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September 30th, 2025

Lung Cancer Canada
133 Richmond St West #208,
Toronto, ON
M5H 2L3

RE: Give a Breath Research Award Application for Dr. Kelsie Thu

Dear Review Committee,

Please find attached my application for Lung Cancer Canada's Give a Breath Research Award. I have uploaded the required documents and my CCV as instructed.

I am passionate about discovering new strategies for improving the care, management and survival of lung cancer patients. With this award, my laboratory will systematically characterize signaling proteins that are regulated by CD47 to drive lung cancer metastasis and the growth of metastatic lung tumor lesions for the first time. By identifying these proteins and their effects on lung cancer progression, our discoveries will provide novel insights into lung tumour biology and nominate specific proteins as targets that could be inhibited to reduce metastasis and the growth of metastatic lung cancer. Such discoveries will advance knowledge in the lung cancer field and could lead to new therapeutic strategies to improve the outcomes of lung cancer patients who often succumb to metastatic disease.

Importantly, my laboratory does not have funding to pursue this impactful project and funds are required for us to continue making progress towards developing anti-metastatic strategies to benefit lung cancer patients. This Lung Cancer Canada Give a Breath Research Award will allow us to generate exciting preliminary data required to: i) secure additional, longer-term funding to explore how CD47-dependent secretion of these signaling proteins affects the metastatic behavior of lung cancer cells; and ii) rigorously evaluate the therapeutic potential of these proteins as targets for novel anti-metastatic treatments for lung cancer patients. Our ultimate goal is to translate our findings into new clinical strategies for combatting metastasis and tumor progression in lung cancer patients to improve survival rates for this deadly disease.

Thank you very much for considering my application.

Sincerely,

Kelsie Thu, PhD
Assistant Professor, Laboratory Medicine and Pathobiology, University of Toronto
Scientist, Keenan Research Centre for Biomedical Science, St. Michael's Hospital

Rationale: *Strategies to combat metastatic lung cancer are desperately needed.* Nearly 50% of lung cancer (LC) patients are diagnosed with advanced disease when curative interventions are not feasible. Metastatic progression defines stage IV disease and is a major driver of poor LC outcomes. Despite therapeutic advances, the 5-year survival rate for Canadians with stage IV LC is a dismal 3%[1]. The incomplete understanding of mechanisms underlying LC metastasis and how they can be disrupted to combat metastatic disease is a significant bottleneck to improving patient outcomes. As such, elucidating these processes is critical for identifying novel strategies to intervene.

CD47 is a prognostic factor associated with LC metastasis. CD47 is a transmembrane protein canonically known as an anti-phagocytic, "don't eat me" signal[2]. Many cancers, including LC, exploit CD47 to prevent their engulfment by phagocytes like macrophages[2]. The oncology field has largely focused on CD47's immunosuppressive function due to its implications for immunotherapy but CD47 also has cell-intrinsic functions in specific physiological and pathological contexts[3,4]. These functions include positive regulation of prometastatic processes like cell adhesion, migration and invasion in smooth muscle, epithelial and immune cells, and cancer cell lines. CD47 is frequently upregulated in lung tumors and is associated with poor prognosis and metastasis in LC patients[2]. Although its role in facilitating immune evasion is well described, the cell-intrinsic mechanisms underlying associations between CD47 and cancer metastasis remain largely undefined. As described below, we recently found that CD47 positively regulates prometastatic phenotypes *in vitro* and LC metastasis *in vivo*[5] (*Lau et al, under review*). This suggests that targeting CD47 or its effector proteins could be a promising treatment strategy for combatting metastatic LC. For this project, we will identify secreted proteins regulated by CD47 and characterize their effects on prometastatic phenotypes in LC cells.

Preliminary Data: Motivated by the limited knowledge of CD47's roles beyond immune evasion in LC, we investigated cell-intrinsic functions of CD47 in mouse (LLC) and human (H1299) LC models that we engineered with CD47 knockout (KO; **Fig.1A-C**). To uncouple CD47's cell-intrinsic effects from its immunosuppressive effects, we evaluated growth and progression of tumors formed by orthotopic injection of LC cells with wildtype (WT) CD47 or CD47 KO in immune deficient mice lacking T, B, and NK cells and with reduced macrophage/dendritic cell function. This revealed that mice with CD47 KO tumors exhibited prolonged survival compared to those with WT tumors (**Fig.1D-E**)[6]. To model key aspects of metastasis (ie. survival in circulation and tissue engraftment[7]), we used intravenous (IV) tail-vein injection of LLC and H1299 cells. Using this system, we found that CD47 KO cells had significantly reduced abilities to seed tumors in the lungs, and that mice that received CD47-deficient cells had prolonged metastasis-free survival compared to mice injected with WT cells (**Fig.1F,G**).

To understand how CD47 promotes LC metastasis, we performed RNA-seq on WT and CD47 KO LLC cells. This identified that CD47 KO reduced the expression of genes enriched for signatures of epithelial-to-mesenchymal transition (EMT), MAPK signaling, and cytokine production (**Fig.2A**). Comparison of genes downregulated in CD47 KO cells and in TCGA lung tumors with low vs high CD47 expression identified 45 common genes including CXCL16, a chemokine known to promote cancer metastasis (**Fig.2B**)[8], indicating the clinical relevance of the downregulated genes identified. Functional studies validated our transcriptomic findings, as we found that CD47 positively regulates cell adhesion and migration, two EMT-related phenotypes[9], in an ERK-dependent manner (*Lau et al, under review, BioRxiv*). To investigate the putative role for CD47 in regulating cytokines, we incubated platelets, cells with established roles in promoting cancer metastasis[10], with cell-free supernatants from LLC and H1299 cells ± CD47 (**Fig.2D**). We observed that platelet activation was diminished upon incubation with supernatants from CD47 KO cells (**Fig.2E**), indicating that CD47 regulates secretion of factors that mediate communication with other cells. These factors could include cytokines or other soluble signaling proteins like CXCL16. Collectively, our findings implicate CD47 in driving LC metastatic progression through a cell-intrinsic mechanism, which likely includes secretion of proteins that mediate prometastatic cell communication. This suggests that secreted CD47 effector proteins could be targeted to inhibit metastatic progression, but first, they must be identified.

Hypothesis, Objectives, & Aims: We hypothesize that CD47 regulates secretion of specific signaling proteins that facilitate cell-cell crosstalk to promote LC metastasis (**Fig.2C**). We suspect that inhibiting or neutralizing such secreted CD47 effector proteins could be an effective strategy for limiting LC metastasis and progression of metastatic tumor nodules. The objectives of this study are to identify specific secreted proteins regulated by CD47 and to characterize their effects on prometastatic phenotypes in LC models. Achieving the following *Specific Aims* will provide insights necessary to inform follow-up studies aimed at defining the biological and therapeutic significance of the prometastatic signaling mechanisms identified.

Aim 1 - Identify secreted proteins regulated by CD47 in LC cells. We will profile the expression of secreted proteins in cell-free supernatants from 2 mouse (LLC, CMT167) and 3 human (H1299, PC9, X137) LC cell lines isogenic for CD47 (eg. WT/CD47 KO). We have already engineered CMT, PC9, and X137 with CD47 KO (not shown). Supernatants will be collected from subconfluent monolayers (~70-80% confluence) 72h after plating. Three independent replicates will be profiled for each genotype in each cell line. Multiple LC models of mouse and human origin will be profiled to ensure generalizability of our findings. Proteins will be profiled using the Olink Target 96 Mouse- Human Exploratory panel at the Network Biology Collaborative Centre/Sinai Health System. This high-throughput platform quantifies 96 secreted proteins including cytokines, growth factors, and other signaling proteins implicated in cancer biology. Comparative expression analyses will be done to identify proteins putatively regulated by CD47 across the LC models (ie. those with significantly lower expression in CD47 KO compared to WT cells). To our knowledge, this will be the first study to characterize CD47-mediated regulation of protein/cytokine secretion in any cell type.

Aim 2 - Characterize the effects of secreted proteins on metastatic phenotypes in LC cells. We will confirm the biological relevance of the top 3 secreted proteins we identify as putatively regulated by CD47. These proteins will be selected based on literature implicating their roles in metastasis, magnitude of downregulation in CD47 KO cells, recurrence across models, and reduced expression in TCGA tumors with low vs high CD47 expression. We will supplement the media of CD47 KO cells ± recombinant versions of the candidate secreted proteins and determine how reintroducing the proteins affects the metastatic nature of LC cells. We expect recombinant proteins to rescue defects in prometastatic phenotypes that we observed in CD47 KO cells if the secreted proteins are EMT/ metastasis-promoting effectors of CD47. We will conduct western blots for canonical EMT markers (eg. cadherins, vimentin), qPCR for EMT target genes (*TWIST, SNAIL, SLUG, CDH2, FN1, MMP2*), and cell adhesion/migration/invasion assays, as we have described (*Lau et al, BioRxiv*). Phenotypes will be statistically compared in CD47 KO cells treated ± recombinant proteins.

Significance: By understanding CD47's non-canonical role in regulating protein secretion to promote the metastatic behavior of LC cells, this project will advance knowledge of the mechanisms underlying LC progression. Our work will inform novel approaches for inhibiting LC metastasis and eradicating metastatic disease to improve patient outcomes. As such, our findings will provide foundational insights to guide additional studies in preclinical models to evaluate the therapeutic potential of inhibiting secreted CD47 effector proteins as a strategy to combat LC metastasis.

Future Directions: The prometastatic secreted proteins we find to be regulated by CD47 could be developed into predictive biomarkers and/or targets for therapies to antagonize metastatic progression, both of which could be leveraged to improve outcomes for patients with early and advanced stage LC in Canada and worldwide. To achieve this translational goal, our future studies will: i) determine the effects of secreted CD47 effector proteins on LC metastasis *in vivo*, ii) engage clinician scientists to measure CD47-regulated secreted proteins in liquid biopsies from LC patients pre/post tumor progression to assess their utility for predicting metastasis, and iii) test the anti-metastatic therapeutic effects of inhibiting candidate secreted proteins in preclinical lung tumor models.

References:

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2. Lau, A. P. Y., Khavkine Binstock, S. S. & Thu, K. L. CD47: The Next Frontier in Immune Checkpoint Blockade for Non-Small Cell Lung Cancer. *Cancers* **15**, 5229 (2023).
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4. Polara, R., Ganesan, R., Pitson, S. M. & Robinson, N. Cell autonomous functions of CD47 in regulating cellular plasticity and metabolic plasticity. *Cell Death Differ.* (2024) doi:10.1038/s41418-024-01347-w.
5. Lau, A. P. *et al.* P3.02F.01 CD47 promotes migration and metastasis in non-small cell lung cancer. *J. Thorac. Oncol.* **19**, S301 (2024).
6. Lau, A. P., Kubli, S. P., Wakeham, A., Mak, T. W. & Thu, K. L. Abstract 5182: CD47 is a promising therapeutic target in non-small cell lung cancer. *Cancer Res.* **83**, 5182–5182 (2023).
7. Hebert, J. D., Neal, J. W. & Winslow, M. M. Dissecting metastasis using preclinical models and methods. *Nat. Rev. Cancer* (2023) doi:10.1038/s41568-023-00568-4.
8. Korbecki, J. *et al.* The role of CXCL16 in the pathogenesis of cancer and other diseases. *Int. J. Mol. Sci.* **22**, 3490 (2021).
9. Allgayer, H. *et al.* Epithelial-to-mesenchymal transition (EMT) and cancer metastasis: the status quo of methods and experimental models 2025. *Mol. Cancer* **24**, 167 (2025).
10. Li, S. *et al.* The dynamic role of platelets in cancer progression and their therapeutic implications. *Nat. Rev. Cancer* **24**, 72–87 (2024).

FIGURES

Figure 1

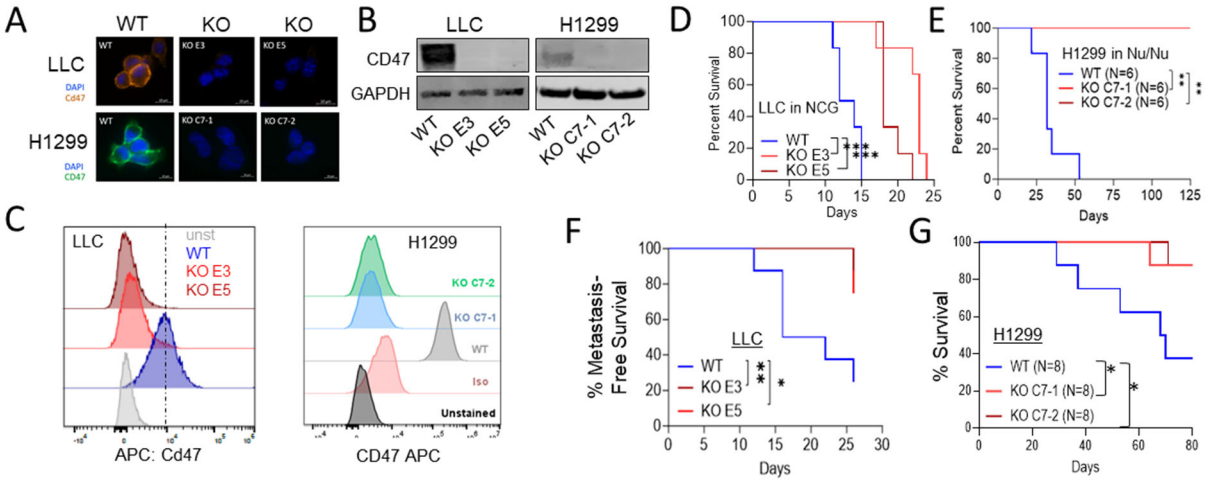

Fig.1. CD47 promotes lung tumor progression and metastasis. A,B,C) Confirmation of *CD47* KO in murine LLC and human H1299 cells by immunofluorescence (A), immunoblotting (B), and flow cytometry (C). D) Survival of immune deficient NCG mice with orthotopic WT and *CD47*$^{-/-}$ LLC tumors (N=6-7/group). E) Survival of immune deficient Nu/Nu mice with subcutaneous WT and *CD47*$^{-/-}$ H1299 tumors (N=6/group). Survival curves were analyzed using log-rank tests. F) Metastasis-free survival of C57BL/6 mice after tail vein injection of WT or CD47-deficient LLC cells. Metastasis-free survival was determined using bioluminescence imaging to detect seeding and outgrowth of luciferase-tagged lung cancer cells in the lungs. Tail-vein injection was used to model cancer cell survival in circulation and extravasation at the distant site, two critical components of the metastatic cascade. G) Overall survival of Nu/Nu mice that received tail vein injections of WT or CD47 KO H1299 cells. For all figures, *p<0.05, **p<0.01, ***p<0.001, ***p<0.0001.

Figure 2

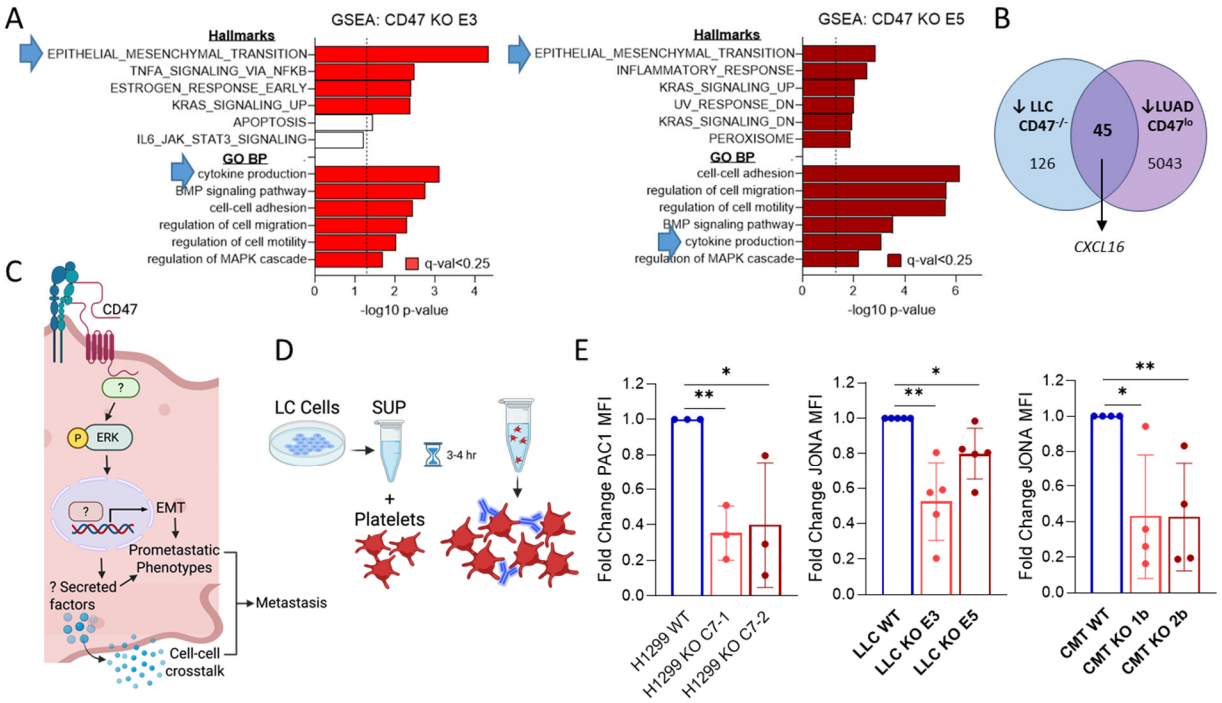

Fig.2. CD47 regulates secretion of proteins that influence cell behavior. A) GSEA results for LLC CD47 KO clones relative to LLC CD47 WT cells. Blue arrows indicate that gene expression signatures for EMT and cytokine production pathways are downregulated in CD47 KO cells, suggesting these pathways are positively regulated by CD47. B) Candidate CD47 effector genes downregulated in LLC CD47 KO relative to WT cells (blue circle) and TCGA LUAD tumors with low CD47 expression (DESeq2 q<0.25; purple circle). Venn diagram indicates genes commonly downregulated in lung cancers with low CD47 expression, including the cytokine CXCL16 which is known to promote cancer metastasis. C) Putative prometastatic signaling program regulated by CD47 in lung cancer, which involves secretion of proteins that mediate metastasis-promoting cell-cell communication. D) Schematic of the platelet activation assay. Supernatants were collected from WT lung cancer cells or cells with CD47 KO. Platelets were incubated with the supernatants and platelet activation markers were assessed by flow cytometry. E) Flow cytometry results for platelet activation markers, PAC1 (human) and JON/A (mouse) after incubation with cell-free supernatants from WT vs CD47$^{-/-}$ H1299/LLC/CMT cells. Fold-change in mean fluorescence intensity of the markers for KO vs WT supernatants (one-sample t-test). Reduced expression of activation markers on platelets treated with supernatants from CD47-deficient lung cancer cells indicates that CD47 regulates secretion of factors that influence cell behavior.

IMPACT STATEMENT

The biological mechanisms underlying metastatic progression of lung tumors are incompletely understood. This knowledge gap represents a significant barrier to improving care for advanced stage lung cancer patients who inevitably succumb to metastatic disease. Therefore, a better understanding of the mechanisms driving lung tumor metastasis and growth of metastatic lesions is imperative for informing new treatment strategies to combat them. This Give a Breath Research Award will allow us to identify specific signaling proteins secreted from lung cancer cells in a CD47-dependent manner and to determine whether they promote metastasis for the first time. Thus, our findings have great potential to identify CD47 effector proteins that could be targeted to combat metastatic progression in lung cancer patients. As such, discoveries from this project could eventually be translated into new treatments to antagonize metastatic disease. The work we have proposed for this Give a Breath Research Award will have an impactful and sustained influence on the lung cancer research field in several ways, as described below.

Immediate: Our findings will advance the current understanding of mechanisms that promote lung tumor growth and progression, and specifically, metastatic dissemination and outgrowth of lung cancers. Our research will define novel mechanisms underlying CD47's non-canonical role in driving cancer metastasis, thereby revealing putative targets for anti-metastatic therapies, which are desperately needed to improve lung cancer patient outcomes. Collectively, our discoveries will advance knowledge in the field and raise enthusiasm for targeting signaling effector proteins downstream of CD47 to combat metastasis and tumor progression.

Short & Medium Term (1-5 years): The results from this project will also have a significant impact by stimulating new avenues of research to investigate the biological and clinical relevance of the CD47-regulated signaling proteins identified. Dissemination of our findings to local, national, and international lung cancer communities through conferences and publications will invigorate interest in this area, ensuring that new discoveries continue to be made by our team and others. We will leverage the findings from our work to secure additional funds to support more comprehensive studies over the next several years (ie. long-term) to further define how the signaling proteins identified drive lung tumor metastasis and to explore their potential as targets for anti-metastatic therapies in preclinical models. This sustained research will include focused follow-up studies investigating specific CD47-regulated signaling proteins deemed to have the greatest therapeutic potential based on our Give a Breath findings. We will: i) decipher the mechanism and validate the biological significance of putative signaling proteins in mediating metastasis in additional lung cancer models; ii) determine whether signaling protein inhibition blocks metastasis and metastatic tumor growth; and iii) assess the tolerability of drugs or biologics targeting candidate proteins in model systems.

Long Term (> 5 years): If our follow-up studies confirm that CD47-regulated signaling proteins strongly influence metastatic progression and that targeting them is well-tolerated in model systems, our initial discoveries could stimulate clinical trials to test whether inhibiting the signaling proteins identified has therapeutic benefits in lung cancer patients. Translation of such

treatments would provide a much-needed therapeutic strategy to combat advanced stage lung cancer, which would improve quality of life, patient prognosis, and overall survival rates.

In summary, this Give a Breath project will yield novel discoveries that significantly advance our understanding of the biology underlying lung cancer metastasis, stimulating further research to investigate CD47-driven signaling proteins as targets for anti-metastatic therapies. Ultimately, the knowledge we generate could lead to the development of new therapies to antagonize the metastatic process and the growth of metastatic nodules in lung cancer patients with advanced stage disease. By informing new therapeutic strategies, our work will contribute to improving the outcomes of lung cancer patients and reducing the burden of lung cancer in Canada and worldwide.

LAY SUMMARY

Lung cancer causes more Canadian deaths than any other cancer type. One reason that lung cancer is so deadly is that it is diagnosed at advanced stages when the cancer has spread to other sites in the body. The spread of cancer cells from the original tumor site to other parts of the body is known as metastasis. Metastasis often results in organ failure and, ultimately, patient demise. This emphasizes the need for research to better understand the biological mechanisms enabling cancer cell metastasis and strategies to block them to improve lung cancer patient outcomes.

Our recent work identified that a protein called CD47 helps lung cancer cells change their shape and behavior through a process called epithelial-to-mesenchymal transition (EMT). EMT causes cancer cells to adopt a metastatic behavior. Accordingly, we found that deletion of CD47 in lung cancer cells reduces their ability to metastasize and limits growth and progression of lung tumors. Moreover, we found evidence that CD47 directs lung cancer cells to produce and release signaling molecules that influence the behavior of cancer and other cells known to promote tumor growth and metastasis. Based on these observations, we believe that the signaling proteins regulated by CD47 represent promising targets whose inhibition could limit metastasis and reduce the growth of metastatic tumor nodules. However, we first need to identify the specific proteins that CD47 regulates and characterize their effects on lung cancer cell behavior and metastasis in order to develop effective targeted therapies to block them in patients.

For this project, we will conduct studies to identify the signaling proteins regulated by CD47 to promote lung cancer metastasis and metastatic tumor growth. We will use a high-throughput screening approach to profile proteins that are secreted by cultured lung cancer cells. This will reveal candidate proteins that CD47 controls to facilitate metastatic progression. We will then investigate how these proteins influence biological processes related to metastasis and their effects on lung tumor growth and metastasis in preclinical models. In summary, our work will identify specific proteins that contribute to lung tumor metastasis and strategies to inhibit them. Ultimately, discoveries from this project could inform new treatment approaches to minimize the lethal effects of metastasis in lung cancer patients.

Budget Justification

Component	Description	Cost
Personnel	0.10 FTE PhD (this project will require 10% of the student's time for 12 months)	$4,200
Research Supplies	General reagents/consumables for growing lung cancer cells, collecting samples, and conducting functional experiments	$4,800
Olink High-Throughput Screening for Secreted Proteins by the Network Biology Collaborative Centre at the Sinai Health System	Olink Target 96 Mouse/Human Exploratory Reagent Kit = $10,540 Olink 96.96 IFC for Protein Expression = $1,705 Shipping and Handling = $394 Screening service = $2,361	$15,000
Unity Health Toronto Core Facility Services	Use of bioimaging and genomics cores (eg. microscopy analysis of metastatic phenotypes, immunofluorescence and qPCR to assess signaling pathways)	$1,000
	Total	**$25,000**

Personnel costs
One PhD student in Dr. Thu's lab will dedicate 10% of their research efforts to this project (0.1 FTE). The student is a senior PhD candidate who has led the Thu Lab's project investigating CD47's role in lung cancer metastasis. The student will grow lung cancer cells, harvest samples for Olink profiling, and conduct the functional experiments proposed. The annual stipend for a PhD student in the Laboratory Medicine and Pathobiology Department at the University of Toronto is $42,000/year. As such, funding requested to support the salary for this 0.1 FTE PhD student is $4,200.
$4 200 is requested to cover personnel.

Research supplies
Plasticware, growth media, supplements (eg. FBS, antibiotics), antibodies for western blots and immunofluorescence assays, qPCR primers and master mix, and recombinant proteins (eg. cytokines) required for culturing lung cancer cells and conducting experiments to test the functional relevance of signaling proteins identified by Olink profiling.
$4 800 is requested to cover the costs of laboratory supplies.

Olink protein screening services
Identification of secreted proteins regulated by CD47 in lung cancer cells will be discovered using the Olink High-Throughput protein screening platform. This service will be provided by the Network Biology Collaborative Centre at the Sinai Health System. The funds requested will cover the costs of the reagent kit, sample quality control and quantitation, hands-on labour for protein screening, and data analysis.
$15 000 is requested to cover the costs of Olink protein profiling.

Core facility fees:
This project will require the use of instruments in the genomics and bioimaging cores at the Keenan Research Centre for Biomedical Sciences of Unity Health Toronto to perform the microscopy and qPCR experiments proposed. We estimate the need for ~25 hours of core facilities use at $40/hour.
$1 000 is requested to support the use of these core facilities.

Grant Total: $25,000

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Ori D. Rotstein, MD, FRCSC, FACS
Professor and Associate Chair of Surgery,
University of Toronto
Vice President, Research & Innovation
Unity Health Toronto

September 30, 2025

Lung Cancer Canada
133 Richmond St. W., Suite 208
Toronto, ON
M5H 2L3

RE: Dr. Kelsie Thu, Give a Breath Research Awards Application

Dear Lung Cancer Canada Adjudication Committee,

As Vice President, Research & Innovation at Unity Health Toronto (Unity Health), I am delighted to provide this letter of support for Dr. Kelsie Thu's Lung Cancer Canada Give a Breath Research Award application, entitled "Defining non-canonical functions of CD47 in lung cancer metastasis and tumor progression".

Dr. Thu's independent research career began in July 2020, when she was appointed as a Scientist within Unity Health's Keenan Research Centre for Biomedical Science (KRCBS), and Assistant Professor in the University of Toronto's Department of Laboratory Medicine and Pathobiology. During her first 5 years in this position, Dr. Thu has built an exciting program of research focused on understanding biological mechanisms driving lung cancer growth and progression and therapeutic strategies to target them, which has attracted support from the Canadian Institutes of Health Research as well as highly qualified trainees.

Dr. Thu's recent research has advanced the oncology field's understanding of how the cell surface protein, CD47, promotes lung tumor growth and progression. Distinct from others who have focused on CD47's role in enabling tumors to escape destruction by the immune system, Dr. Thu has been studying its role in different tumor-promoting processes. Excitingly, she has discovered that CD47 drives lung cancer metastasis. She has also discovered that CD47 regulates specific molecular programs that cause lung cancer cells to produce and release signaling molecules that influence the behavior of other cells known to promote tumor growth and metastasis. Dr. Thu suspects that lung tumors use this function of CD47 to drive lung cancer metastasis and promote the growth of metastatic lesions, and that blocking the signaling molecules controlled by CD47 represents a promising treatment strategy to reduce the burden of metastasis in lung cancer patients. Dr. Thu's Give a Breath award proposal seeks to identify the specific signaling molecules released from lung cancer cells, as directed by CD47. Characterization of these molecules and how they affect the biological process required for lung cancer metastasis and metastatic tumor growth will inform therapeutic strategies to block them. Dr. Thu will leverage the knowledge and evidence gained from this project to secure further support to determine how CD47 controls these molecules, how they contribute to metastatic processes in lung cancer cells, and whether inhibition of these molecules represents a promising approach to reduce metastasis and metastatic tumor growth in lung cancer patients.

Unity Health is strongly committed to support for Dr. Thu's research, including 100% protected time dedicated to research activities. Unity Health provided start-up funds for Dr. Thu to establish her laboratory, which is fully operational and equipped with all of the infrastructure required to conduct the proposed research activities. Dr. Thu also has access to the KRCBS Research Core Facilities (RCF). Among these are the Genomics and Bioimaging Cores that house state-of-the-art infrastructure to support the experiments she has proposed.

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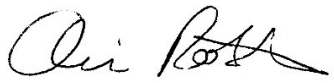
Dr. Thu is a promising early career scientist whose research generates new knowledge about lung cancer at the cellular level, and applies it to develop new treatment strategies for people with lung cancer. I strongly endorse her application for the Give a Breath Research Award and assure you that Unity Health will continue to support Dr. Thu's exciting research and its immense potential to reduce the burden of lung cancer and optimize patient care.

Sincerely,



Ori D. Rotstein, MD, FRCSC, FACS
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