (C). $\lambda_{ex} = 561$ nm, $\lambda_{em} = 640–745$ nm. Scale bar: 10 μm. Data are presented as the mean value ± standard deviation (SD) ($n = 3$). $^{****}P \le 0.0001$, $^{**}P < 0.01$. ns, no significance.

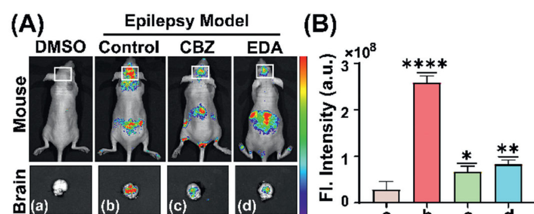


Fig. 3. (A) Top: fluorescence imaging of mice; Bottom: fluorescence imaging of corresponding cerebrum of mice. (a) Control group, only treated with **DCI-ONOO-3** (200 μmol/L) for 0.5 h. (b–d) The mice treated with none, or CBZ (50 mg/kg), or EDA (50 mg/kg) for 0.5 h after injection with KA (20 mg/kg) for 0.5 h, then injection with **DCI-ONOO-3** (200 μmol/L) for 0.5 h, respectively. $\lambda_{ex} = 510$ nm, $\lambda_{em} = 600–700$ nm. (B) The relative fluorescence intensity of (A). $\lambda_{ex} = 510$ nm, $\lambda_{em} = 600–700$ nm. Data are presented as the mean value ± SD ($n = 3$). $^{****}P \le 0.0001$, $^{**}P < 0.01$, $^{*}P < 0.05$.

tensity of epileptic group increased in comparison to control group. However, compared to epileptic group, the fluorescence intensity of CBZ or EDA treated epileptic mice groups decreased to some extent, indicating the good antiepileptic activity of EDA. This provides a promising method for the clinical treatment of epilepsy.

In summary, we focused on developing a novel fluorescent probe passing through the BBB to monitor $ONOO^-$ in the brain. The rational strategy for designing probe **DCI-ONOO-3** is proposed by introducing trifluoromethyl groups, solving the requirements of NIR emission and lipophilicity of probe for brain imaging. The **DCI-ONOO-3** has high signal-to-noise ratio and BBB penetration, achieving real-time monitoring of $ONOO^-$ fluctuations in the epileptic mice brains and evaluating antiepileptic effect of EDA. This study provides a new strategy for exploring the pathogenesis of epilepsy, which helps to better understand the pathology of epilepsy. It also provides a new reference for clinical treatment of epilepsy, and accelerates the discovery of potential anti-epileptic drugs.

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.

Acknowledgments

The work supported by the National Natural Science Foundation of China (Nos. 22177025, 21807021, 22325703, 22177026), Science Fund for Creative Research Groups of Nature Science Foundation of Hebei Province (No. B2021201038), the Interdisciplinary Research Program of Natural Science of Hebei University (No. DXK202113), the Central Government Guided Local Science, Technology Development Fund (Hebei Province, No. 216Z2603G), National High-End Foreign Expert Recruitment Plan (No. G2022003007L).

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.cclet.2024.109572.

References

[1] P. Kwan, M.J. Brodie, N. Engl. J. Med. 342 (2000) 314–319.
[2] R.D. Thijs, R. Surges, T.J. O'Brien, J.W. Sander, Lancet 393 (2019) 689–701.
[3] E. Perucca, Epilepsia 55 (2014) 473–474.
[4] N.T. El Tantawi, D.S. Abd Elmegid, E. Atef, Seizure 65 (2019) 166–171.
[5] J.K. Knowles, H. Xu, C. Soane, et al., Nat. Neurosci. 25 (2022) 596–606.
[6] J. Goldstein, C.S. Kwon, M. Harmon, et al., Epilepsy Behav. 125 (2021) 108383.
[7] P.G. Winyard, C.J. Moody, C. Jacob, Trends Biochem. Sci. 30 (2005) 453–461.
[8] M.R. Ashrafi, S. Shams, M. Nouri, et al., Epilepsia 48 (2007) 1750–1755.
[9] H. Li, X. Li, X. Wu, W. Shi, H. Ma, Anal. Chem. 89 (2017) 5519–5525.
[10] N. Ma, M. Sasoh, S. Kawanishi, H. Sugiura, F.J. Piao, Biomed. Sci. 17 (2010) S7.
[11] N. Ahmed, W. Zareen, Y. Ye, Chin. Chem. Lett. 33 (2022) 2765–2772.
[12] D.H. Ma, S.M. Hou, C. Bae, et al., Chin. Chem. Lett. 32 (2021) 3886–3889.
[13] T. Obata, Neural. Transm. 113 (2006) 1131–1144.
[14] N.N. Wang, H. Wang, J. Zhang, et al., Chin. Chem. Lett. 33 (2022) 1584–1588.
[15] Y.T. Yang, Y.Y. Zhang, M. Ma, et al., Anal. Chem. 94 (2022) 14443–14452.
[16] R.S. Fisher, C. Acevedo, A. Arzimanoglou, et al., Epilepsia 55 (2014) 475–482.
[17] K. Takei, K. Watanabe, S. Yuki, et al., Amyotroph. Lateral Scler. Frontotemporal Degener. 18 (2017) 5–10.
[18] N. Tanahashi, Y. Fukuuchi, Intern. Med. J. 41 (2002) 337–344.
[19] B. Daly, J. Ling, A.P. De Silva, Chem. Soc. Rev. 44 (2015) 4203–4211.
[20] Y. Fu, N.S. Finney, RSC Adv. 8 (2018) 29051–29061.
[21] J.V. Jun, D.M. Chenoweth, E.J. Petersson, Org. Biomol. Chem. 18 (2020) 5747–5763.
[22] D. Wu, A.C. Sedgwick, T. Gunnlaugsson, et al., Chem. Soc. Rev. 46 (2017) 7105–7123.
[23] M. Yang, J. Fan, J. Du, X. Peng, Chem. Sci. 11 (2020) 5127–5141.
[24] H.H. Han, H. Tian, Y. Zang, et al., Chem. Soc. Rev. 50 (2021) 9391–9429.
[25] N. Karton-Lifshin, E. Segal, L. Omer, et al., J. Am. Chem. Soc. 133 (2011) 10960–10965.
[26] P. Li, J. Wang, X. Wang, et al., Chem. Sci. 10 (2019) 2805–2810.
[27] S. Siriwibool, N. Kaekratoke, K. Chansaenpak, et al., Sci. Rep. 10 (2020) 1283.
[28] W. Liu, C. Fan, R. Sun, Y. Xu, J. Ge, Org. Biomol. Chem. 13 (2015) 4532–4538.
[29] J.J. Willard, E. Pacsu, J. Am. Chem. Soc. 82 (1960) 4347–4350.
[30] O.A. Tomashenko, V.V. Grushin, Chem. Rev. 111 (2011) 4475–4521.
[31] X. Wang, D. Su, C. Liu, et al., Anal. Chem. 94 (2022) 14965–14973.
[32] S. Sombati, R.J.J. Delorenzo, J. Neurophysiol. 73 (1995) 1706–1711.
[33] X. Zhang, T. Liu, X. Fan, N. Ai, Mol. Graph. Model. 75 (2017) 347–354.
[34] G. Marsicano, S. Goodenough, K. Monory, et al., Science 302 (2003) 84–88.