FIRST JUDICIAL DISTRICT OF PENNSYLVANIA
COURT OF COMMON PLEAS OF PHILADELPHIA

[redacted]

[redacted]

NOTICE TO DEFEND

NOTICE

You have been sued in court. If you wish to defend against the claims set forth in the following pages, you must take action within twenty (20) days after this complaint and notice are served, by entering a written appearance personally or by attorney and filing in writing with the court your defenses or objections to the claims set forth against you. You are warned that if you fail to do so the case may proceed without you and a judgment may be entered against you by the court without further notice for any money claimed in the complaint of for any other claim or relief requested by the plaintiff. You may lose money or property or other rights important to you.

You should take this paper to your lawyer at once. If you do not have a lawyer or cannot afford one, go to or telephone the office set forth below to find out where you can get legal help.

Philadelphia Bar Association
Lawyer Referral
and Information Service
1101 Market St., 11th Floor
Philadelphia, Pennsylvania 19107
(215) 238-6333

AVISO

Le han demandado a usted en la corte. Si usted quiere defenderse de estas demandas expuestas en las paginas siguientes, usted tiene veinte (20) dias de plazo al partir de la fecha de la demanda y la notificacion. Hace falta ascentar una comparencia escrita o en persona o con un abogado y entregar a la corte en forma escrita sus defensas o sus objeciones a las demandas en contra de su persona. Sea avisado que si usted no se defiende, la corte tomara medidas y puede continuar la demanda en contra suya sin previo aviso o notificacion. Ademas, la corte puede decider a favor del demandante y requiere que usted cumpla con todas las provisiones de esta demanda. Usted puede perder dinero o sus propiedades u otros derechos importantes para usted.

Lleve esta demanda a un abogado immediatamente. Si no tiene abogado o si no tiene el dinero suficiente de pagar tal servicio. Vaya en persona o llame por telefono a la oficina cuya direccion se encuentra escrita abajo para averiguar donde se puede conseguir asistencia legal.

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10-284

PHILADELPHIA COUNTY, PENNSYLVANIA
COURT OF COMMON PLEAS

DOMENICO PRATICO [redacted] Philadelphia, PA 19130 **Plaintiff,** **v.** **PHILLIP GIANNOPOULOS** [redacted] Philadelphia, PA 19111 **Defendant.**	**NO. _____** **JURY TRIAL DEMANDED**

COMPLAINT

Plaintiff, Domenico Praticò (Dr. Praticò) through his undersigned counsel, complains against Phillip Giannopoulos, (Dr. Giannopoulos) as follows:

This matter involves the falsification of research data by Defendant, and intentional representations by Defendant to induce Plaintiff to rely on such data to Plaintiff's detriment.

PARTIES

1. Dr. Praticò is an individual who resides at [redacted] Philadelphia, PA 19130.

2. Upon information and belief, Dr. Giannopoulos is an individual who resides at [redacted] Philadelphia, PA 19111 in Philadelphia, Pennsylvania.

VENUE

3. Venue is proper in this Court pursuant to Pennsylvania Rules of Civil Procedure 2179(a)(3) and (4) because the causes of action asserted hereunder arose from transactions and occurrences in Philadelphia County.

FACTS

4. Dr. Praticò is a tenured Professor of Neural Sciences and the Director of the Alzheimer's Center at Temple University.

5. Dr. Praticò's research area is clinical pharmacology with a special focus on the cellular and molecular aspects of cell oxidative biology and a particular interest in small molecules such as bioactive oxidized lipids.

6. Dr. Praticò has authored or participated in authoring over 300 articles, and chapters of several books.

7. Dr. Praticò is the principal researcher in the Praticò Lab which is part of the School of Medicine at Temple University.

8. Students at Temple University use the Praticò Lab to research diseases of the brain, including those that affect memory, language, and the ability to learn.

9. Dr. Praticò served as doctoral advisor to Dr. Giannopoulos during his doctoral program at Temple University. Dr. Giannopoulos received his Ph.D. from Temple University in 2015.

10. Dr. Giannopoulos used Dr. Praticò's lab and certain testing equipment that is housed in the animal facility outside of Dr. Praticò's lab to perform studies that constituted original research ("the Giannopoulos Data"), which Dr. Giannopoulos used for his doctoral dissertation at Temple University.

11. At the time Dr. Giannopoulos received his Ph.D., neither Dr. Praticò nor any other person on his dissertation committee, nor the external reviewer was aware of issues or problems with the Giannopoulos Data. There were three permanent members of the thesis committee.

12. Springer Nature Academic Publishing, Inc. ("Springer Nature") published two articles (hereinafter "the Articles") in which Dr. Praticò and Dr. Giannopoulos were named as authors:

a. Overexpression of 5-lipoxygenase Worsens the Phenotype of a Mouse Model of Tauopathy. Mol Neurobiol 2018: 55(7):5926-5936;

b. Learning Impairments, Memory Deficits, and Neuropathology in Aged Tau Transgenic Mice Are Dependent on Leukotrienes Biosynthesis: Role of the cdk5 Kinase Pathway. Mol Neurobiol. 2019 Feb; 56(2):1211-1220

13. Dr. Praticò was the primary author of the Articles.

14. The Articles contains Giannopoulos Data.

15. Prior to submitting the articles to Springer Nature, Dr. Praticò spoke to Dr. Giannopoulos about the publications; in 2015, Dr. Giannopoulos had prepared and formatted text and figures for submission of the article to a different journal, which ended up not publishing the material. *See* **Exhibits A and B**.

16. Dr. Giannopoulos was aware of the publication of the Articles and used the Articles in his public profile on ResearchGate. *See* **Exhibit C**.

17. In addition, the Articles was listed in Dr. Giannopoulos' public profile on pubmed. *See* **Exhibit D**.

18. In March of 2020, after a website name "Pubpeer" posted comments challenging the Giannopoulos Data as set forth in the Articles, Dr. Praticò emailed Dr. Giannopoulos regarding the data.

19. In 2020, after Pubpeer criticized the Giannopoulos Data, Dr. Giannopoulos did not challenge authorship.

20. Upon information and belief, in 2023, Dr. Giannopoulos represented, through his attorney, to the Associate Editor and Publisher at Springer Nature that Dr. Giannopoulos was 'unaware of the submission and publication' of the Article, and that he did not consent to the publication of his data.

21. This representation directly conflicts with Dr. Giannopoulos' ResearchGate and pubmed profiles, in which he indicates he is an author.

22. In 2023, after receiving feedback from peers in the academic community, Dr. Praticò conducted a very careful review of Dr. Giannopoulos' source data, and of Dr. Giannopoulos' dissertation, and is now extremely concerned about the integrity of the Giannopoulos Data.

23. To Dr. Praticò's knowledge, Dr. Giannopoulos never expressed any concern to any person about authorship of the Article until the year 2023. Prior to the year 2023, Dr. Giannopoulos did not ever advise Dr. Praticò that he disagreed with the use of the Giannopoulos Data in the Article, or that he was unaware of its submission or publication.

COUNT I- DEFAMATION

24. All preceding allegations are incorporated herein by reference.

25. Because Dr. Giannopoulos' attorney is his agent, the defamatory statement by the attorney constitutes a statement by Dr. Giannopoulos.

26. Dr. Giannopoulos' made a false statement of fact when his attorney represented to Springer Nature Journal that Dr. Giannopoulos was unaware that he was named as an author of the Article and when he claimed that he did not consent to being in the authorship for the Article.

27. Dr. Giannopoulos' statement to Springer Nature that he did not consent to the publication of the Article is a statement of defamatory character, which blackened Dr. Praticò's reputation, exposed him to public contempt, and injured his business or profession.

28. Moreover, the statement tends to lower Dr. Praticò's estimation in the community and deter people from associating or dealing with him.

29. The defamatory statement was published when it was made to Springer Nature Journal.

30. The recipient of the defamatory statement understood its defamatory meaning, and understood the statement was intended to be applied to Dr. Praticò.

31. The defamatory statement is not subject to any privilege, or alternatively, to the extent there is a privilege, the privilege was abused.

32. Upon information and belief, when Dr. Giannopoulos, by his counsel, made the false statements, he intended to make them.

33. Dr. Praticò has suffered actual injury as a result of the defamatory statement including an impairment to his reputation and standing in the academic community, personal humiliation, and mental anguish and suffering.

WHEREFORE, Plaintiff respectfully requests (i) judgment be entered in its favor and against Defendant; (ii) an award be entered in an amount in excess of $50,000, plus attorney's fees, costs and interest; and (iii) the Court grant any further relief deemed appropriate under the circumstances.

COUNT II- FRAUD

34. All preceding allegations are incorporated herein by reference.

35. Dr. Giannopoulos represented to Dr. Praticò on at least two occasions that his data was reliable for scholarly publications, first when he used the data in his dissertation, and second in 2015, when he prepared and formatted text and figures for submission of the article to a journal.

36. Dr. Giannopoulos' misrepresentations were material to the matter at hand and Dr. Praticò continued to rely on the representations when Dr. Praticò submitted the same text and figures to Springer Nature for publication.

37. Dr. Giannopoulos developed the false data knowingly, and with knowledge of his deception.

38. After concerns were raised about the data, in 2023, a third-party expert used specialized software to review the Giannopoulos Data, and found that Dr. Giannopoulos had:

a. duplicated, altered, and improperly reused data;

b. appropriated experimental images from other sources not authored by himself, mislabeling them and then presenting them in the thesis as a purported result of his own research activity; and

c. mislabeled images in an inconsistent way and reusing them to prepare experimental material in support of several research behavior.

39. Dr. Giannopoulos intended for Dr. Praticò to believe in the veracity and accuracy of the Giannopoulos Data both when Dr. Giannopoulos received his Ph.D. and when Dr. Giannopoulos responded to an email encouraging publication of the data.

40. Dr. Praticò relied on the Giannopoulos Data when he submitted Articles containing the Giannopoulos Data to various journals for publication.

41. Dr. Praticò was justified in relying on Dr. Giannopoulos. Neither Dr. Praticò nor anyone else on Dr. Giannopoulos' dissertation committee were aware of problems with the Giannopoulos Data until well after Dr. Giannopoulos received his doctorate and Dr. Pratico submitted the articles containing the Giannopoulos Data for publication.

42. Dr. Praticò has been damaged by this fraudulent conduct. Dr. Giannopoulos' reckless behavior profoundly polluted several manuscripts prepared by Dr. Praticò's lab, and thereby undermining Dr. Praticò's scientific reputation.

43. Dr. Praticò has suffered actual injury as a result of the defamatory statement including an impairment to his reputation and standing in the academic community, personal humiliation, and mental anguish and suffering.

WHEREFORE, Plaintiff respectfully requests (i) judgment be entered in its favor and against Defendant; (ii) an award be entered in an amount in excess of $50,000, plus attorney' s fees, costs and interest; and (iii) the Court grant any further relief deemed appropriate under the circumstances.

WISLER PEARLSTINE, LLP

Dated: January 9, 2024

By: */s/ Deborah R. Stambaugh*
Christopher E. Ezold, Esquire
Deborah R. Stambaugh, Esquire
Attorneys for Plaintiff

EXHIBIT “A”

Htau-5LO AAV manuscript

Phillip Giannopoulos [redacted]

Fri 1/9/2015 12:54 PM

To:DOMENICO PRATICO <praticod@temple.edu>

8 attachments (8 MB)

htau 5LO AAV- figure 7 in vitro 1-9-15.ppt; htau 5LO AAV figure 6 in vitro 1-9-15.ppt; htau-5LO AAV figure 5 inflam 1-9-15.ppt; htau 5LO AAV-figure 4 syp 1-9-15.ppt; htau 5LO AAV figure 3 insoluble tau-kinases 1-9-15.ppt; htau 5LO AAV figure 2-ptau 1-9-15.ppt; htau 5LO AAV behavior figure 1- 1-9-15.ppt; htau 5LO AAV manuscript 1-9-15 nature neuroscience.doc;

Here is the text formatted for nature neuroscience and all the figures (1-7)

Phillip-Electronic Files-1

Phillip Giannopoulos <[redacted]>
Fri 3/13/2015 12:32 PM
To:DOMENICO PRATICO <praticod@temple.edu>

3 attachments (20 MB)
htau-5LO AAV.zip; htau zileuton regression.zip; htau-12LO.zip;

Phillip-Electronic Files- 2

Phillip Giannopoulos [redacted]
Fri 3/13/2015 12:32 PM
To:DOMENICO PRATICO <praticod@temple.edu>

3 attachments (20 MB)
12LO tau.zip; 3xTg-FLAP.zip; P301S-zileuton.zip;

Phillip-Electronic Files-3

Phillip Giannopoulos [redacted]
Fri 3/13/2015 12:32 PM
To:DOMENICO PRATICO <praticod@temple.edu>

2 attachments (21 MB)
P301S-5LO.zip; htau-zileuton.zip;

Re: greetings et al

Phillip Giannopoulos [redacted]
Thu 4/13/2017 12:49 PM

To:DOMENICO PRATICO <praticod@temple.edu>

I believe they have should at least have my initials on them. If there are any questions let me know. Also, Alana can take a picture on her phone of the box cover and message and I will be able to tell if it's my handwriting.

On Thu, Apr 13, 2017 at 12:23 PM, DOMENICO PRATICO <praticod@temple.edu> wrote:

> Phillip,
>
> thanks for your prompt response. We'll look in the freezer, I am sure the boxes will have your name also?
>
> Best
>
> On Thu, Apr 13, 2017 at 12:16 PM, Phillip Giannopoulos <[redacted]> wrote:
>
> > Hey Dr. Pratico,
> >
> > Great to hear from you. Excited to hear that Alana is prepping to submit. You can leave the Temple affiliation since I performed the work there.
> >
> > As for the biochemistry files, I will take a look at my external hard drive at home in Philadelphia over the weekend where I have the files saved to see exactly what I have and compare it to what I sent you before I left. I will send you over any necessary files.
> >
> > In the case I do not have the biochemistry data, I spoke with Alana and told her to check the freezers for the P301S/5LOAAV and P301S/zileuton tissue. I believe it is in the newer of the two freezers but she will double check for me.
> >
> > Hope things are well with you and we'll be in touch soon.
> >
> > On Thu, Apr 13, 2017 at 11:51 AM, DOMENICO PRATICO <praticod@temple.edu> wrote:
> >
> > > Hey Phillip,
> > >
> > > How are you?I hope this message finds you well!!
> > > Two things:
> > >
> > > 1. Alana is putting together her data on the P301S 5LO KO mice and since she is using some of your data showing the age/region dependent changes in the 5LO levels you will be in the authorship.
> > > Do you want us to put your current address/work? or just leave the Temple affiliation since the work was performed here?

2. In reviewing the files you gave me with all the unpublished work I noticed that among the different studies, the P301S treated with zileuton and the P301S with aav5LO do not have any biochemistry data.
Can you please tell me where we can find the tissues of these studies? We have a new person in the lab that could complete them.

Looking forward to hearing back form you.

Domenico

--

--
Domenico Pratico, MD
Professor of Pharmacology, Immunology and Microbiology
Center for Translational Medicine
Temple University
3500 North Broad Street
947 MERB
Philadelphia, PA 19140
Tel, 215-707-9380
Fax, 215-707-9890

P301S zileuton and P3015LO

Phillip Giannopoulos [redacted]

Sun 4/16/2017 11:03 PM

To:DOMENICO PRATICO <praticod@temple.edu>

3 attachments (1 MB)

P301S lab meeting 7-21-14 kinases-5LO 12 monthsl.ppt; P301S zileuton figure 7-14-14.ppt; P301S zileuton syp-inflam 7-28-14.ppt;

Hey Dr. Pratico,

Below are all the biochemistry files for the P301 zileuton study.It includes the ptau, insoluble tau, kinases, synaptic and inflammatory markers. For P301S 5LO i could not find any files on the external.hard drive. I will continue to look and see if I find anything. i do not recall completing the western blot for that study. I will continue to look and if i come up with any files, i will send them your way. Anything else you need let me know. Hope you had a wonderful Easter!!

Talk to you soon

EXHIBIT “B”

Htau-5LO AAV manuscript

Phillip Giannopoulos [redacted]

Fri 1/9/2015 12:54 PM

To:DOMENICO PRATICO <praticod@temple.edu>

8 attachments (8 MB)

htau 5LO AAV- figure 7 in vitro 1-9-15.ppt; htau 5LO AAV figure 6 in vitro 1-9-15.ppt; htau-5LO AAV figure 5 inflam 1-9-15.ppt; htau 5LO AAV-figure 4 syp 1-9-15.ppt; htau 5LO AAV figure 3 insoluble tau-kinases 1-9-15.ppt; htau 5LO AAV figure 2-ptau 1-9-15.ppt; htau 5LO AAV behavior figure 1- 1-9-15.ppt; htau 5LO AAV manuscript 1-9-15 nature neuroscience.doc;

Here is the text formatted for nature neuroscience and all the figures (1-7)

EXHIBIT “C”

Article Publisher preview available

Learning Impairments, Memory Deficits, and Neuropathology in Aged Tau Transgenic Mice Are Dependent on Leukotrienes Biosynthesis: Role of the cdk5 Kinase Pathway

February 2019 · Molecular Neurobiology 56(1):1-10
DOI:10.1007/s12035-018-1124-7

Authors:

Phillip Fotios Giannopoulos
Temple University

Jian Chiu

Domenico Praticò

Abstract and Figures

Previous studies showed that the leukotrienes pathway is increased in human tauopathy and that its manipulation may modulate the onset and development of the pathological phenotype of tau transgenic mice. However, whether interfering with leukotrienes biosynthesis is beneficial after the behavioral deficits and the neuropathology have fully developed in these mice is not known. To test this hypothesis, aged tau transgenic mice were randomized to receive zileuton, a specific leukotriene biosynthesis inhibitor, or vehicle starting at 12 months of age for 16 weeks and then assessed in their functional and pathological phenotype. Compared with baseline, we observed that untreated tau mice had a worsening of their memory and spatial learning. By contrast, tau mice treated with zileuton had a reversal of these deficits and behaved in an undistinguishable manner from wild-type mice. Leukotriene-inhibited tau mice had an amelioration of synaptic integrity, lower levels of neuroinflammation, and a significant reduction in tau phosphorylation and pathology, which was secondary to an involvement of the cdk5 kinase pathway. Taken together, our findings represent the first demonstration that the leukotriene biosynthesis is functionally involved at the later stages of the tau pathological phenotype and represents an ideal target with viable therapeutic potential for treating human tauopathies.

Chronic administration … | Chronic administration … | Leukotrienes biosynthesis… | Leukotriene reduction… | Anti-leukotriene therapy…

Molecular Neurobiology (2019) 56:1211–1220
https://doi.org/10.1007/s12035-018-1124-7

Case ID: 240101083

Learning Impairments, Memory Deficits, and Neuropathology in Aged Tau Transgenic Mice Are Dependent on Leukotrienes Biosynthesis: Role of the cdk5 Kinase Pathway

Phillip F. Giannopoulos[1] · Jian Chiu[1] · Domenico Praticò[1]

Received: 3 April 2018 /Accepted: 11 May 2018 /Published online: 7 June 2018

Abstract
Previous studies showed that the leukotrienes pathway is increased in human tauopathy and that its manipulation may modulat the onset and development of the pathological phenotype of tau transgenic mice. However, whether interfering with leukotriene biosynthesis is beneficial after the behavioral deficits and the neuropathology have fully developed in these mice is not known. T test this hypothesis, aged tau transgenic mice were randomized to receive zileuton, a specific leukotriene biosynthesis inhibitor, c vehicle starting at 12 months of age for 16 weeks and then assessed in their functional and pathological phenotype. Compare with baseline, we observed that untreated tau mice had a worsening of their memory and spatial learning. By contrast, tau mic treated with zileuton had a reversal of these deficits and behaved in an undistinguishable manner from wild-type mice Leukotriene-inhibited tau mice had an amelioration of synaptic integrity, lower levels of neuroinflammation, and a significar reduction in tau phosphorylation and pathology, which was secondary to an involvement of the cdk5 kinase pathway. Take together, our findings represent the first demonstration that the leukotriene biosynthesis is functionally involved at the later stage of the tau pathological phenotype and represents an ideal target with viable therapeutic potential for treating human tauopathies

Keywords Tauopathy · cdk5 kinase pathway · Five-lipoxygenase · Leukotrienes · Neuroinflammation · Behavior

Introduction

Neurodegenerative diseases represent a large and heterogeneous group of chronic disorders both sporadic and familial, often characterized by the progressive accumulation of signature protein aggregates, which in most cases provide the basis for their neuropathological classification [1]. To this end, the term "tauopathies" is typically used to define some of these diseases whose main feature is the presence of filamentous accumulations of highly phosphorylated tau protein only in neurons or neurons and glial cells [2, 3]. They comprise several different clinical and pathological entities and have been sub-classified into primary and secondary, depending on whether tau neuropathology is considered the major contributing factor to the pathogenesis or simply associated with it [4].

✉ Domenico Praticò
praticod@temple.edu

[1] Alzheimer's Center at Temple, Lewis Katz School of Medicine, Scott Richards North Star Foundation Chair, Alzheimer's Research, Temple University, 947, Medical Education and Research Building, 3500 North Broad Street, Philadelphia, PA 19140, USA

While pathological post-translational modifications of tau have unequivocally been shown to be able to cause neurode generation, the precise molecular and cellular mechanism whereby this protein is involved in the pathogenesis of thes diseases are still poorly understood. Interestingly, besides th accumulation of highly phosphorylated tau, its filaments, an ultimately the neurofibrillar tangles, consistent evidence ha demonstrated that both human tauopathies as well as thei animal models are also characterized by intense humoral an cellular neuroinflammatory responses [5, 6].

We recently showed that post-mortem brain tissues from sub jects with a clinical and pathological diagnosis of progressiv supranuclear palsy, one of the most common form of tauopathy have a significant up-regulation of the 5-lipoxygenase (5LO), a enzyme whose metabolic products, the leukotrienes, are poter pro-inflammatory lipid mediators [7, 8]. Further, in relevar mouse models of tauopathy genetic absence or early pharmacc logical blockade of 5LO activation resulted in significant im provement of behavioral deficits and delay in the developmer of tau phosphorylation and pathology [7, 9, 10]. However, a these studies are to be considered as preventative in nature sinc all of them have used mice at an early stage of their phenotyp

... Cdk5 is also associated with early inflammation of AD (Wilkaniec et al., 2018). Leukotriene, an inflammatory factor, may accelerate tau pathological accumulation through the Cdk5 pathway (Giannopoulos et al., 2019) . Tau affects the function of subcellular organelles, such as mitochondria and The role of Cdk5 in neurological disorders and the underlying molecular mechanisms. ...

The role of Cdk5 in neurological disorders

Article Full-text available

Jul 2022

Chuncao Ao · Chenchen Li · Jinlun Chen · Liuwang Zeng

View Show abstract

... More edges in Bayesian Network are at S2 Further investigation is needed for the mixed effects of inflammation, such as considering the confounding effect of inflammation itself and/or medications related to inflammation. For example, Zileuton, a leukotriene biosynthesis inhibitor that is widely used for chronic inflammation (asthma), has shown a significantly reduced level of neuroinflammation and in tau phosphorylation in the tau transgenic mice [65] . ...

Counterfactual analysis of differential comorbidity risk factors in Alzheimer's disease and related dementias

Article Full-text available

Mar 2022

Yejin Kim · Kai Zhang · Sean I Savitz · Xiaoqian Jiang

View Show abstract

... Secondly, we analyzed pharmacokinetics of MTK in a transgenic mouse model of AD, because recently it was shown that pharmacological inhibition of leukotriene signaling had beneficial effects in several mouse models of AD [30][31][32] [33] [34] . The leukotriene receptor antagonist MTK has been proposed as an interesting candidate for drug repurposing in AD patients [13][14][15], due to its potential to modulate neuroinflammation and improve memory in animal models of stroke [35], epilepsy [36,37] and Lewy body dementia [38]. ...

Improved Bioavailability of Montelukast through a Novel Oral Mucoadhesive Film in Humans and Mice

Article Full-text available

Dec 2020

Johanna Michael · Diana M. Bessa de Sousa · Justin Conway · Ludwig Aigner

View Show abstract

... Consistent with previous studies, our results showed that AMI inhibits the expression of the tau upstream kinase GSK-3β. In addition, tau protein kinase II (TPKII) formed by a complex containing two subunits of Cyclin-dependent Kinase 5 (CDK5) and p35 can synergistically increase the efficiency of GSK-3β phosphorylation of tau protein (Xiao et al., 2018; Giannopoulos et al., 2019) . Therefore, we need to investigate the expression of tau protein kinase II (TPKII) in the next experiment. ...

AMI, an Indazole Derivative, Improves Parkinson's Disease by Inhibiting Tau Phosphorylation

Article Full-text available

Nov 2020

Zhang Mao · Zhu Wen-ting · Wang Hai-tao · Wang Wen-ya

View Show abstract

... RIPA extracts from human and mouse brain homogenates were used for western blot analyses as previously described [16, 23] . In brief, samples were electrophoresed on 10% Bis-Tris gels or 3-8% Tris-acetate gel (Bio-Rad, Richmond, CA), transferred onto nitrocellulose membranes (Bio-Rad), and then incubated overnight at 4°C with the appropriate primary antibodies; anti-VPS35 [dilution: After three washings with T-TBS (pH 7.4), membranes were incubated with IRDye 800CW-labeled secondary antibodies (LI-COR Bioscience, Lincoln, NE) at room temperature for 1 h. ...

VPS35 regulates tau phosphorylation and neuropathology in tauopathy

Article Full-text available

Jul 2019

Alana N. Vagnozzi · Jian-Guo Li · Jin Chiu · Domenico Praticò

View Show abstract

Leukotriene A4 hydrolase inhibition improves age-related cognitive decline via modulation of synaptic function

Article

Nov 2023

Julia M. Adams · Sanket Rege · Angela T. Liu · Meghan Kerrisk Campbell

View Show abstract

Microglia in neurodegenerative diseases: mechanism and potential therapeutic targets

Article Full-text available

Sep 2023

Chao Gao · Jingwen Jiang · Yuyan Tan · Shengdi Chen

View Show abstract

Lipids and brain inflammation in APOE4-associated dementia

Article

Dec 2021

Marlon Vincent Venancio Duro · Brandon Ebright · Hussein N Yassine

View Show abstract

5-Lipoxygenase as an emerging target against age-related brain disorders

Article

May 2021 · AGEING RES REV

Mengdie Yan · Siran Zhang · Chengtan Li · Lihui Zhang

View Show abstract

Leukotrienes in Tumor-Associated Inflammation

Article Full-text available

Aug 2020

Wen Tian · Xinguo Jiang · Dongeon Kim · Stanley G Rockson

View Show abstract

Show more

Case ID: 240101083

Phillip Fotios Giannopoulos · Domenico Praticò

View full-text

Last Updated: 12 Jun 2023

EXHIBIT “D”

21 results

1 The 12/15-lipoxygenase as an emerging therapeutic target for Alzheimer's disease.
Joshi YB, **Giannopoulos PF**, Praticò D.
Trends Pharmacol Sci. 2015 Mar;36(3):181-186. doi: 10.1016/j.tips.2015.01.005. Epub 2015 Feb 20.
PMID: 25708815 Free PMC article. Review.

2 Retraction Note to: Overexpression of 5-Lipoxygenase Worsens the Phenotype of a Mouse Model of Tauopathy.
Giannopoulos PF, Praticò D.
Mol Neurobiol. 2023 May;60(5):2970-2971. doi: 10.1007/s12035-023-03283-1.
PMID: 36823264 No abstract available.

3 Novel lipid signaling pathways in Alzheimer's disease pathogenesis.
Giannopoulos PF, Joshi YB, Praticò D.
Biochem Pharmacol. 2014 Apr 15;88(4):560-4. doi: 10.1016/j.bcp.2013.11.005. Epub 2013 Nov 21.
PMID: 24269629 Free PMC article. Review.

4 Retraction Note to: Gene knockout of 5-lipoxygenase rescues synaptic dysfunction and improves memory in the triple-transgenic model of Alzheimer's disease.
Giannopoulos PF, Chu J, Joshi YB, Sperow M, Li JG, Kirby LG, Praticò D.
Mol Psychiatry. 2023 Feb;28(2):964. doi: 10.1038/s41380-022-01813-2.
PMID: 36198769 Free PMC article. No abstract available.

5 Overexpression of 5-Lipoxygenase Worsens the Phenotype of a Mouse Model of Tauopathy.
Giannopoulos PF, Praticò D.
Mol Neurobiol. 2018 Jul;55(7):5926-5936. doi: 10.1007/s12035-017-0817-7. Epub 2017 Nov 11.
PMID: 29128902 Retracted.

6 Antileukotriene therapy by reducing tau phosphorylation improves synaptic integrity and cognition of P301S transgenic mice.
Giannopoulos PF, Chiu J, Praticò D.
Aging Cell. 2018 Jun;17(3):e12759. doi: 10.1111/acel.12759. Epub 2018 Apr 1.
PMID: 29607621 Free PMC article.

7 Brain 5-lipoxygenase over-expression worsens memory, synaptic integrity, and tau pathology in the P301S mice.
Vagnozzi AN, **Giannopoulos PF**, Praticò D.
Aging Cell. 2018 Feb;17(1):e12695. doi: 10.1111/acel.12695. Epub 2017 Nov 4.
PMID: 29106033 Free PMC article.

8 The 12-15-lipoxygenase is a modulator of Alzheimer's-related tau pathology in vivo.
Giannopoulos PF, Joshi YB, Chu J, Praticò D.
Aging Cell. 2013 Dec;12(6):1082-90. doi: 10.1111/acel.12136. Epub 2013 Aug 19.
PMID: 23862663 Free PMC article.

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VERIFICATION

I, Domenico Pratico, hereby state that I am the Plaintiff in this matter, that the facts set forth in the foregoing Complaint are true and correct to the best of my knowledge, information, and belief, and that this verification is being made subject to 18 PA. C.S. § 4904, relating to unsworn falsification to authorities.

Date: 01/08/2024 By: _____