BIOC5915.03 — Scientific Communication in Biochemistry and Molecular Biology II

Transcript Title: Biochem Communications II

Instructor: Dr. David Waisman **E-mail:** david.waisman@dal.ca **Office LocationTupper:** 11-L2

Time and place: Winter Term 2024 Beginning January 9th

Tuesday from 1:05 – 2:25. Classes are online unless notified otherwise. Room Tupper BA3 has been booked.

Course Description:

This course provides students with experience in the oral presentation of scientific data and organizing a scientific symposium. Interactive faculty and peer feedback are used to hone student skills, emphasizing both clarity of presentation and the ability of students to discuss specialist topics in general terms.

Format: Symposium (including practice, organization, and presentation) and attendance at Department Seminars

Credit Hours: 3

General Learning Outcomes: By the end of this course, it is expected that students should be able to:

1. Prepare and deliver an oral scientific presentation to a peer audience.
2. Appraisal of scientific presentations to determine features that enhance or deter from the communication of the science.
3. Present scientific data by critically analyzing the seminars of others.
4. Summarize the objectives and results of a published scientific study in a format similar to a scientific journal abstract.
5. Critically evaluate a scientific publication.
6. Organize an oral presentation session for a scientific meeting.

Course Material and Assigned Readings:

Course prospectus, class material, links to papers discussed in class, and other assigned readings are available for download from the course website (http://www3.biochem.dal.ca/5915/).

Course Organization:

Part 1 applies and hones presentation skills. Students select a scientific topic of their choice and prepare individual presentations (specific guidelines for topic selection are provided). Through a 5-10 minute

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'sales pitch' talk, each student first convinces their BIOC 5915 classmates and instructor that their selected topic is exciting and worthwhile. Then, the students present a practice seminar to the class, and the students will critique each other's presentations using the seminar evaluation form. Afterward, an afternoon symposium is presented to the Department (and Faculty), with each student presenting 10 minutes in length, including a 5-minute question period. Emphasis is on clarity of presentation and the ability to discuss the topic in general terms. Each student writes an abstract summarizing their presentation and a "News and Views" summary of their paper, highlighting its significance and content. Two faculty members evaluate the News and Views. Finally, as a group, the students organize the symposium, generate a symposium abstract booklet, and disseminate notices of the symposium and the booklet, in advance of the symposium date, to relevant departments at Dalhousie University. The symposium presentation of each student is evaluated by at least two faculty excluding the course instructor. It is based on the clarity of the presentation, the ability to discuss the topic in general terms, and the answer to questions based on background reading relevant to the topic.

Part 2
This aspect runs throughout the term and provides opportunities to evaluate seminars by experienced speakers in the Departmental Seminar program. The critique form has been simplified to focus on the key messages you glean from each talk while providing opportunities to comment on what you did and did not like about the presentation. By critiquing the seminar presentations, the student will learn how to fine-tune their presentations to maximize clarity and promote the flow of ideas. These critiques are designed to develop the ability to fine-tune one's presentations to optimize clarity and encourage the flow of ideas.

Schedule for Winter 2024

Part 1 – Tuesday 1:05 - 2:25 PM
January 19 -(1/2 h) Introduction, topic selection advice & abstract requirements
January 16 - Elements of giving a good talk-DMW
January 23 - 'Sales pitch' talks: 5-10 minutes
January 30 - Practice I
January 30- Symposium abstracts emailed to the instructor
February 6 - Practice II
February 13 -Practice III
February 19-23, no classes (Study Break)
February 27-Practice IV
March 5 Practice (if required) - *News and Views* emailed to the instructor.
March 12- Open practice and PowerPoint testing – probably via Microsoft Teams
March – **GRADUATE STUDENT SYMPOSIUM** –TBA– 10:30 - 1:00 p.m

Part 2
January 11- April 3. (Wednesdays 4:00-5:00) Biochemistry Seminars.
Critiques of all three Departmental seminars must be completed by April 1.

COURSE EVALUATION
Evaluation components: Presentations, written abstract and news and views summary of the paper, and seminar critiques.

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Course evaluation: Final grades as per the Dalhousie University grading protocol https://www.dal.ca/campus_life/academic-support/grades-and-student-records/grade-scale-and-definitions.html. The following grading scheme is used to ensure that minimum standards are met and that students perform consistently throughout the course. *NOTE: A total grade of > 70% is required in each of the course parts for a passing grade.

90% for Part 1

20%-*News and Views* style analysis of the paper to be presented at the symposium (average of two faculty members' evaluations).
60% = presentation at the symposium (average of two faculty members' evaluations).
10% = abstract (graded by instructor)

10% for Part 2 = critiques of departmental seminars – see guidelines below

Guidelines for Mini-Symposium Topics and Talks

1. Each student chooses a scientific topic and designs a presentation on that topic as part of a mini-symposium by the course participants.
2. Each topic is based on a 2023 research paper published in the journal, Nature, Science, or Cell.
3. The overall topic permits an in-depth understanding needed to prepare the talk and answer questions posed by the audience and faculty evaluators.
4. As a group, the course participants determine whether the symposium follows a specific theme or covers diverse topics, in each case focusing on exciting/interesting new discoveries.
5. Each student prepares a 200-250-word abstract for their topic suitable for compilation into a symposium abstract booklet. A selection of key references, including the primary paper, accompanies each abstract.
6. Course participants evaluate each other's abstracts and practice talks.
7. Course participants organize the symposium, including compilation and distribution of the abstract booklet and a notice, advertising the symposium to members of relevant departments at Dalhousie University sufficiently in advance of the symposium to promote attendance.
8. The length of each talk, the amount of time allotted for questions at the end of each talk, and the order in which the talks are given is determined by the course participants in consultation with the course instructor (with eight students and a maximum of 4 h available, talks of 10 minutes with 5-10 minutes for questions could be accommodated with a short break in the middle of the session).

Guidelines for Abstract of paper-Background--describe the topic in simple terms that the readers will understand. 1. Describe the key research question or hypothesis. 2. Summarize why this research is unique. 3. Summarize how the researchers attacked the research question. 4. Summarize the major findings, and 5. impact of the research. The abstract should answer the following--What was done, why it was done, how it was done, what was discovered, and what is the significance of the research. The references to the paper should be included at the end-use the journal Nature format.

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Guidelines for *News and Views* analysis of the student's symposium publication

The *News and Views* articles are intended to inform nonspecialist readers about new scientific advances. Your *News and Views* paper (2 pages, single-spaced, 12-point Times Roman font, plus a third page consisting of a single figure (graphical abstract with figure legend). Your paper should highlight its importance and provide a synopsis of your paper in terms of what had been known previously, why this publication is not merely an incremental advance, and how it has advanced the field. For specific examples, see the end of the document.

Guidelines for Critiques of seminars. A portion of your grade for this course is based on the critiquing of seminars. Three (3) seminars are critiqued for a grade in this part of the course. To pass the course, all three seminar evaluations must be submitted to the instructor on or before **April 1.** It is highly recommended that these critiques be emailed to the instructor within several days after the seminar presentation.

Critiques must be for original research seminars (not journal club or group meeting presentations), of departmental seminars in Biochemistry & Molecular Biology. Note that attendance at departmental seminars is expected of *all* Biochemistry & Molecular Biology graduate students. For critiques of seminars outside the Department, the seminar must be related to the general theme of a graduate degree in Biochemistry & Molecular Biology (i.e., thinking about biomolecule(s) at the molecular and/or submolecular level). If you have a degree requirement (e.g., TA activity or a class) that conflicts with the Wednesday 4 PM seminar slot, tell the 5915 instructor so that arrangements can be made.

Seminar evaluations should be provided for speakers at various levels: evaluations will be based on seminars given by faculty members and some by graduate students.

Your completed evaluation forms are submitted by email to Dr. Waisman at the end of the seminar or, at the latest, within one week of the seminar in question. A critique must be analytical and detailed to count for credit – that is, a critique consisting only of a phrase such as “pretty PowerPoint slides and good speaking dynamics” would not result in a passing grade. Your evaluations are confidential and will not be available to the speaker. Recognize the added value: keep in mind what you do and don’t like about the presentations given by others as you design your own seminars.

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COURSE POLICIES ON MISSED OR OVERDUE ASSIGNMENTS

A student who misses an evaluation component of the course due to illness must notify the instructor, course coordinator, or department office prior to the scheduled time or due date for that component. The student must also complete a Student Declaration of Absence form (available on the course website) or provide alternate verification of the absence to their instructor via instructor e-mail within three (3) calendar days following the last day of absence. An alternative due date will be established by the instructor and shall normally be within seven calendar days after the original due date. Absence for non-medical reasons is not ordinarily acceptable unless prearranged with the instructor. A missed evaluation component for which no satisfactory arrangement has been made will be given a mark of zero. All attempts will be made to accommodate requests for extensions of deadlines where illness or personal crisis, i.e., extenuating circumstances that would affect the student's ability to fulfill the criteria for the award of credit points or to perform to the best of the student's ability in assessment events, have occurred. It is the responsibility of the student to notify the instructor and/or the course coordinator of any extenuating circumstances and to request an extension.

If weather-related events and other natural disasters are serious enough for the university to be closed, classes will be canceled, and the schedule will be adjusted to accommodate the missed date(s).

DEPT. OF BIOCHEMISTRY & MOLECULAR BIOLOGY POLICY ON PLAGIARISM

What is plagiarism?

"Dalhousie University defines plagiarism as the presentation of the work of another author in such a way as to give one's reader reason to think it to be one's own. Plagiarism is a form of academic fraud."† The Department is committed to protecting honest students against the devaluation of their work by students who resort to plagiarism.

Some examples of plagiarism include (but are not restricted to):

Submitting as your own work any material created, in whole or in part, by someone else, **including material created in collaboration with other students**, unless specifically allowed by the course instructor and credited appropriately.

Paraphrasing extensively or copying from sources such as the Internet, journal articles, or books (including textbooks) without crediting the original author or source.

Using another student's laboratory data, unless specifically allowed by the course instructor and credited appropriately.

Submitting, in whole or in part, any work that has been submitted in another course, or re-submitting the same work in different years of the same course.

How can plagiarism be detected?

If required by the Instructor, work submitted for credit must be submitted in electronic as well as hard copy form. Submissions may be screened by one or both of the following methods:

A pattern recognition program that compares all submissions with one another as well as submissions from previous years. Every individual has a unique pattern of writing. This program will detect submissions that are derived from a common source, even if words or phrases have been changed.

A third-party computer-based assessment system that compares submissions against a large database, including previous submissions and Internet sources.

What are the consequences of plagiarism?

"Plagiarism is a serious academic offense which may lead to loss of credit ['F' in a course], suspension or expulsion from the University or even the revocation of a degree."† **At Dalhousie University, the Department is obligated to refer any cases of suspected plagiarism to the Senate Discipline Committee**, which will then conduct a hearing to evaluate the innocence or guilt of students alleged to have committed an act of plagiarism.

† http://www.dal.ca/dept/university_secretariat/academic-integrity/academic-policies.html

How can accusations of plagiarism be avoided?

You can avoid accusations of plagiarism by:

9. Preparing all submissions independently and ensuring that they are expressed in your own unique writing style.
10. Never share any written or electronic material with other students. You may discuss ideas with other students, but you may not work with another student while preparing materials you are planning to hand in.
11. Acknowledging any material paraphrased extensively or copied from sources such as the Internet, journal articles, or textbooks. Paraphrasing of short phrases from the course textbook need not be acknowledged.
12. Guarding all your work, both drafts, and final submissions, to ensure that no one else can copy it. If you provide access to your work and someone copies it, then you may have to appear before the Senate Discipline Committee to establish that you are the original creator of the work. If you suspect that someone has taken any of your work, notify your course instructor immediately.
13. Using only laboratory data that you actually collected in the lab. Altering laboratory data is not permitted.

University Policies and Statements

This course is governed by the academic rules and regulations set forth in the University Calendar and by Senate (https://academiccalendar.dal.ca/Catalog/ViewCatalog.aspx)

Academic Integrity

At Dalhousie University, we are guided in all of our work by the values of academic integrity: honesty, trust, fairness, responsibility, and respect (The Center for Academic Integrity, Duke University, 1999). As a student, you are required to demonstrate these values in all of the work you do. The University provides policies and procedures that every member of the university community is required to follow to ensure academic integrity.

Information: https://www.dal.ca/dept/university_secretariat/academic-integrity.html

Accessibility

The Advising and Access Services Centre is Dalhousie's center of expertise for student accessibility and accommodation. The advising team works with students who request an accommodation as a result of a disability, religious obligation, or any barrier related to any other characteristic protected under Human Rights legislation (Canada and Nova Scotia).

Information: https://www.dal.ca/campus_life/academic-support/accessibility.html

Student Code of Conduct

Everyone at Dalhousie is expected to treat others with dignity and respect. The Code of Student Conduct allows Dalhousie to take disciplinary action if students don't follow this community expectation. When appropriate, violations of the code can be resolved in a reasonable and informal manner—perhaps through a restorative justice process. If an informal resolution can't be reached or would be inappropriate, procedures exist for formal dispute resolution.
Code: https://www.dal.ca/dept/university_secretariat/policies/student-life/code-of-student-conduct.html

Diversity and Inclusion – Culture of Respect

Every person at Dalhousie has a right to be respected and safe. We believe inclusiveness is fundamental to education. We stand for equality. Dalhousie is strengthened in our diversity. We are a respectful and inclusive community. We are committed to being a place where everyone feels welcome and supported, which is why our Strategic Direction prioritizes fostering a culture of diversity and inclusiveness.
Statement: (http://www.dal.ca/cultureofrespect.html)

Recognition of Mi'kmaq Territory

Dalhousie University would like to acknowledge that the University is on the Traditional Mi'kmaq Territory. The Elders in Residence program provides students with access to First Nations elders for guidance, counsel, and support. Visit the office (Rm 3037, McCain Building), e-mail (elders@dal.ca) or leave message (902-494-6803).
Information: https://www.dal.ca/campus_life/communities/indigenous.html

Important Dates in the Academic Year (including add/drop dates)
https://www.dal.ca/academics/important_dates.html

University Grading Practices
https://www.dal.ca/dept/university_secretariat/policies/academic/grading-practices-policy.html

Student Resources and Support

Advising

General Advising https://www.dal.ca/campus_life/academic-support/advising.html

Science Program Advisors: https://www.dal.ca/faculty/science/current-students/academic-advising.html

Indigenous Student Centre: https://www.dal.ca/campus_life/communities/indigenous.html

Black Advising Centre: https://www.dal.ca/campus_life/communities/black-student-advising.html

International Centre: https://www.dal.ca/campus_life/international-centre/current-students.html

Academic supports

Library: https://libraries.dal.ca/

Writing Centre: https://www.dal.ca/campus_life/academic-support/writing-and-study-skills.html

Studying for Success: https://www.dal.ca/campus_life/academic-support/study-skills-and-tutoring.html

Copyright Office: https://libraries.dal.ca/services/copyright-office.html

Fair Dealing Guidelines https://libraries.dal.ca/services/copyright-office/fair-dealing.html

Other supports and services

Student Health Services: https://www.dal.ca/campus_life/health-and-wellness/health-services/services.html

Counselling: https://www.dal.ca/campus_life/health-and-wellness/counselling.html

Student Advocacy: https://www.dsu.ca/dsas

Ombudsperson: https://www.dal.ca/campus_life/safety-respect/student-rights-and-responsibilities/where-to-get-help/ombudsperson.html

Safety

Research Lab Safety https://www.dal.ca/content/dam/dalhousie/pdf/dept/safety/lab_policy_manual_2007.pdf

Biosafety: https://www.dal.ca/dept/safety/programs-services/biosafety.html

Chemical Safety: https://www.dal.ca/dept/safety/programs-services/chemical-safety.html

Radiation Safety: https://www.dal.ca/dept/safety/programs-services/radiation-safety.html

Scent-Free Program: https://www.dal.ca/dept/safety/programs-services/occupational-safety/scent-free.html

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EXAMPLE OF A NEWS AND VIEWS ARTICLE

NEWS AND VIEWS

Updated Model of Autophagy Leads to New Potential Cancer Treatment

By: Jordan Thompson

Macroautophagy (autophagy) is the process of cellular recycling that allows for the reuse of cellular phospholipids and amino acids[1]. The process occurs by the formation of the isolation membrane that elongates and then engulfs the cytosolic material. The enclosed double membrane structure is now the fully formed autophagosomes. These structures can then be brought to lysosomes for degradation and complete the process of recycling.

Though researchers understood the process by which autophagy occurs, they did not fully understand the source of phospholipids for the formation of autophagosomes. Due to this, Ogasawara et al. (2020)[2], hypothesized that the process of isolation membrane formation involved the enzyme CTP: phosphocholine cytidylyltransferase (CCT) given that it is a regulating enzyme the formation of phosphocholine (PC)[3]. Since PC is the most abundant phospholipid in mammalian cells, it would follow that *de novo* synthesis might be supplying the necessary membrane phospholipids from the Kennedy pathway.

To test this theory, the authors implemented a fluorescent PC label through the use of a modified choline head group. Their co-localization studies indicated that PC synthesis was occurring near autophagy-related proteins such as ULK1, DFCP1, WIPI1, and LC3. This indicated that PC synthesis is occurring near sites of autophagy. They then labeled the preexisting PC and compared the number and size of the PC puncta with the *de novo* PC. From these experiments, they determined that the majority of the PC incorporated into the autophagic membrane is generated from de novo synthesized PC, though preexisting PC was also incorporated, albeit at a lower density.

After validating the hypothesis that *de novo* PC is forming autophagic membranes, the authors then looked to the regulating enzyme of PC synthesis, CCT. There are 3 isoforms of CCT: nuclear CCTα, cytoplasmic CCTβ2, and cytoplasmic CCTβ3[4]. To test what isoforms were important in regulating autophagy, the authors subjected cells to starvation condition media. This causes the PC to gradually become exhausted. This increases the relative importance of *de novo* PC synthesis for autophagy as a result. The researchers used radiolabeled choline incorporation experiments to show that only the CCTβ3 isoform had a starvation-induced increase in PC synthesis. This was accompanied by an autophagic flux experiment. This consists of treating cells so that the fully formed autophagosomes cannot fuse with the lysosome. The activated LC3-II is quantified and compared against the untreated cells to determine the excess accumulated protein to determine the amount of autophagy occurring. Cells that overexpressed CCTβ3 had minimally elevated autophagy during a short period of starvation in comparison to the basal levels. Further, the isoform CCTβ3 also localized with the autophagy-related proteins DFCP1, WIPI1, and

LC3 using immunofluorescence imaging. These data indicate that CCTβ3 is recruited to autophagic membranes to activate PC synthesis.

The authors then tested to see how long-term starvation affected the autophagic flux. In contrast with the previous experiment, cells were subjected to starvation periods of 8 or 24 h in contrast with the previous 1 h timepoints. This time, they saw drastic effects in the autophagic flux in cells overexpressing the CCTβ3 isoform. This was then validated with siRNA experiments showing that cells depleted of CCTb3 had a decreased autophagic flux and lower radiolabeled choline integration. This demonstrated that CCTβ3 activation becomes increasingly more important for autophagy during prolonged starvation.

The CCT enzymes have previously been shown to interact with lipid droplets (LDs), so the authors were curious if starvation played a role in CCTβ3 interaction with LDs. LDs are storage organelles that consist of a neutral lipid core surrounded by a phospholipid monolayer[5]. Because of their composition, they act as centers of lipid and energy homeostasis[6]. The authors first tested to see which of the CCT isoforms were recruited to LDs during prolonged starvation. Only the CCTβ3 isoform showed any interaction with lipid droplets in a starvation-dependent manner. The authors also tested if the oleate-induced CCT recruitment to LDs was shared with the CCTβ3, as the other isoforms have been extensively characterized with this response[7]. Oleate treatment alone was insufficient for CCTβ3 recruitment to LDs. This indicated that only prolonged starvation recruited CCTβ3 to LDs. The authors followed this up with both live cell imaging and electron microscopy and showed that the isolation membrane originates in the vicinity of LDs in starved cells.

The researchers then developed a CCTβ knockout cell line to further test its importance in human cancer cells. The knockout cell lines had essentially no autophagy during starvation, while transfection of the knockout cells with CCTβ3 reestablished the autophagic flux. This indicated that CCTβ3 is necessary for autophagy. Next, they did survival experiments to see if the knockout cell line could survive in starvation conditions. The knockouts died out significantly more than the wild-type cells after day 3, and essentially all were dead by day 4.

This paper set out with the goal of determining the source of phospholipids for autophagic membranes and was able to develop a comprehensive model of starvation-induced autophagy (Fig 1). The authors postulated that their data supported the concept that recruitment of CCTβ3 to LD allows for the production of CDP-choline in close proximity to the diacyl-glycerol (DAG) required for PC synthesis. The source of the DAG is coming from the large pools of triacylglycerol in the lipid droplets being hydrolyzed. This localized enzyme machinery for the production of PC is what ultimately allows for the formation of autophagic membranes. The researchers also managed to sprinkle in a potential cancer treatment on top of the robust updated model. Previous inhibitors of autophagy have had the issue of not being selective to just cancer cells and having other side effects than just autophagy inhibition[8]. Due to the faster metabolism of cancer cells, they rely on autophagy more than healthy cells. They proposed that inhibition of CCTβ3 could act as a potential cancer treatment. The authors leave us with questions surrounding the overall applicability of such a treatment but do show an intimate relationship between cancer and phospholipid research.

Graphical abstract

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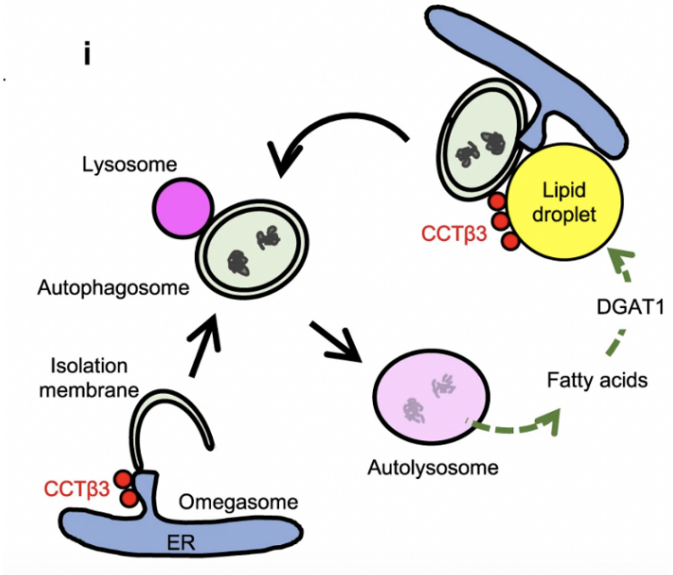

Figure 1. Updated model of starvation-induced autophagy by CCTβ3 regulation.
In cells starved for a short time (1 h), CCTβ3 is activated near autophagic membranes. After prolonged starvation (8 h), CCTβ3 is recruited to LDs generated from autophagic digests. The TAG pools in the LDs are hydrolyzed to form DAG. The CDP-choline synthesized by CCTβ3 and the DAG is then used to form PC by CETP. This PC synthesis provides the phospholipid content required for the formation of the isolation membrane, thereby sustaining autophagy.

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EXAMPLE OF AN ABSTRACT (SUMMARY)

ACSL4 dictates ferroptosis sensitivity by shaping cellular lipid composition

Sebastian Doll, at al.[8]

Ferroptosis, a recently discovered mode of cell death, is an iron-dependent pathway that occurs as a consequence of lipid peroxidation.[1–4] Previously, the regulation of ferroptosis has been known to be controlled by a single pathway catalyzed by glutathione peroxidase 4 (GPX4), which reduces toxic lipid hydroperoxides into lipid alcohols.[2,5,6] Inducing ferroptosis by targeting different regulators of the GPX4 pathway has been of significant therapeutic interest.[5,7] As previous studies have found that cancer cells treated with GPX4 inhibitors exhibit varying levels of resistance to ferroptosis induction, Doll *et al.* (2019) hypothesized that an unidentified anti-ferroptosis pathway may exist that is GPX4-independent.[2,3,6,8] An expression cloning technique in a ferroptosis-resistant cell line was used to identify ferroptosis suppressor protein 1 (FSP1) as an anti-ferroptotic protein.[2] The cell viability in overexpressed FSP1 or mock cells was analyzed upon treatment with GPX4 inactivator, Ras selective lethal molecule 3 (RSL3).[2] In contrast to the mock cells, the FSP1 overexpression cells continued to proliferate upon GPX4 inactivation, thereby confirming the FSP1 pathway as GPX4-independent.[2] A G2A mutation of the FSP1 N-terminal myristylation site revealed that N-myristylation is essential in targeting FSP1 to the plasma membrane.[2,3] This mechanism of FSP1 activity involves the reduction of ubiquinone to ubiquinol, which targets lipid peroxyl radicals to suppress lipid peroxidation.[2,3] Various human cancer cell lines were treated with RSL3 alone or in combination with a potent FSP1 inhibitor, iFSP1, to evaluate their sensitivity to ferroptosis induction.[2] As expected, the targeting of both regulatory pathways was required to effectively induce ferroptosis.[2] These findings demonstrate that FSP1 is a potent suppressor of lipid peroxidation and ferroptosis, and can effectively protect against cell death in the absence of GPX4.[2] Moreover, the discovery of this pathway provides a highly promising target for inducing ferroptosis in cancer cells.[2]

Laura McGary, Bearne Lab

References:

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4. Stockwell BR, Friedmann Angeli JP, Bayir H, et al. Ferroptosis: A Regulated Cell Death Nexus Linking Metabolism, Redox Biology, and Disease. *Cell*. 2017;171(2):273–285.
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