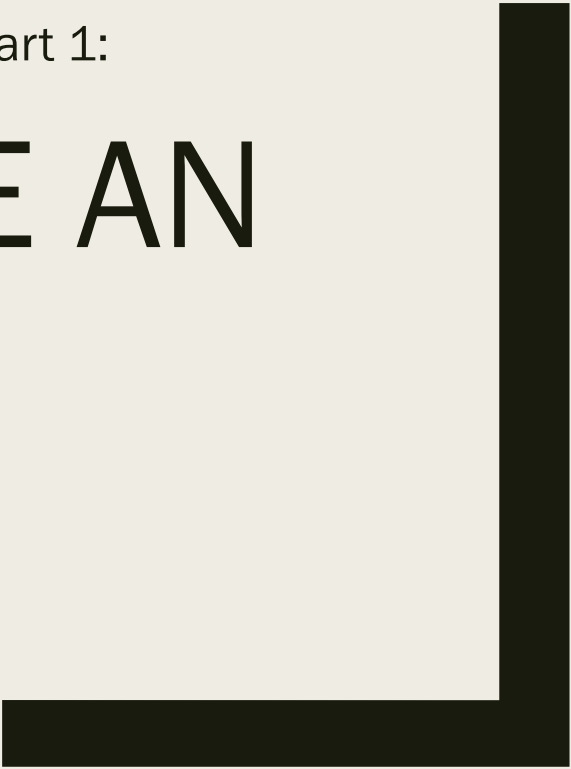




MSBSS/GSLC Get Noticed! Webinar, part 1:

HOW TO WRITE AN ABSTRACT

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SOT Rules and Guidelines

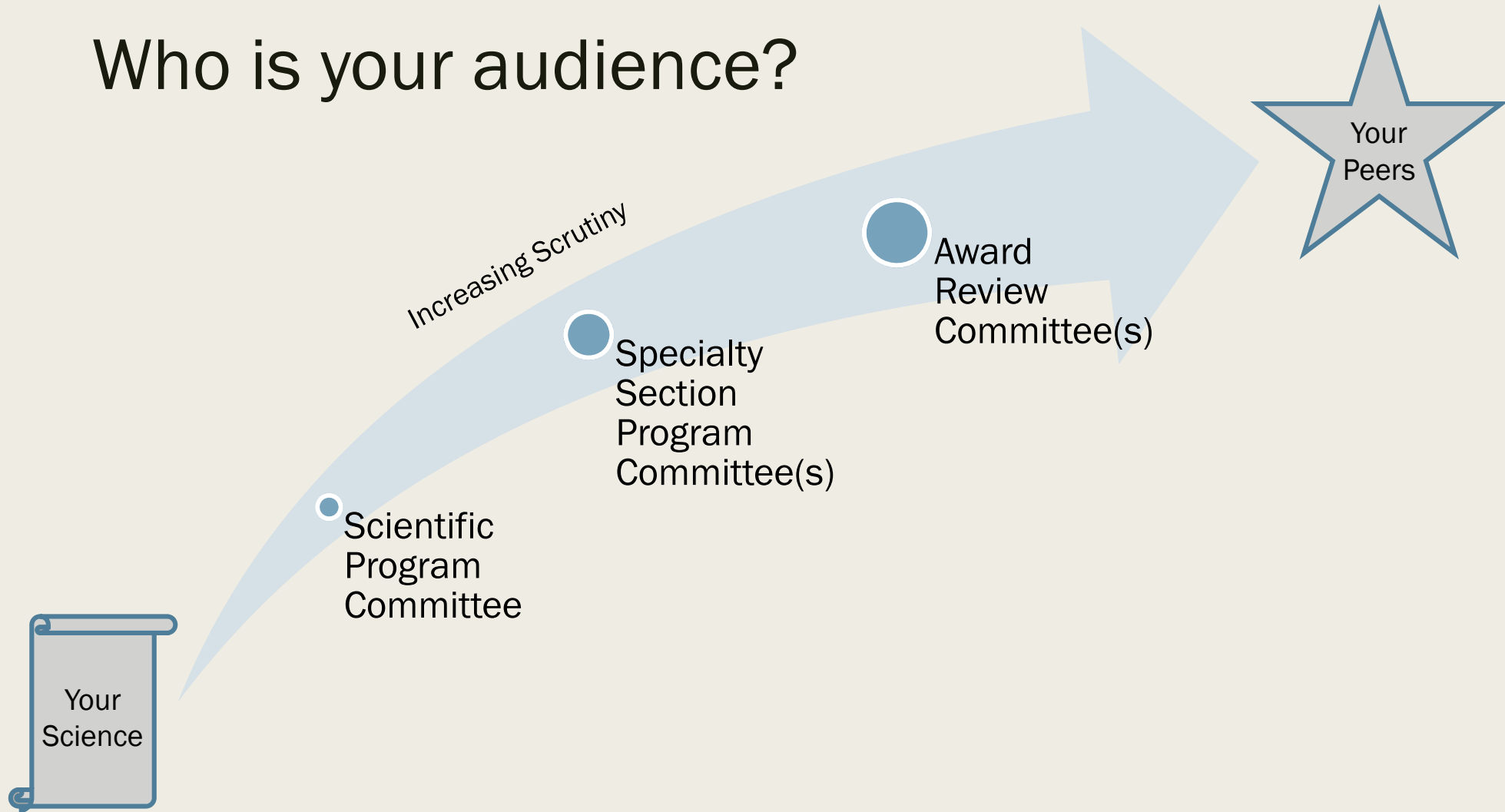
<https://www.toxicology.org/events/am/AM2020/abstracts-presenters.asp>

- Abstract deadline: **October 18, 2019**
- 2,300 character limit
- No tables, figures, or chemical structures
- No headers
- Define your acronyms.
- Identify your compounds.
- Be sure that your work follows ethical guidelines, including IACUC/IRB approval.
- Show up at the meeting, and present your work.

Key Points

1. Know your audience(s).
2. Tell them the following about your project:
 - *Introduction/background: Introduce the problem.*
 - *Hypothesis/purpose statement: Say how you approached the problem (conceptually).*
 - *Methods: Tell the audience what you did (nuts and bolts).*
 - *Results: Tell the audience what you found.*
 - *Conclusions: Tell the audience what it means.*
3. Give your work a compelling title.
4. Be clear, concise, and honest.

Who is your audience?



Example: an Abstract That Worked

PL 3274 Mono-(2-Ethylhexyl) Phthalate Disrupts Retinoic Acid Signaling and Gonadal Sex-Determination Pathways in *Ex Vivo* Cultured Rat and Mouse Fetal Testes

D. J. Spade¹, S. J. Hall¹, J. D. Wortzel¹, G. Reyes², and K. Boekelheide¹. ¹Brown University, Providence, RI; and ²College of Mount Saint Vincent, Riverdale, NY.

In utero phthalate exposure results in significant testicular pathology in mammalian testes, despite the lack of an anti-androgenic effect on mouse and human fetal testes. Retinoic acid signaling is critical for sex determination, and exogenous retinoic acid disrupts sex determination in cultured rat fetal testes. Phthalates are peroxisome proliferators that bind to peroxisome proliferator-activated receptors and may engage in crosstalk with other nuclear receptors, including retinoic acid receptors. Therefore, we hypothesized that retinoic acid signaling is disrupted by phthalates, contributing to phthalate toxicity in the fetal testis. To test this hypothesis, rat and mouse fetal testes isolated on gestation day 15 and 14, respectively, were exposed in tissue culture to 10⁻⁶ M all-trans retinoic acid (ATRA) with or without 10⁻⁶ to 10⁻⁴ M mono-(2-ethylhexyl) phthalate (MEHP). In rat fetal testes, ATRA exposure caused a loss of seminiferous cord structure, accompanied by an increase in expression of a retinoic acid receptor target gene, *Rbp1*, and sex determination genes *Nr0b1* and *Wnt4*. The testicular pathology was reversed in a concentration-dependent manner by addition of MEHP. The interaction between MEHP and ATRA also led to an additive increase in *Rbp1* expression, but non-linear response in expression of *Nr0b1* and *Wnt4*. In the mouse, ATRA-MEHP co-exposure resulted in similar testicular pathology. Given this evidence that MEHP interacts with ATRA to influence both retinoic acid signaling and sex determination in the fetal testis, we conclude that disruption of retinoic acid signaling is a mechanism of phthalate toxicity in the fetal testis of both rats and mice.

- Introduction/Background
- Hypothesis
- Methods
- Results
- Conclusion
- Is it perfect? No!

Introduction/Background

- Just a few sentences
- Tell the audience what is known about the compound(s) and why the toxicity is important
- End with a question or knowledge gap ★

Compound-of-Interest causes acute liver injury in the rat...

Human exposure to Compound-of-Interest has increased rapidly...

An estimated 3,000 new cases of acute Compound-of-Interest toxicity occur annually...

★ The role of Biological Process in Compound-of-Interest toxicity is unknown...

Statement of Purpose/Hypothesis

- One sentence
- Tell the audience how you approached the problem. Be as direct as possible.

We **hypothesized** that activation of Imaginary Pathway was **required** for Compound-of-Interest-induced hepatotoxicity.

The **purpose** of this study was to **quantify the effect of age** on Compound-of-Interest-induced hepatotoxicity.

The **purpose** of this study was to **identify the major routes** of Compound-of-Interest metabolism.

Methods

- Tell the audience what you did.
- Include details about the exposure model and any unique tools or analyses.
- Don't get too far into the weeds.

Adult male and female Sprague Dawley rats were exposed to a single dose of 3-30 μg Compound-of-Interest/kg body weight by oral gavage.

We created a novel human primary hepatocyte organoid model to test the gene expression response to Compound-of-Interest.

We tested Compound-of-Interest toxicity in wild-type and Imaginary Receptor knockout rats.

Results

- Tell the audience what you found.
- What were the key outcomes of your experiments?
- You don't have enough space to include everything. No tangents!

Imaginary Receptor activity increased in a dose-dependent manner, beginning 1 hour after exposure to Compound-of-Interest.

The Imaginary Receptor inhibitor, Compound 2, rescued acute hepatotoxicity of Compound-of-Interest.

Compound-of-Interest enhanced expression of genes involved in Biological Process, measured by RNA-seq. ★

88% of Compound-of-Interest was converted to Metabolite A in the liver.

Conclusion(s)

- Bring it home.
- What is the ONE key point that the audience will learn by **attending your presentation?**

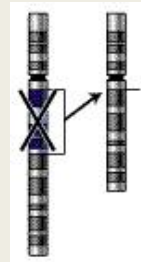
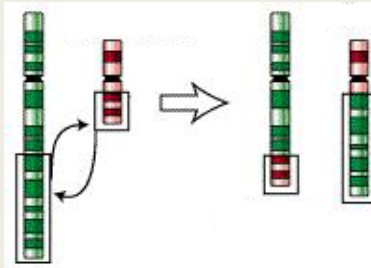
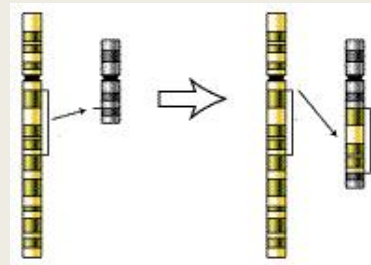
Therefore, Metabolism of Compound-of-Interest to Metabolite A contributes to Compound-of-Interest hepatotoxicity.

We conclude that Imaginary Receptor activation is required for Compound-of-Interest hepatotoxicity.

These results **support the hypothesis** that hydroxylation by CYP1X is a primary route of Compound-of-Interest metabolism.

Don't jump around. Remember, structure determines function.

Background
Hypothesis
Methods
Results
Conclusion



Background
Methods
Results
Background
Results
Conclusion

Be clear and concise.

- Remove extraneous words.
— ~~*Several studies have shown...*~~
- Resist the urge to digress.
— ~~*Interestingly...*~~
- Triple-check your abstract for jargon.
- Help the audience follow the story.
 - *We hypothesized that...*
 - *To address this gap...*
 - *We found...*
 - *We conclude...*

Stay focused!

- You know a lot of things. Not all of them belong in your abstract.

Be honest.

- Tell the audience what you found. Don't overstate your findings.

Finally, give your work a compelling title.

- Your title will help you capture the interest of program committee, award committee, and conference attendees.
- Readers will scan your title for:
 - Toxicant(s)
 - Organ systems, targets, mechanisms, or disease states
 - Key conclusions
- Use an action verb.
- State a key finding in a declarative sentence.

Don't do this:

Effect of Compound-of-Interest on
Imaginary Pathway in the Liver

Do this:

Compound-of-Interest Induces Liver
Toxicity through Imaginary Pathway

Good luck. Remember your goals.

