ISHR-NAS 2024 Abstract Submission Guidelines

Conference registration is mandatory for consideration of the abstract by the Scientific committee for oral and/or poster presentation. Abstracts must be original and must not have been published or presented at any international meeting. By submitting an abstract, the author transfers copyright ownership to the organizing committee for publications; the Organizing Committee reserves the right to reproduce the abstract in print and/or electronic media.

Deadline for submission for consideration for oral presentation: June 19th, 2024.
Deadline for submission for consideration for poster presentation: June 30, 2024.

Abstract Guidelines:

- All abstracts are to be submitted in English.
- The abstract should be as informative and succinct as possible (250 word limit).
- The title should be short and concise (20 word limit).
- List all author and co-author names and affiliations.
- Use numerals to indicate numbers, except at the beginning of a sentence.
- You may use standard abbreviations and acronyms.
- Define unusual abbreviations or acronyms in parentheses after the first appearance of the word or phrase.
- Sample abstract included below.

Abstract Submission:

- All abstracts must be submitted via the Abstract Submission portal.
- All submissions will be acknowledged by email and the presenting author indicated on the submission form will be the contact person for all correspondence regarding the presentation.
- The Scientific Committee reserves the right to allocate a session time or change the presentation type.

Lightning Talks:

- You will receive an assigned number from NAS2024 for your poster. All odd numbered posters will present their posters on Wednesday, August 21st, 2024. All even numbered posters will present their posters on Thursday, August 22nd, 2024.

- All poster presenters are invited to also do a **2-min long Lightning Talks** (2 ppt maximum) on the same day their poster is presented.

- Lightning Talks will be scored by the co-chairs/judges; one platinum awardee will receive a $500 Amazon gift card; two gold awardees will each receive a $100 Amazon gift card; six silver awardees will each receive a $50 Amazon gift card.

- Presenters can be in any of their academic rankings; e.g., Professor, graduate student, junior faculty members, all are treated as equal.

- **Posters can be up to 30"x42". No bigger please.**

Example:

Reactive oxygen species (ROS) in cardioprotection: which ROS does the signaling?

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ROS are the second messengers of cardioprotective signaling; however, the ROS species responsible for this effect under physiological conditions is not known. Cellular ROS transformations proceed from superoxide (O_2^-) to hydrogen peroxide (H_2O_2) and then to hydroxyl radical (•OH), each of which has been investigated as the signaling ROS. H_2O_2 is an appealing candidate for a second messenger because it is relatively stable and can readily diffuse to target kinases. Nevertheless, there is no direct evidence to support or exclude H_2O_2 as the physiological ROS messenger. We examined the effects of exogenous H_2O_2 and endogenous (mitochondrial) ROS produced by the electron transport chain on three ROS-dependent processes: PKCε-dependent mitochondrial ATP-sensitive potassium channel (mitoK_{ATP}) opening, PKCε-dependent inhibition of the mitochondrial permeability transition (MPT), and cardioprotection of the *ex vivo* heart. Dimethylsulfoxide (DMSO) and dimethylformamide (DMF) scavenge •OH but neither influenced PKCε activation by exogenous H_2O_2. This indicates that they neither scavenge H_2O_2 nor are they thiol reductants. However, both reagents blocked PKCε activation by endogenous (mitochondrial) ROS, and DMF blocked cardioprotection by diazoxide and ischemic postconditioning. The putative •OH scavenger N-2-mercaptopropionylglycine (MPG) blocked PKCε activation by exogenous H_2O_2, indicating that MPG is acting here as a thiol reductant rather than as a ROS scavenger. These results appear to exclude H_2O_2 as the signaling ROS and implicate a downstream oxidation product of •OH. Our evidence points to hydroperoxy fatty acids as the signaling ROS in cardioprotection.

Keywords: mitochondria; cardioprotection; hydroxyl radical; K-ATP channels; cardiac ischemia; hydroperoxy fatty acids; ROS signaling