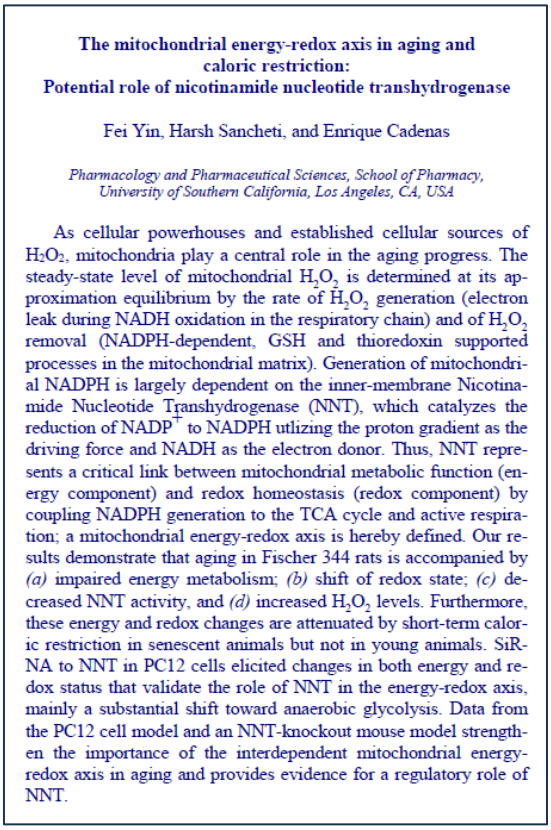
Rules and Guidelines for Abstract Submission

- The **Abstract File Name** must contain your first and last names as in the following example:
  If you are *John Doe*, your Abstract File Name should be: "***John_Doe_Abstract.doc***"

- Your abstract will be reviewed by the Advisory Board and you will be informed about the acceptance of it about 1 week after submission. Final presentation details will be communicated by April 25th, 2014.

- Prepare your abstract in a **Microsoft Word** document (*.doc* or *.docx*) and follow these guidelines:
  - Text should be single spaced, not justified and in Times New Roman.
  - The abstract should include title, authors, affiliation and text, in that order.
  - **Title:** bold, lower case 12-point font. Start only first word with upper case and avoid the use of a dot at the end of the abstract title (leave a free line between title and authors).
  - **Authors:** [First name] [Middle] [Last name] (*Cesar G. Fraga*, *Tamara Zaobornyj*, etc.), 12-point font (leave a free line between authors and affiliation).
  - **Affiliation:** italic, upper and lower case 10-point font.
  - **Body text:** maximum 250 words; 12-point font (leave a free line between author´s affiliation and text).
  - Insert Greek letters as Symbol fonts.

Abstract Example:



The mitochondrial energy-redox axis in aging and caloric restriction: Potential role of nicotinamide nucleotide transhydrogenase

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As cellular powerhouses and established cellular sources of H_2O_2, mitochondria play a central role in the aging progress. The steady-state level of mitochondrial H_2O_2 is determined at its approximation equilibrium by the rate of H_2O_2 generation (electron leak during NADH oxidation in the respiratory chain) and of H_2O_2 removal (NADPH-dependent, GSH and thioredoxin supported processes in the mitochondrial matrix). Generation of mitochondrial NADPH is largely dependent on the inner-membrane Nicotinamide Nucleotide Transhydrogenase (NNT), which catalyzes the reduction of $NADP^+$ to NADPH utlizing the proton gradient as the driving force and NADH as the electron donor. Thus, NNT represents a critical link between mitochondrial metabolic function (energy component) and redox homeostasis (redox component) by coupling NADPH generation to the TCA cycle and active respiration; a mitochondrial energy-redox axis is hereby defined. Our results demonstrate that aging in Fischer 344 rats is accompanied by *(a)* impaired energy metabolism; *(b)* shift of redox state; *(c)* decreased NNT activity, and *(d)* increased H_2O_2 levels. Furthermore, these energy and redox changes are attenuated by short-term caloric restriction in senescent animals but not in young animals. SiRNA to NNT in PC12 cells elicited changes in both energy and redox status that validate the role of NNT in the energy-redox axis, mainly a substantial shift toward anaerobic glycolysis. Data from the PC12 cell model and an NNT-knockout mouse model strengthen the importance of the interdependent mitochondrial energy-redox axis in aging and provides evidence for a regulatory role of NNT.