Writing a scientific abstract

The abstract is the shortest yet most important part of a scientific report. This is your research 'business card' that presents your research and its significance. Every reader will make a decision after reading your abstract: proceed to read the remainder of the report or move on to the next report. A well-written abstract will prompt the reader to learn more about your research.

The abstract should be meaningful by itself containing all the necessary information for understanding the research and its contribution. An abstract consists of the following sections:

Objective and Rationale: Statement of the research topic and the addressed question

Background: Succinct summary of research topic

Methods: Brief discussion of the experimental methods

Results: Observations derived from experiments

Conclusions: Main message derived from research findings and its significance

Brainstorm about the information you want to include using the questions listed below. Use this information to begin writing your abstract. Improve your abstract by getting feedback from your colleagues. Make sure your abstract follows the format guidelines requested by the journal or conference.

What are your research rationale and objectives?

What is the knowledge gap that your research addresses? In a global scheme: what makes your research interesting and important? How does your research contribute to the field?

How was your research done?

Describe your approach and explain the methods used.

What were the main observations?

Describe what was directly observed from your described methods.

What are the main conclusions?

What do your results mean and how do they aid our understanding of the topic? What new significant information is derived from your research?

What is the relevant background to understand your work and its conclusions?

What is the minimal information that is necessary to understand your research and its importance?

How does your research advance the field or motivate future research? In what direction should future research proceed?

Useful online resources:

http://writing.wisc.edu/Handbook/presentations_abstracts.html http://writingcenter.unc.edu/resources/handouts-demos/specific-writing-assignments/abstracts#ex2 http://research.berkeley.edu/ucday/abstract.html

Sample Abstract:

Title	
	Gluconeogenic carbon flow of TCA cycle intermediates is critical for Mycobacterium tuberculosis to establish and maintain infection
Authors	Joeli Marrero ^a , Kyu Rhee ^b , Dirk Schnappinger ^a , Kevin Pethe ^c and Sabine Ehrt ^{a,*} ^a Department of Microbiology and Immunology, ^b Department of Medicine, Division of Infectious Diseases, Weill Cornell Medical College, New York, NY 10065; ^c Novartis Institute for Tropical Diseases Pte Ltd., Singapore 138670, Singapore
Rationale	<i>Mycobacterium tuberculosis</i> (Mtb) infection prevalence has escalated to more than one third of the world's population and, concurrently, tuberculosis (TB) disease has an alarming death rate of 1.7 million per year.
Objective	Our overall research goal is to identify Mtb virulence factors that will serve as targets for the development of much needed TB therapies. We have focused on understanding how carbon metabolism serves as a defining feature of Mtb pathogenesis.
Background	Evidence suggests that Mtb primarily relies on fatty acid carbon substrates during <i>in vivo</i> growth. Fatty acid derived carbons are metabolized through central carbon metabolism pathways to meet Mtb's demand for production of energy and biomass. Production of essential macromolecules from fatty acid-derived carbons is mediated through the carbon metabolism pathway gluconeogenesis.
Methods and Results	We employed a genetic strategy to investigate the role of gluconeogenesis in Mtb virulence using a knockout mutant of the gene encoding phosphoenolpyruvate carboxykinase (PEPCK) enzyme, which catalyzes the first committed step of the gluconeogenic pathway. ¹³ C-carbon tracing analysis of this mutant confirmed that PEPCK is sole pathway in Mtb for carbon flow from tricarboxylic acid cyclederived metabolites to glycolytic intermediates. The Mtb PEPCK mutant was severely attenuated for replication and survival in the mouse infection model. Furthermore, depletion of PEPCK during the chronic phase of infection led to killing of the bacilli independently of the IFN _γ dependent immune response.
Conclusion and Future Directions	Our studies highlight that Mtb relies on gluconeogenesis to maintain its fitness throughout the infection pointing to this carbon metabolism pathway as a promising target for the development of novel tuberculosis chemotherapies.