Submit an Abstract

* Fields are required

Student Information

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***Pronouns:**
- ✓ She/Her/Hers
- ○ He/Him/His
- ○ They/Them/Theirs
- ○ Other:

***Academic Year**
- ○ First year
- ○ Sophomore
- ✓ Junior
- ○ Senior
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***Major (Please select)**
Biomedical Engineering

Home University (if not MSU): N/A

***Academic College** (check all that apply)
- ○ I am not a student enrolled at MSU
- ○ College of Academic Affairs
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- ○ College of Forest Resources
- ○ College of Veterinary Medicine
- ✓ James Worth Bagley College of Engineering

*Are you a member of Shackouls Honors College or an Honors program at another university?
- ✓ Yes
- ○ No

Funding source for this research project (e.g., an REU or other funded undergraduate research program) (select all that apply)
- ✓ Shackouls Honors College Research Fellowship
- ○ College of Agriculture and Life Sciences URSP
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- ○ Other:

***Does the abstract that you are submitting represent a completed research project or research that is still in progress?**

- ○ Completed
- ✓ In progress

If this research was completed as a part of an academic course, please identify which course (e.g., AN 1143 Intro to Cultural Anthropology):

Course Code: N/A Course Name: N/A

Faculty Advisor(s) Information

This information will appear in the abstract booklet exactly as submitted below. Please be sure to verify the spelling of your advisor's name and the accuracy of their department and email address.

***Is your Primary Advisor from MSU?**

- ✓ Yes
- ○ No

If yes is selected, a field for the student to input their advisor's MSU NetID should appear. When that information is entered, the fields for the primary advisor's name, departmental affiliation, and email should autofill.
***Primary Advisor MSU NetID:** ex1234
***Primary Advisor First Name:** Matthew
***Primary Advisor Last Name:** Ross
***Primary Advisor Departmental Affiliation**: Department of Comparative Biomedical Sciences
***Primary Advisor Email:** ex1234@msstate.edu

Secondary Advisor First Name (Do not include title)**:** N/A
Secondary Advisor Last Name (Do not include suffix)**:** N/A
Secondary Advisor Departmental Affiliation: N/A
Secondary Advisor Email: N/A

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***Primary Advisor First Name** (Do not include title)**:** N/A
***Primary Advisor Last Name** (Do not include suffix)**:** N/A
***Primary Advisor Departmental Affiliation**: N/A
***Primary Advisor Email:** N/A

Secondary Advisor First Name (Do not include title)**:** N/A
Secondary Advisor Last Name (Do not include suffix)**:** N/A
Secondary Advisor Departmental Affiliation: N/A
Secondary Advisor Email: N/A

Project Information

***Category**

- ✓ Biological Sciences & Engineering
- ○ Physical Sciences & Engineering
- ○ Social Sciences
- ○ Humanities & Arts

If Biological Sciences & Engineering, Physical Sciences & Engineering, or Social Sciences is selected, automatically select project type as poster:

***Project Type**

- ✓ Poster

***Project Title**

Hydrolytic Metabolism of Arachidonic Acid-containing Triacylglycerols by CES1 in Macrophages

In addition to you, how many Co-Authors does this project include?

Two

Co-Author names(s) (Do not include any Faculty Advisors listed above):

First Name: Jane Last Name: Doe
First Name: John Last Name: Smith
Add another Co-Author

Abstract Submission

***Abstract** (max 300 words)

Triacylglycerols (TAGs) are lipids found in cytoplasmic lipid droplets. Toll-like receptor activation causes increased TAG accumulation in macrophages and enhances their inflammatory function. The increase in TAG-containing lipid droplets in the setting of inflammation is due to both enhanced biosynthesis and reduced fatty acid release. Human carboxylesterase 1 (CES1) is a member of the serine hydrolase superfamily and catalyzes the hydrolysis of TAG lipids, including those containing oxidized polyunsaturated fatty acids. CES1 is expressed in monocytes/macrophages, although its function in these cells is unclear. When CES1 expression was stably knocked down in THP-1 macrophages, marked increases in arachidonic acid-containing TAGs was detected by LC-HRMS; ~15-20-fold higher levels than those in control THP-1 macrophages where CES1 expression was normal. In addition, the metabolic fate of 15-hydroxyeicosatetraenoic acid (15-HETE), which is the enzymatic product derived from IL-4-induced ALOX15, was found to be significantly altered in the CES1 knockdown (KD) cells. Exogenously added 15-HETE (300 nM) was rapidly metabolized in macrophages by peroxisomes to tetranor 15-HETE derivatives, or it was metabolically incorporated into the TAG pool. Interestingly, we found that the level of esterified 15-HETE in TAGs was ~7-fold higher in CES1KD cells than in control cells after a 6-hr treatment. Furthermore, both RNA-seq and RT-qPCR data indicated that there were pronounced differences in the responses of control and CES1KD macrophages to M1 and M2 inflammatory stimuli (LPS/IFNg and IL-4, respectively), with the CES1KD macrophages exhibiting a more pro-inflammatory phenotype. The observed metabolic changes in TAG/oxylipin disposition and the resulting altered immunophenotype in the CES1KD cells is likely due to the reduced TAG hydrolytic activity and subsequent buildup of cellular TAGs, which enhances inflammation.

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