How to write an abstract

The ***abstract title, authors, affiliations and abstract body must not exceed 3000 characters*** (excluding spaces) and must contain at least 100 characters. If character guidelines are not followed, you will not be allowed to proceed.

Title – Capitalized first letters, no period

- Make the title something interesting that tells the main point of your research. Don't be wordy.

List each Author in your word version of the abstract.
First name, middle initial and last name. My name goes last. Joseph J Provost
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There is a clear algorithm to writing an abstract – write down your answer to the following questions:

1) Before writing – ask yourself what is the main point of your research? NOT THE ASSAY? Remember the assay is just how you are finding an answer to your cool question. So what IS your cool question? Write it down.
2) Then – think about what does your data tell you the answer to your cool question is? Or as abstracts are done so early in the school year, what do you THINK it will tell you. Then write the answer down.
3) Then – what is the WDIC moment based on #1 and #2. Write that down.
4) What are the three main things someone wants to know to understand your science? Write that down.

- Use #1 and #3 to create your title.
- Use #4 to start the first three or so sentences.
- Use actual data or planned data to write the middle.
- Close with #3.

Specific instructions:
The purpose of the abstract is to provide the reader with an overview of the essential aspects of the paper or presentation. Readers often decide whether to listen to a talk or to read an article on the basis of the abstract. Thus, being able to write a good abstract is an important skill to have. Writing an abstract is often a challenge because one is usually limited to just a few hundred words. These constraints can be helpful though since they force the writer to identify the fundamental aspects of the research or presentation. Basically, an abstract provides a very brief overview of the four major parts of a scientific paper… introduction, methods results and discussions. The following guidelines are intended for abstracts with 150 – 250 word restrictions. This is a typical length restriction for most papers presented at scientific meetings.

The first one or two sentences of an abstract should provide a context for the specific study being presented.

A good approach is to briefly describe the larger scientific issues or questions that are motivating scientists to conduct his or her research.

Following a one or two sentence introduction, one should clearly and explicitly state the purpose of the study. This can be done in a variety of ways, e.g., " The purpose of the study was…" , "This study attempted to answer the following questions to test the hypothesis", "This study focused on the…" "To better understand the mechanism of…" or "The aim of this study was to…".

Following the statement of purpose, the general methodical approach should be described (if possible in one or two sentences). In other word, what major techniques did you use to find the results. Do not explain the how but rather the what.

Major results should also be summarized in two or four sentences.

The abstract should conclude with at final sentence or two in which the significance or ramifications of the findings are briefly stated. These final sentences should connect the findings with one or more of the larger ideas stated in the opening two sentences.

A good abstract is not hard to write once you know the key elements to include. In many cases, it will be possible to lift entire sentences out of your paper or poster to include in you abstract. The hardest part is in the planning. The more detailed a plan or outline the easier your abstract and poster/paper will be to write.

Model of a Quinary Structure between Krebs TCA Cycle Enzymes: A Model for the Metabolon

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ABSTRACT: The enzymes which are responsible for catalyzing sequential reactions in several metabolic pathways have been proposed to be highly organized in supramolecular complexes termed metabolons. However, the in situ existence of these weak complexes is difficult to demonstrate because many of them are dissociated during isolation due to dilution effects. Consequently, the metabolon concept is subject to controversy. A model system consisting of genetically prepared bienzymatic fusion proteins has been used to immobilize sequential metabolic enzymes in close proximity and to demonstrate possible kinetic advantages of metabolons. These experiments use the sequential Krebs TCA cycle enzymes from yeast mitochondrial malate dehydrogenase (MDH), citrate synthase (CS), and aconitase (ACO). Using the porcine high-definition structures of these three enzymes, we have performed computer-modeling studies in order to understand how the molecules may interact. Among the thousands of docking orientations we have tried, one was found to respond to the structural and experimental constraints from the results obtained with the yeast fusion proteins. Interestingly, this quinary structure model shows substantial interacting surface areas with spatial and electrostatic complementarities which make the complex thermodynamically stable. This structure also contains an unbroken electrostatically favorable channel connecting the active sites of ACO and CS, as well as the one previously reported between CS and MDH active sites. Charged amino acids which could be involved in interactions stabilizing the complex have been identified. This model will be used as the basis for further experimental work on the structure of the Krebs TCA cycle metabolon.

Calcineurin homologous protein isoform 2 supports tumor survival via the sodium hydrogen exchanger isoform 1 in non-small cell lung cancer

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Abstract Maintaining intracellular pH is crucial for preserving healthy cellular behavior and, when dysregulated, results in increased proliferation, migration, and invasion. The Na^+/H^+ exchanger isoform 1 is a highly regulated transmembrane antiporter that maintains pH homeostasis by exporting protons in response to intra- and extracellular signals. Activation of Na^+/H^+ exchanger isoform 1 is exquisitely regulated by the extracellular environment and protein cofactors, including calcineurin B homologous proteins 1 and 2. While Na^+/H^+ exchanger isoform 1 and calcineurin B homologous protein 1 are ubiquitously expressed, calcineurin B homologous protein 2 shows tissue-specific expression and upregulation in a variety of cancer cells. In addition, calcineurin B homologous protein 2 expression is modulated by tumorigenic extracellular conditions like low nutrients. To understand the role of calcineurin B homologous protein 2 in tumorigenesis and survival in lung cancer, we surveyed existing databases and formed a comprehensive report of Na^+/H^+ exchanger isoform 1, calcineurin B homologous protein 1, and calcineurin B homologous protein 2 expression in diseased and non-diseased tissues. We show that calcineurin B homologous protein 2 is upregulated during oncogenesis in many adeno and squamous carcinomas. To understand the functional role of calcineurin B homologous protein 2 upregulation, we evaluated the effect of Na^+/H^+ exchanger isoform 1 and calcineurin B homologous protein 2 depletion on cellular function during cancer progression in situ. Here, we show that calcineurin B homologous protein 2 functions through Na^+/H^+ exchanger isoform 1 to effect cell proliferation, cell migration, steady-state pH_i, and anchorage-independent tumor growth. Finally, we present evidence that calcineurin B homologous protein 2 depletion in vivo has potential to reduce tumor burden in a xenograft model. Together, these data support the tumor-promoting potential of aberrant calcineurin B homologous protein 2 expression and position calcineurin B homologous protein 2 as a potential therapeutic target for the treatment of non-small cell lung cancer.