



**CENTER FOR DIABETES
AND METABOLIC DISEASES**
INDIANA UNIVERSITY

8th Annual Diabetes Symposium Guidelines for Writing an Abstract

The purpose of the abstract is to provide a synopsis of your work in a clear (readable, well organized, avoiding jargon), concise (without excess wordiness or unnecessary information), and cohesive (nice flow between the parts) manner. These are guidelines that can help you write a compelling abstract:

Title

The title should clearly describe what your abstract is about, but also be interesting enough to encourage readers to want to learn more

Background and Objective

1. Should explain why your abstract is important or novel
2. Provide the context or explanation for doing the study, not the whole history but the current situation
 - 2.1. What is already known about the subject?
 - 2.2. What is not known, and hence what do you intend to examine?
3. Should state the aim of the study
 - 3.1. What are you hoping to find out or what is your hypothesis?
4. Length: 1 to 3 sentences. If you have just started working on your project, it is appropriate to expand a bit more in this section

Methods

1. Should explain how you did your study
2. Specific population studied
 - 2.1. Include sampling frames and response rates when appropriate
 - 2.2. How many people/animals/samples were included in the research?
3. Quantitative or qualitative methods
 - 3.1. Specific statistical analysis conducted
 - 3.2. Measures and outcomes explored
4. Time frame duration of the study
5. Length: 3 to 8 sentences. If you have just started working on your project, it is appropriate to expand a bit more in this section and reduce the length of Results and Conclusions

Results

1. The results section should explain what you found
2. Describe your main findings with data
 - 2.1. The intervention group was more likely than the control to use metformin - **less good**
 - 2.2. The intervention group was more likely than the control to use metformin ($p < 0.01$) - **better**
 - 2.3. The intervention group was more likely than the control to use metformin (45% vs. 30%, $p < 0.01$) - **best**
3. Concisely describe how your results pertain to your study aim or hypothesis

4. Remember to report nonsignificant differences too
5. Usually the longest section, 3 to 8 sentences

Conclusions

1. This section should explain your main findings and why they are important
2. Describe the primary take-home message
3. Conclusions should be reasonable and supported by the findings
4. Include the Scientific/Clinical/Policy Impact of the research and Implications
5. Length: 2 to 3 sentences

EXAMPLE (STRUCTURED):

Carbon Monoxide–Activated Nrf2 Pathway Leads to Protection Against Permanent Focal Cerebral Ischemia

Bing Wang, Wangsen Cao, Shyam Biswal and Sylvain Doré 

Originally published 18 Aug 2011 | <https://doi.org/10.1161/STROKEAHA.110.607101> | Stroke. 2011;42:2605–2610

[Other version\(s\) of this article](#) 

Abstract

Background and Purpose—

Carbon monoxide (CO) is a gaseous second messenger produced when heme oxygenase enzymes catabolize heme. We have demonstrated that CO can be therapeutic in ischemia-reperfusion brain injury; however, it is unclear whether CO can also offer protection in permanent ischemic stroke or what mechanism(s) underlies the effect. Heme oxygenase-1 neuroprotection was shown to be regulated by Nrf2; therefore, we investigated whether CO might partially exert neuroprotection by modulating the Nrf2 pathway.

Methods—

To evaluate the potential protective effects of CO, we exposed male wild-type and Nrf2-knockout mice to 250 ppm CO or control air for 18 hours immediately after permanent middle cerebral artery occlusion. Infarct volume and neurologic deficits were assessed on day 7. Molecular mechanisms of Nrf2 pathway activation by CO were also investigated.

Results—

Mice exposed to CO after permanent ischemia had $29.6 \pm 12.6\%$ less brain damage than did controls at 7 days, although amelioration in neurologic deficits did not reach significance. Additionally, 18-hour CO treatment led to Nrf2 dissociation from Keap1, nuclear translocation, increased binding activity of Nrf2 to heme oxygenase-1 antioxidant response elements, and elevated heme oxygenase-1 expression 6 to 48 hours after CO exposure. The CO neuroprotection was completely abolished in Nrf2-knockout mice.

Conclusions—

Low-concentration CO represent a neuroprotective agent for combination treatment of ischemic stroke, and its beneficial effect would be at least partially mediated by activation of the Nrf2 pathway.

EXAMPLE (UNSTRUCTURED):

Pancreatic islet cryopreservation by vitrification achieves high viability, function, recovery and clinical scalability for transplantation

Li Zhan^{1,8}, Joseph Sushil Rao^{2,3,8}, Nikhil Sethia⁴, Michael Q. Slama⁵, Zonghu Han¹, Diane Tobolt², Michael Etheridge¹, Quinn P. Peterson^{5,6}, Cari S. Dutcher^{1,4}, John C. Bischof^{1,7,9} and Erik B. Finger^{2,9} 

Pancreatic islet transplantation can cure diabetes but requires accessible, high-quality islets in sufficient quantities. Cryopreservation could solve islet supply chain challenges by enabling quality-controlled banking and pooling of donor islets. Unfortunately, cryopreservation has not succeeded in this objective, as it must simultaneously provide high recovery, viability, function and scalability. Here, we achieve this goal in mouse, porcine, human and human stem cell (SC)-derived beta cell (SC-beta) islets by comprehensive optimization of cryoprotectant agent (CPA) composition, CPA loading and unloading conditions and methods for vitrification and rewarming (VR). Post-VR islet viability, relative to control, was 90.5% for mouse, 92.1% for SC-beta, 87.2% for porcine and 87.4% for human islets, and it remained unchanged for at least 9 months of cryogenic storage. VR islets had normal macroscopic, microscopic, and ultrastructural morphology. Mitochondrial membrane potential and adenosine triphosphate (ATP) levels were slightly reduced, but all other measures of cellular respiration, including oxygen consumption rate (OCR) to produce ATP, were unchanged. VR islets had normal glucose-stimulated insulin secretion (GSIS) function in vitro and in vivo. Porcine and SC-beta islets made insulin in xenotransplant models, and mouse islets tested in a marginal mass syngeneic transplant model cured diabetes in 92% of recipients within 24–48 h after transplant. Excellent glycemic control was seen for 150 days. Finally, our approach processed 2,500 islets with >95% islets recovery at >89% post-thaw viability and can readily be scaled up for higher throughput. These results suggest that cryopreservation can now be used to supply needed islets for improved transplantation outcomes that cure diabetes.

Adapted from:

- Andrade C. How to write a good abstract for a scientific paper or conference presentation. Indian J Psychiatry 2011;53:172-5.
- IMPRS 2020 Abstract Submission, IUSM
- How to write an abstract: https://www.cdc.gov/stdconference/2016/how-to-write-an-abstract_v3.pdf
- Stroke 42(9):2605-10 (2011); doi: 10.1161/STROKEAHA.110.607101
- Nature Medicine 28(4):798-808 (2022); doi: 10.1038/s41591-022-01718-1