



Cardiac Electrophysiology Society Poster Forum

Guidelines for Submission of Abstracts

Abstract Submission

Abstract submission opens: July 15, 2009

Abstract submission deadline: September 1, 2009 - 5 p.m. EST.

There is a non-refundable processing fee of US \$30 for each abstract submission

Rules for Preparation and Submission of Abstracts

- Presenting author must be a **member in good standing** of the Cardiac Electrophysiology Society. Society Dues: Fellows: \$20; Regular: \$50
- Abstracts accepted for presentation at the AHA meeting are **not eligible** for submission or presentation at this CES Poster Forum.
- Only electronic submissions will be accepted.
- Due to the anticipated volume of abstract submissions, there is a limit of **only (1) submission per person**.
- Expenses associated with the submission and presentation of an abstract are the responsibility of the presenter.
- Abstracts should follow the format of the **SAMPLE ABSTRACT** shown below.
- Abstracts should be submitted in MS WORD format and uploaded at www.CardiacEPS.org. **File name should be [B for Basic or C for Clinical-FirstName_LastName] (e.g., B-Lai_Xie.doc). Payment can be made using credit card at www.cardiaceps.org**

Author Name(s)

- The First Author will be presumed to be the Presenting Author.

Type of Abstract

To ensure that your abstract receives proper scientific consideration, be sure indicate appropriate category – **Basic Science or Clinical**

- All abstracts must be submitted in English.

Abstract Title

- Do not use abbreviations in the title.

- The title is limited to 250 characters. Spaces do not count as characters.

Abstract Text

- The abstract should contain the following headings in bold: **Background, Methods, Results and Conclusions.**
- **Text (excluding, title, authors and institutions) is limited to 1950 characters . Deduct 250 characters for addition of a table and 500 characters for submission of a graphic. Spaces do not count as characters. Graphics must be legible when reduced to an image of 2" height and 3" length. All graphics must be black & white images.**
- Abstracts should not describe research in which the chemical identity or source of the reagent is proprietary or cannot be revealed.
- Use generic drug names.
- Do not include references.

Abstract Acceptance

- Notification of acceptance of abstract will be e-mailed **no later than September 15, 2009.**

Presentation

- **Twenty Basic Science and Twenty Clinical abstracts** will be selected for presentation at the CES meeting on November 14, 2009.
- Posters accepted for presentation should be mounted for display no later than **10:30 AM on Saturday, November 14.**
- Viewing and judging of posters will take place between **11:00 AM and 1:30 PM.**

Awards

- **Five Finalists** to be selected in each category (Basic Science and Clinical) based on top scores will receive one year free membership to CES.
- These five posters will be judged at the Poster Session preceding the CES meeting and **two winners will be selected in each category.**
- The **four winners** will be announced and be presented with a \$500 check and a certificate at the end of the CES meeting.

Questions

Questions should be submitted to abstracts@cardiaceps.org

- ***It is the submitter's responsibility to comply with prescribed character limits. Abstracts that fail to comply will be rejected.***

SAMPLE ABSTRACT

Basic Science Abstract

Hydrogen Peroxide-Induced Afterdepolarizations are Dependent on Calmodulin Kinase II Signaling

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Background: In the heart, hydrogen peroxide (H₂O₂) has been shown to cause early afterdepolarizations (EADs) and triggered activity by impairing Na current (I_{Na}) inactivation. Since H₂O₂ has been recently shown to activate Ca²⁺/calmodulin kinase II (CaMKII), and since CaMKII activation has also been reported to impair I_{Na} inactivation and predispose to EADs, we hypothesized that CaMKII activation by H₂O₂ may be an important factor in the genesis of EADs induced by oxidative stress.

Methods and Results: Patch-clamped Fluo-4 AM-loaded rabbit ventricular myocytes were exposed to H₂O₂ (0.1–1mM), which induced spontaneous EADs after 5–15 min. Both the I_{Na} blocker tetrodotoxin (TTX, 10 μM) and the I_{Ca,L} blocker nifedipine shortened AP duration (APD) and suppressed EADs. H₂O₂ increased both peak and steady-state I_{Ca,L} under square-pulse voltage clamp, and enhanced I_{Ca,L} to a greater extent during the AP plateau than during the AP upstroke under AP clamp conditions. In addition, by prolonging the AP plateau and increasing Ca influx via maintained I_{Ca,L}, H₂O₂-induced EADs frequently caused DADs delayed afterdepolarizations (DADs) due to spontaneous SR Ca release waves after repolarization. KN-93(1 μM), a CaMKII inhibitor, prevented H₂O₂ -induced EADs (n=4), whereas the inactive analogue KN-92 did not (n=5).

Conclusion: These findings indicate that H₂O₂-induced EADs depend on both impaired I_{Na} inactivation to reduce repolarization reserve and enhanced I_{Ca,L} to reverse repolarization. Intact CaMKII signaling is necessary for EAD generation in this setting, presumably via its actions on I_{Na} and I_{Ca,L}, although direct redox effects on other ion channels/transporters may also be important. Our observations support a link between increased oxidative stress, CaMKII activation and afterdepolarizations as triggers of lethal ventricular arrhythmias in diseased heart.

Character Limit: 1950

(Note : Text (excluding, title, authors and institutions) is limited to 1950 characters .)

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