Authorship

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Editor Physiological Reviews
Past Editor, AJP Cell Physiology
Several Editorial boards:

Why do we care about authorship?

- Communication of scientific results
- Allocation of credit
- Recognition by peers - respect

- Finding a job
- Funding
- Promotion
- Fame, fortune, Nobel prizes etc


KEEP
CALM
AND
PUBLISH
OR PERISH

ICMJE Guidelines for Authorship

(International Committee of Medical Journal Editors)

- Substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data AND
- Drafting the article or revising it critically for important intellectual content AND
- Final approval of the version to be published AND
- Agreement to be accountable for the work

What does not count?

- Providing technical advice, reagents, patient data, funding
- Providing technical personnel, students to perform studies
- Routine data collection
- General supervision of group

Q: How is order of authors determined?

- **All authors:** fulfill ICMJE requirements
- **First author:** performs “bulk” of the work, may write first draft of manuscript – often obvious
- **Last author:** takes responsibility for accuracy of the entire publication, usually more senior
- **Middle authors:** contributions do not rise to the level of first or senior authors
- **Corresponding author:** communicates with editor/reviewer – can be any of the authors

Q: A colleague and I meet weekly to discuss project, ideas and troubleshooting. He did not contribute through benchwork. I would like to add him as co-author for helpful input.

Is this OK?

“Promiscuous” Authorship

- Awarding authorship to someone who has not contributed to manuscript in an intellectually significant manner
- Types:
  - Coercive: seniority or supervisory status to claim authorship
  - “Honorary” or guest: authorship awarded out of respect /friendship/to increase status of work/improve CV for promotion

“Guest” Authorship turns bad

Stem cell paper in Science, 2005, retracted

Patient-Specific Embryonic Stem Cells Derived from Human SCNT Blastocysts
Woo Suk Hwang, Sung Il Roh, Byeong Chun Lee, Sung Keun Kang, Dae Kee Kwon, Sue Kim, Sun Jong Kim, Sun Woo Park, Hee Sun Kwon, Chang Kyu Lee, Jung Bok Lee, Jin Mee Kim, Curie Ahn, Sun Ha Paek, Sang Sik Chang, Jung Jin Koo, Hyun Soo Yoon, Jung Hye Hwang, Youn Young Hwang, Ye Soo Park, Sun Kyung Oh, Hee Sun Kim, Jong Hyuk Park, Shin Yong Moon, and Gerald Schatten
Science 17 June 2005 308: 1777-1783; published online 19 May 2005 [DOI: 10.1126/science.1112286] (in Reports)
Abstract » Full Text » PDF » Supporting Online Material » Correction » **Retraction »**

Woo Suk Hwang and Gerald Schatten (U. Pitt)

Q: A non-collaborating researcher suggests an additional test during an informal conversation. It is incorporated into the final manuscript. She was not otherwise involved.

Should she be a co-author?

Q: If someone contributes essential equipment or reagents, but was not involved in design, collecting data, or analysis, does this merit a middle authorship?

Or “mere” mention in the acknowledgements section?

Q: What about co-first authors?

Q: How is senior author selected?

Q: What if two postdocs are working on the same project?

Q: What about technicians?

Q: What if postdoc leaves before work is finished?

Q: What about multi-author or multi-center clinical trials?

Complicated - requires extensive pre-planning

Ann Intern Med. 2009 Sep 15;151(6):414-20.

Method for establishing authorship in a multicenter clinical trial.

Whellan DJ[1], Ellis SJ, Kraus WE, Hawthorne K, Piña IL, Keteyian SJ, Kitzman DW, Cooper L, Lee K, O'Connor CM.

Author information

Acta Anaesthesiol Scand. 2011 Oct;55(9):1037-43. doi: 10.1111/j.1399-6576.2011.02477.x. Epub 2011 Jun 20.

Determining authorship in multicenter trials: a systematic review.

Dulhunty JM[1], Boots RJ, Paratz JD, Lipman J.

Author information

Q: Who resolves authorship disputes?

- Authors whenever possible
- Institution
- Ombuds office

- NOT THE JOURNAL

Disputes: Example 1

- Post-doc 2 replaces post doc 1 in lab
- PD2 thinks s/he has done more work
- PI will think about the situation
- PD2 is impatient – demands 1st place
- PD2 threatens to remove data from lab if not 1st author

- What should the PI do?
- What should the PD have done?
- Who owns the data?

Disputes: Example 2

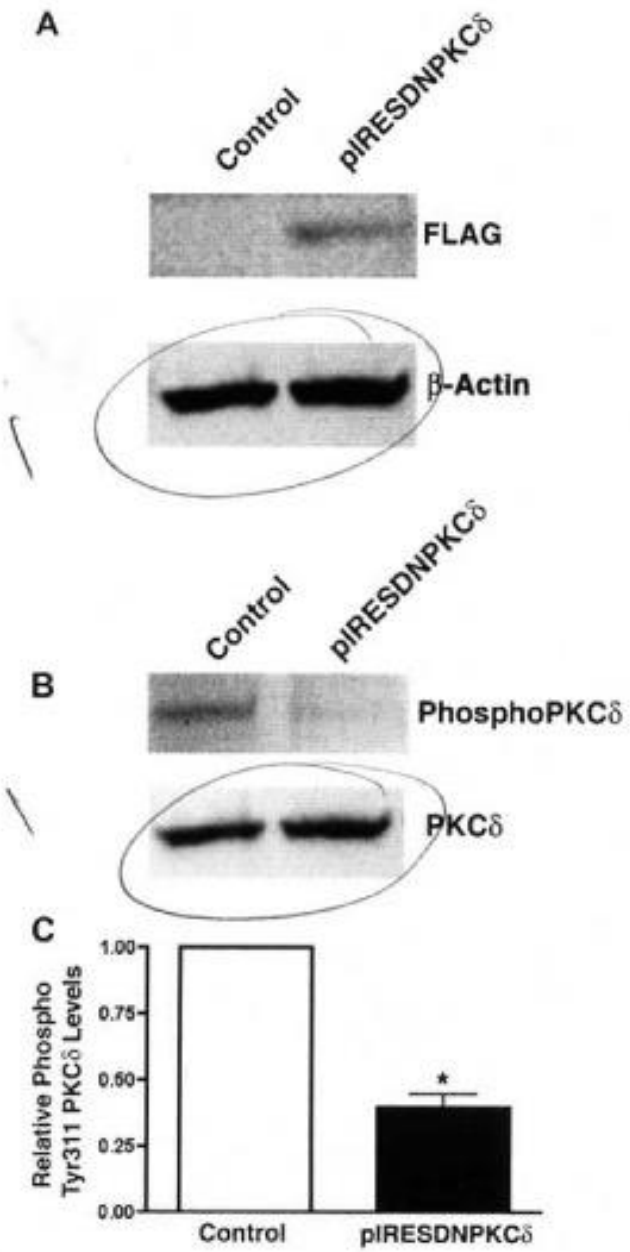
- Post-doc performs studies and presents data at scientific meeting
- 8 months later, manuscript is prepared for submission
- PDs name is not among the 6 authors
- PD complains to PI who says that s/he performed sloppy work and does not deserve to be an author. None of the original figures were used in the manuscript but they were repeated by another PD in the same lab.

- How did this situation happen?
- Is it fair and what should the PD do?

Author responsibility example

- Paper with 3 authors is published
- After publication, irregularities are seen by a reader, who contacts the journal
- Journal ethics committee finds major issue
- Gels have been duplicated
- First author admits guilt
- All three authors are banned for 3 years from the journal and institution is notified

FIGURE 3



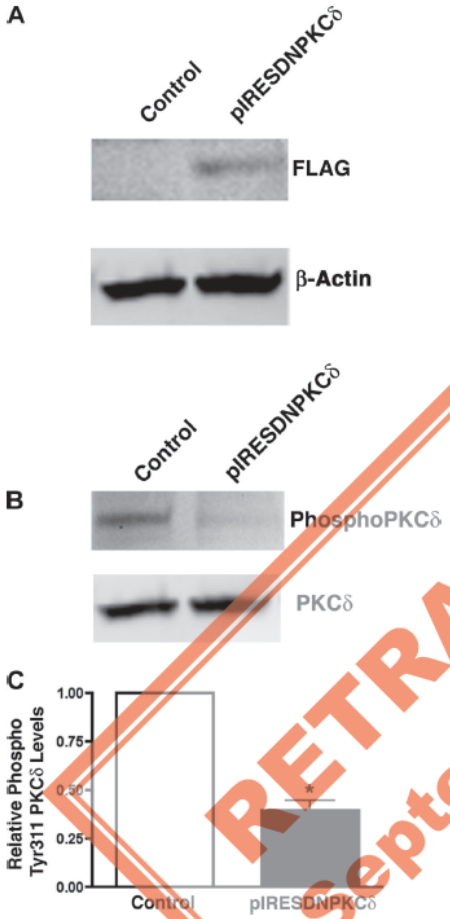


Fig. 3. Generation of a PKCδ dominant negative mutant expression plasmid. FPAECs were transfected or not with the dominant negative PKCδ mutant plasmid, pIRES-DNPKCδ. After 24 h, whole cell lysates were subjected to Western blot analysis for the FLAG epitope (*A*) as well as total and phospho-Tyr311 PKCδ (*B* and *C*). Representative images are shown for each. Although total PKCδ (*B* and *C*) is unchanged by pIRES-DNPKCδ transfection phospho-Tyr311 PKCδ (*C*) levels were significantly decreased. Data are presented as means ± SE, $n = 3$. *$P < 0.05$ vs. control cells.

though PKCδ levels were unchanged by pIRES-DNPKCδ (Fig. 3, *B* and *C*), there was a significant decrease in the activating (Tyr311) phosphorylation of PKCδ (Fig. 3, *B* and *C*), confirming the dominant negative effect.

Prolonged effect of PKCδ inhibition on eNOS expression. FPAECs were exposed to rottlerin (10 μM) for 24 h and then analyzed for eNOS protein expression by Western blot analysis. eNOS protein expression was also compared in cells overexpressing a dominant negative PKCδ mutant, pIRES-DNPKCδ. Our data indicated that PKCδ inhibition significantly reduced eNOS protein levels (Fig. 4, $P < 0.05$ vs. control). To determine if this effect was at the level of transcription, we transfected FPAECs with a 1.6-kb eNOS promoter construct linked to a luciferase reporter gene (51) in the presence or absence of the dominant negative PKCδ mutant and then incubated with or without rottlerin. We then analyzed eNOS promoter activity after a further 24 h. Our data indicate that PKCδ inhibition with either rottlerin or dominant negative PKCδ mutant overexpression significantly reduces eNOS promoter activity (Fig. 5, $P < 0.05$ vs. control).

Effect of Akt inhibition on eNOS expression and activity. To further elucidate the potential role of the PKCδ/Akt axis in regulating eNOS expression, we utilized an adenoviral construct to overexpress a dominant negative mutant of Akt in FPAECs. Initial Western blot analyses confirmed the overexpression of the dominant negative Akt mutant. We found that Akt expression was approximately twofold higher in dominant negative Akt mutant-transduced cells compared with GFP-transduced cells (Fig. 6, *A* and *B*, $P < 0.05$ vs. GFP control). We next determined if the dominant negative Akt mutant had effects on eNOS phosphorylation at Ser1177 or total eNOS expression. Our data indicate that the overexpression of the dominant negative Akt mutant significantly decreased both total eNOS protein levels (Fig. 6, *C* and *D*, $P < 0.05$ vs. control) and eNOS phosphorylation at Ser1177 (Fig. 6, *C* and *D*, $P < 0.05$ vs. control). These changes were associated with decreased NO generation (Fig. 6*E*, $P < 0.05$ vs. control). Furthermore, we found that overexpression of the dominant negative Akt mutant significantly decreased eNOS promoter activity (Fig. 7, $P < 0.05$ vs. control).

Effect of endogenous NO on eNOS expression. Our data indicated that the inhibition of PKCδ/Akt signaling leads to decreased NO generation and a subsequent decrease in NOS expression. Thus, we next determined whether the decrease in eNOS expression causally related to the reduced NO generation. Initially, we confirmed that the eNOS inhibition using ETU decreased NO generation. We found that NO_x levels were significantly decreased after 24 h of ETU (100 μM) treatment (Fig. 8*A*, $P < 0.05$ vs. control). Furthermore, the reduction of NO generation caused significant decreases in both eNOS promoter activity (Fig. 8*B*, $P < 0.05$ vs. control) and eNOS protein levels (Fig. 8, *C* and *D*, $P < 0.05$ vs. control). To verify that NO signaling is involved in regulating eNOS promoter activity, cells were treated with both ETU and the NO donor, sodium nitroprusside (SNP). Our data indicate that the addition of SNP prevents the ETU-mediated reduction in eNOS promoter activity (Fig. 9*A*) and eNOS protein levels (Fig. 9, *B* and *C*).

DISCUSSION

In the fetus, pulmonary vascular resistance is high and pulmonary blood flow is low. With the initiation of ventilation and oxygenation at birth, pulmonary vascular resistance decreases, and pulmonary blood flow increases. Increasing evidence suggests that changes in pulmonary vascular tone are mediated by NO, possibly in response to increased shear stress on the pulmonary vascular endothelium (20, 28, 30). eNOS plays an important role in providing the NO necessary to allow these birth-related changes. In a number of clinical conditions, the pulmonary circulation fails to undergo the normal transition to postnatal life, resulting in persistent pulmonary hypertension of the newborn (PPHN) (27). Thus, a better understanding of

Author responsibility continued

- 3rd author (the PI) protests and appeals to journal ethics committee
- It was not my fault!
- Ethics committee considers appeal
- Sentence upheld

- Was this the correct decision?
- What are your responsibilities?

Take home points

- DISCUSS authorship early and often – no HARD rules
- All authors should meet CRITERIA
- AVOID duplicate publication , plagiarism, other misconduct


RUSSELL P
DINSMORE
1907-1984
PUBLISHED,
BUT PERISHED
ANYWAY

lipoic acid injection reduces

sensitivity to noxious thermal and mechanical stimuli in 296

mice (120). Collectively, these observations agree

with the results that no low-threshold Ca2+ current remains in small DRG neurons of CaV3.2/ mice (31)—cells known to be peripheral nociceptors 1

(218). Thus, these results provide explicit evidence for the role of

CaV3.2 T-type channels in pain perception and propose that CaV3.2 may be a good candidate to target for treatment of pain at the 39

peripheral level. Inflammatory and visceral pain CaV3.2/ mice also show a decreased pain response to visceral pain, an observation that agrees with a previous report that no low-threshold

Ca2+ currents remained in small dorsal root ganglion (DRG) neurons in these 242

mice (31). This result proposes that small DRG neurons conduct substantial role in carrying visceral pain signals. Recently, it was reported that

T-type Ca2+ channels in primary sensory neurons in colonic and 73

DRG cells are involved in mediating colonic pain transmission (136, 137). In this context, it is notable that CaV3.2

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Hee-Sup Shin. "Genetic Studies on the Role of T-Type Ca2+ Channels in Sleep and Absence Epilepsy", CNS & Neurological Disorders - Drug Targets (Formerly Current Drug Targets - CNS & Neurological Disorders), 12/01/2006

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sleep, pain, and even fear disorders. In addition, we expect

that the study of T-type channels in the 268

neurobiology of cognition will continue to evolve. Figure Legends Figure 1.

Intrinsic firing properties of TC neurons located at the ventrobasal complex. (A) 15
Burst firing patterns elicited by 100-ms pulses of negative step-current inputs at 70 mV. Holding membrane potentials were maintained by DC current input. The amount of current injected is indicated below each trace; pA, picoampere. Scale bars: 100 ms (horizontal) and 50 mV (vertical). (B) Burst firing patterns elicited by positive step-current inputs at -80 mV. Note the increasing firing frequency of lower-frequency spikes in wild-type TC neurons

with positive input currents greater than 700 pA. Only

high-frequency spikes are missing in CaV3.1/ TC neurons. (C) Tonic firing patterns 5
elicited by positive step-current inputs at -60 mV. Low-frequency spikes are elicited equally in wild-type and CaV3 .1/ TC neurons. (D) The relationship between the number of spikes and the amount of current injected. The number of spikes during 100-ms positive step-current inputs when membrane potentials are held at -60 mV (left) or -80 mV (right).

Modified from Reference (106). Figure 2. Baclofen-induced SWDs in

wild-type and CaV3.1/ mice. Top: Representative EEG traces 150

show that injection of baclofen induces abundant SWD

TC neurons were often shifted from tonic to low-threshold burst firing (J), whereas wild-type TC neurons never showed such a transition in firing mode (I). The bottom panel displays the applied current steps. Injection of prepulses, which slightly hyperpolarized the membrane potentials, elicited low-threshold burst firing in PLC4/ TC neurons (L), but not in wild-type TC neurons (K). (M) Spike numbers in a burst induced by various prepulses that hyperpolarized the membrane potentials to between -73 and -63 mV in wild-type (closed circle) and PLC4/ (open circle) TC neurons. 2

Modified from References (36, 37). Figure

5. Deletion of thalamic PLCβ4 leads to the genesis of absence seizures. (A) Lentiviral vectors containing control shRNA or an shPLCβ4 construct were injected bilaterally into wild-type mice and EEGs were recorded from frontal and parietal lobes. Lower panel: mice injected with LV- shPLCβ4 showed sporadic SWDs; upper panel: mice injected with pLKO-control never showed such a high-amplitude paroxysmal EEG pattern. (B) Seven of 12 mice injected with LV- shPLCβ4 showed spontaneous SWDs. The number of SWDs varied from 3 to 17 per hour. (C) The total duration of SWDs per minute induced by 20 mg/kg RS(+/-)-baclofen was greater in mice injected with LV- shPLCβ4 than in mice injected with pLKO-control. 2

Modified from Reference (37). Figure 6.

EEG power density at delta waves was decreased in CaV3.1-/- mice compared with wild-type mice during NREM sleep. Sample traces show EEG and EMG signals recorded from REM (A) and NREM (B) sleep states in 12

wild-type (CaV3.1+/+) mice (upper) and CaV3.1/ mice (lower).