

To: "Julie Orlando-Castro" <jorlando@sfn.org>, marty@sfn.org  
From: Alexei Koudinov <alexeikoudinov@neurobiologyoflipids.org>  
Subject: Important memo // Re: Abstract for Neuroscience 2005  
Cc: "Melissa Garcia" <melissa@sfn.org>, "Eve Marder" <marder@brandeis.edu>, "Barbara Goldman" <bgoldman@sfn.org>  
Bcc:  
Attached:

16 June 2005

Dear Ms. Orlando,

I am in receipt of the letter by Eve Marder (below) stating that "Program Committee has not decided to change its opinion" regarding the withdrawal of my teaching abstract. Dr.Marder further says that this is because my "second abstract is not a teaching abstract but a scientific abstract".

I can not accept Program Committee Chairperson statement (see my previous appeal request). Sadly, I have several additional concerns that cast doubt on the integrity of the SFN abstract selection process.

First, I doubt that Dr.Marder could come to the Program Committee consensus decision in just few after-business-hours on Friday, June 10, and weekend hours Saturday, June 11, 2005.

Second, I know that the Society for Neuroscience Program Committee Chairperson can mispresent the opinion by the Program Committee members. Recent example is provided by SFN Program committee Chairperson 2003 Dr. Robert Malenka. In his cancelation letter regarding my teaching/history abstract "Research on Alzheimer's disease and amyloid beta protein: a science for sale story?" Dr. Malenka referred to the decision by the Program Committee. However, one of the Program Committee members reported that he did not know about my submission, and did not participate in the Committee decision to withdraw my abstract from the presentation at the Neuroscience 2003.

Third, my past years teaching sessions (written in the similar "teaching new neuroscience concepts" style), including two teaching sessions 2001 (entitled "Neuronal cholesterol pathology is the cause of Alzheimer's disease" and "Brain cholesterol pathology and neuronal function") were not only scheduled for presentation at Neuroscience 2001, but also included in the SFN Annual Meeting 2001 Press Book as invited Lay Language Summaries ([available here](#)).

I request you to kindly **A.** disclose the Neuroscience 2005 Program Committee membership, and **B.** request re-consideration of the decision by Program Committee Chairperson on my Neuroscience 2005 teaching abstract submission #7257.

Sincerely,

Alexei Koudinov, MD, PhD  
neuroscientist and editor  
SFN member

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At 04:09 11/06/05, Eve Marder, Ph.D wrote:

I am sorry, but the Program Committee has not decided to change its opinion that your second abstract is

not a teaching abstract but a scientific abstract, and therefore not suitable for inclusion in the program.  
Eve Marder, Chair, Program Committee

At 06:53 PM 6/10/2005, you wrote:  
10 June 2005

Dear Ms. Orlando,

The letter below is an official Appeal request to reverse my Teaching Session unjustified withdrawal by the Program Committee Chairperson.

I look forward to hear from Program Committee Chairperson about the actions undertaken by the Program Committee to evaluate my request.

Please note that I do not accept the early note by Barbara M. Goldman, Ph.D. (email date Tue, 7 Jun 2005 17:15:36) and Eve Marder (email date Tue, 07 Jun 2005 12:27:13) stating that Teaching sessions "usually describe new experiments for undergraduate labs or software for teaching, etc.," as lacking the complete representation of the SFN teaching session scope. For details on this please read carefully my Appeal Request below.

As a separate request I ask you to kindly disclose the membership of Neuroscience 2005 Program Committee, so I would make my own judgement whether there is a conflict of interest by committee members in the withdrawal of my Teaching Abstract No 7257.

Thank you.

Sincerely,

Alexei Koudinov, MD, PhD  
SFN member

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7 June 2005

Dear Ms. Orlando,

This is an official appeal to re-consider unjustified withdrawal by the Neuroscience 2005 Program Committee my SFN 2005 Teaching Abstract submission No. 7257 entitled "Homeostasis Of Cholesterol And Lipid Peroxidation Are Integrated Components Of Neural Membrane Neuroplasticity Mechanisms".

In your earlier letter of today (below) Eve E. Marder, PhD, Program Committee Chairperson, stated that "the Program Committee has determined that the content of the ["Teaching of Neuroscience"] abstract is scientific and was falsely identified in the submission. As a result, the aforementioned Teaching of Neuroscience abstract has been withdrawn from the Neuroscience 2005 scientific program."

As you should know while preparing and submitting this Teaching abstract (provided below along with my "scientific" abstract No.2258) I noticed and fully agreed with the following SFN terms:

"Learning Objectives:

The Program Committee has determined that your presentation may be eligible to provide

Continuing Medical Education for physicians. We ask you certify that your work will satisfy the needs and meet the objective stated below.

You have chosen History and Teaching of Neuroscience as the theme for this submission. Please confirm that your abstract submission complies with the stated needs and objective for this theme.

**NEEDS:**

The understanding of modern neuroscience knowledge from the molecular and cellular levels to the systems level is of the utmost importance for physicians because of the high prevalence of brain disorders and their great morbidity. Such knowledge is mandatory for a sophisticated understanding of the pathophysiology of brain disorders and the mechanisms of the therapeutic interventions that physicians use to treat these illnesses. The posters, lectures and symposia covered under this theme will discuss how best to impart this information and how over the course of decades of research it has been generated.

**OBJECTIVE:**

The objective is to provide physicians with the opportunity to obtain information about the history of neuroscience and how modern molecular, cellular, and systems concepts were generated, as well as to provide physicians with the opportunity to obtain information about how best to teach these concepts to colleagues, patients, and students.

I have read and understood the needs and objective for my selected theme and verify that my abstract submission complies with these learning needs and objective."

In full accord with the above my Teaching session serves to help physicians and scientists to understand the integration of different mechanisms (specifically cholesterol homeostasis and lipid peroxidation) in regulation synaptic plasticity, damage that cause neurodegenerative disorders.

There is a significant research background for this abstract generated by many years of research by best research groups all over the world. Surely, being limited by the abstract space, we could not provide references to many works by others. However, we included a number of free access links, so, that an abstract reader would easily come to other publications with more detailed info on the subject of this abstract, and further reading suggestions.

In my abstract pre-submission inquiry I even asked Ms. Orlando (see my correspondence with Ms.Orlando of May 9, 2005) whether there is a chance to link my Scientific Abstract (also provided below) to the Teaching Session. As I told Ms. Orlando, "such arrangement would be important, because my science session will be extending important background knowledge to be described in a teaching session."

Quoted above SFN terms for Teaching Session abstract submission specify that "such knowledge is mandatory for a sophisticated understanding of the pathophysiology of brain disorders and the mechanisms of the therapeutic interventions that physicians use to treat these illnesses."

As you could note, the above SFN terms for Teaching sessions, do not fit Program Chairperson determination (presented in Eve Marder email of today, Date Stamp: Tue, 07 Jun 2005 12:27:13 -0400) stating that Teaching sessions "usually describe new experiments for undergraduate labs or software for teaching, etc."

My Teaching session that you attempted to withdraw would further integrate the concepts, teaching physicians and scientists about more complete understanding of the complex subject of the neuroscience of fats and their role in a variety of neurological diseases, a hot Neuroscience topic (that you appreciated by selecting my previous years SFN abstracts to lay language presentations) that I lead as founder and

managing editor of the Neurobiology of Lipids (ISSN 1683-5506).

I therefore do not understand why you disqualified my teaching abstract from the presentation at the Neuroscience 2005 Teaching session.

I request you to reverse the falsified decision of the Neuroscience 2005 Program Committee.

I look forward hearing from you.

Sincerely,

Alexei Koudinov, MD, PhD  
Neuroscientist and Editor  
Russian Academy of Medical Sciences  
Neurobiology of Lipids

-----Teaching Submission No.7257-----

#### **HOMEOSTASIS OF CHOLESTEROL AND LIPID PEROXIDATION ARE INTEGRATED COMPONENTS OF NEURAL MEMBRANE NEUROPLASTICITY MECHANISMS**

A.R. Koudinov\*; T.T. Berezov; N.V. Koudinova

Russian Academy of Med Sci; Neurobiol Lipids, <http://neurobiologyoflipids.org/>, Moscow, Russian Federation

Cholesterol dynamics failure is the primary pathogenic cause in a number of diseases, such as

Alzheimer's (AD), Down syndrome, neuromuscular disorders, and Niemann-Pick's type C (NPC) disease<sup>1-3</sup>

. Other neurodegenerative diseases may have another primary pathological break (e.g. the break of the forebrain dopamine system in Parkinson's disease). In any type neurodegeneration, however, the brain operates its standard set of compensatory mechanisms, causing the overlap of morphologic changes, and explaining the unity of markers across the spectrum of neurodegenerative disorders. We previously showed that fine tuning of neural cholesterol dynamics is essential for synapse function, plasticity,

behavior<sup>1-4</sup>; and suggested that in familial cases of Alzheimer disease, mutations in amyloid precursor protein (APP) and presenilin (PS) genes are translated into the disorder via membrane cholesterol

sensitivity of APP processing by secretases and amyloid beta protein generation<sup>5</sup>. We also reported that impaired long-term potentiation (LTP) in Alzheimer's transgenic mice model depends on human antioxidant enzyme Cu/Zn-SOD, not APP expression; and that lipid peroxidation (LPO, see our scientific

session 2005) modulates synaptic plasticity<sup>6</sup>. Cholesterol homeostasis and LPO are essential for neuronal membrane structure-functional plasticity/integrity, and membrane ability to fine tune activity dependent synaptic strength and associated enzyme/receptor function. This is maintained by the integration of cholesterol synthesis, intracellular and intramembrane redistribution, cellular lipoprotein uptake and efflux of cholesterol; and membrane fluidity modulation by LPO.

#### **References:**

1 <http://neurobiologyoflipids.org/content/3/7>

2 <http://dx.doi.org/10.1371/journal.pmed.0020081>

3 <http://dx.doi.org/10.1096/fj.00-0815fje>

4 <http://dx.doi.org/10.1016/j.jns.2004.11.036>

5 <http://neurobiologyoflipids.org/content/1/8>

6 <http://koudinov.info/reports/sfn2005.html>

-----Scientific Submission No.2258-----

**LIPID PEROXIDATION MODULATES SYNAPTIC PLASTICITY, IMPLICATES COMPENSATORY NATURE OF OXIDATIVE MECHANISMS IN NEURODEGENERATION**

A.R. Koudinov\*; T.T. Beresov

Academy of Med Sci; Neurobiol Lipids, <http://neurobiologyoflipids.org/>; \*Weizmann Inst, Moscow, Russian Federation

Excessive oxidative stress is implicated in a variety of degenerative neurological diseases such as Alzheimer's. Missing is the knowledge about its' role in brain neuroplasticity. We previously reported that hydrogen peroxide (one of the intermediate products of the oxidation cascade) modulates synaptic plasticity<sup>1</sup>. In the present study<sup>2</sup> we investigated the role of lipid peroxidation (LPO), a regulator of cellular membrane physical properties, a target of H<sub>2</sub>O<sub>2</sub> concentration change, and a downstream oxidation product. We used extracellular two channel recording in CA1 area of the ex-vivo rat hippocampal slices using one recording and two stimulating electrodes. LPO induction with a promoter 2,2'-azobis (2-amidinopropane) dihydrochloride (AAPH) slowly decreased both *i*) potentiated EPSP slope values of the pathway tetanized previously (to near the baseline recording), and *ii*) baseline EPSP slopes of the non-potentiated pathway (to near half of the baseline values). The effect was not abolished by the AAPH washout. Moreover, as in case of high dose H<sub>2</sub>O<sub>2</sub>, the naive pathway lacked the ability to express tetanus induced (t) long-term potentiation (LTP). We also performed experiments with classical LPO blocker, vitamin E. This compound did not affect normal maintenance of the tLTP on the channel tetanized previously. However, vitamin E produced slow onset potentiation of the naive non-potentiated pathway (about 50 % above the baseline), and abolished further induction of the tLTP on this channel. Taken together, these experiments suggest that physiologic regulation of LPO and free radical mechanisms are of great importance to fine tune activity dependent synaptic strength/function. This is additionally supported by our observation of the increase of LPO in slices of rat hippocampus (identical to those used for the LTP studies) stimulated with high K<sup>+</sup>.

1 <http://neurobiologyoflipids.org/content/3/5>

2 <http://koudinov.info/reports/sfn2005.html>

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At 18:29 07/06/05, you wrote:

**MEMORANDUM**

TO: Dr. Alexei Koudinov

FROM: Eve E. Marder, PhD

Chairperson, Program Committee

RE: Abstract for Neuroscience 2005

DATE: June 7, 2005

It has come to the attention of the Program Committee that your abstract, HOMEOSTASIS OF CHOLESTEROL AND LIPID PEROXIDATION ARE INTEGRATED COMPONENTS OF NEURAL MEMBRANE NEUROPLASTICITY MECHANISMS, is in violation of the Rules for Submission and Presentation of Abstracts. As stated in the Rules for Submission, which are currently available on the Neuroscience 2005 homepage,

**“2. A member of SfN may sign only one scientific abstract.** A student member may sign a scientific abstract only as first (presenting) author. In addition, a member may also sign one Teaching of Neuroscience Abstract and/or History of Neuroscience abstract according to the rules in the Call for Abstracts. Affiliate members may not sign abstracts. In cases where a member has signed two abstracts or is the first (presenting) author on two abstracts, one of the abstracts must be withdrawn by the member.”

It appears that you had two submissions, one classified as scientific and the other as teaching. Although the latter abstract was submitted under the theme “Teaching of Neuroscience,” the Program Committee has determined that the content of the abstract is scientific and was falsely identified in the submission. As a result, the aforementioned Teaching of Neuroscience abstract has been withdrawn from the Neuroscience 2005 scientific program.

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