BIOGRAPHICAL SKETCH

NAME: **Lisa M. Coussens**

eRA COMMONS USER NAME (credential, e.g., agency login): **coussens**

POSITION TITLE: **Professor, Chairwoman, and Deputy Director**

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
San Francisco State University, San Francisco	B.A.	05/1980	Marine Biology
University of California, Los Angeles	Ph.D.	08/1993	Biological Chemistry
University of California, San Francisco	Postdoctoral	12/1997	Tumor Biology

A. PERSONAL STATEMENT: For decades, it was generally accepted that leukocytes in tumors represented a failed attempt of the immune system to eradicate malignant cells. Some aspects of that tenet are true. However, my research investigating functional significance of inflammation in cancer resulted in a paradigm shift by revealing that in vivo, activation of chronic inflammatory programs in early neoplasms is required for *promotion* to malignancy. As such, my lab focused on elucidating immunologic, molecular and cellular underpinnings of pro-tumor immunity, where we discovered previously unappreciated roles for immune cells as critical regulators of solid tumor development. Using transgenic mouse models, we identified and molecularly deciphered tissue-specific roles for B and $CD4^+$ T lymphocytes in driving protumorigenic T_H2 activities of myeloid cells in breast, pancreas, and cutaneous carcinomas. By identifying critical mediators of these tissue-specific paracrine interactions, we uncovered previously unappreciated targets for anti-cancer therapy, that when inhibited, neutralize protumoral chronic inflammation and unleash $CD8^+$ T cell cytotoxicity to slow tumor progression.

We evaluated the translational significance of our preclinical findings in proof-of-concept clinic trials in patients with solid tumors. Based on our preclinical studies identifying Brutons Tyrosine Kinase (BTK) as a mediator of B cell and macrophage-mediated T cell suppression (*Andreu et al., 2010*; *Affara et al., 2014; Gunderson et al., 2016*), Drs. Lopez (OHSU) and Tempero (UCSF) conducted an investigator-initiated *SU2C-Lusgarten Pancreas Cancer Immune Therapy* funded trials (NCT02562898 & NCT02436668) examining immune responses to a BTK inhibitor (BTKi) in patients with metastatic pancreatic adenocarcinoma (*Tempero et al., 2021; Sinha et al., in press*). In a related industry-sponsored phase II trial (NCT02454179), Dr. Taylor (OHSU) evaluated a BTKi plus pembroluzimab in advanced head & neck squamous carcinoma (*Taylor et al., 2022*). And, based on our findings in murine mammary carcinoma models identifying macrophage CSF1/CSF1R signaling pathways mediate $CD8^+$ T cell suppression (*DeNardo et al., 2009, 2011*), we conducted a phase Ib/II investigator-initiated trial supported by a KOMEN Promise award (PIs: Coussens, Hwang, Rugo), evaluating Pexidartinib, a potent CSF1R kinase small molecule inhibitor, plus eribulin in patients with metastatic triple negative breast cancer (NCT01596751). Results from these trials revealed, as predicted by the preclinical studies, that inhibition of BTK or CSF1R results in reprograming of immune microenvironments, blunting myeloid-mediated T cell suppression and fostering anti-tumor type immune signatures both within tumors and systemically in peripheral blood. My laboratory, while still firmly rooted in discovery-based science utilizing murine models of human cancer, has played a critical role in these clinical trials utilizing innovative technology platforms we developed (*Ruffell et al., 2012; Tsujikawa et al., 2017; Banik et al., 2019*) to evaluate longitudinal biopsies and peripheral blood to audit immune contexture (*Reddy et al., 2019; Means et al., 2019; Casetta et al., 2019; Liudahl et al., 2021; Kitko et al 2022)*, and ultimately identify immune-based predictive biomarkers for patient stratification (*Hassan et al., 2019; Tsujikawa et al., 2020; Byrne et al., 2021, Labrie et al., 2021; Yoshimura et al., 2021; Taylor et al., 2022; Blise et al., 2022; Johnson et al., 2022; Mi et al 2022*), and further hypothesis testing.

I have been involved in intra- and extramural research for >20 years with track record as a reliable collaborator, mentor, and leader in cancer tumor immunobiology. I have directly mentored ~40 postdoctoral/medical fellows, and 14 graduate students, most of which have gone on to develop successful academic careers. I was recently elected President for the American Association for Cancer Research (AACR; 2022-23), served on the AACR Board of Directors (elected, 2008-11), AACR Nominating Committee (elected, 2016-19), AAAS Nominating committee (2014-20), numerous National Cancer Institute committees, have previous service as Deputy and Senior editor for the journal *Cancer Research,* the board of reviewing editors for *Science*, and am currently on editorial boards for *Cancer Immunology Research, Cancer Cell* and *Cancer Discovery*. I currently serve on External Advisory Boards of four NCI-designated Cancer Centers (Koch Inst, UCSD Moores, DF/HCC, Jackson), multiple biotechnology companies and philanthropic foundations. I have organized ~20 national/international

meetings, e.g., CELL, Keystone, AACR, short Courses for Jackson Laboratories, all focused on aspects of *Inflammation and Cancer*. I was recruited to OHSU as Chair of the Cell, Developmental & Cancer Biology Department, and Associate Director for Basic Science in the Knight Cancer Institute, where I now serve as Deputy Director for Basic and Translational Research.

B. Positions and Scientific Appointments

2011-present Hildegard Lamfrom Endowed Chair in Basic Science, OHSU.
2022-present Deputy Director for Basic and Translational Research, Knight Cancer Institute, OHSU.
2011-2022 Associate Director for Basic Research, Knight Cancer Institute, OHSU.
2011-present Professor (with tenure) and Chairwoman: Dept. of Cell, Developmental & Cancer Biology, OHSU.
2012-2018 Co-Leader, Program in *Cancer Biology*, Knight Cancer Institute, OHSU.
2012-2014 Adjunct Professor, Dept. of Pathology, UCSF.
2009-2011 Co-Leader, Program in *Cancer, Immunity and Microenvironment*, Helen Diller Comprehensive Cancer Center, UCSF.
2007-2011 Professor (with tenure): Dept. of Pathology and Helen Diller Comp, Cancer Center, UCSF.
2006-2007 Assoc. Professor (with tenure): Dept. of Pathology and Cancer Research Institute, UCSF.
2004-2006 Assoc. Professor, In-Residence: Cancer Research Inst. & Dept. of Pathology, UCSF.
2001-2012 Co-Director, *Mouse Pathology Core*: Helen Diller Comprehensive Cancer Center, UCSF.
1999-2004 Asst. Professor, In-Residence: Cancer Research Inst. & Dept. of Pathology, UCSF.
1997-1999 Assistant Research Biochemist: Hormone Research Institute, UCSF.
1993-1997 Postdoctoral Fellow: Hormone Res. Inst., Univ. of Calif., San Francisco (UCSF).
1988-1993 Graduate Student: Dept. of Biological Chemistry, Univ. of Calif., Los Angeles, Los Angeles CA.
1981-1988 Research Associate: Dept. of Molecular Biology. Genentech, Inc., S. San Francisco, CA.

Honors, Distinctions & Notable Awards

2022 Fellow of the Academy of Immuno-Oncology, Society of Immunotherapy of Cancer (SITC)
2022-23 President, American Association for Cancer Research (AACR)
2020-2024 Komen Scholar, Susan G. Komen Foundation
2019 Fellow of the AACR Academy (American Association for Cancer Research)
2019-22 *Highly Cited Researcher*, Web of Science
2018 AAAS Fellow, American Association for Advancement of Science
2019-20 Endowed Professor, TEFAF Oncology Alternating Chair, Maastricht University, The Netherlands
2018 Career Award, European Academy of Tumor Immunology
2018 Susan G. Komen Brinker Award for Scientific Distinction in Basic Science
2018 12th AACR-Princess Takamatsu Memorial Lectureship
2017 Doctor in Medicine (honoris causa), University of Buenos Aires, Argentina
2015 13th Rosalind E. Franklin Award (NIH, National Cancer Institute)
2012 Charlotte Friend Memorial Lectureship (American Association for Cancer Research)
2011-2016 Susan G. Komen for the Cure, PROMISE award
2011-2016 Era of Hope Scholar Expansion Award, Dept. of Defense Breast Cancer Research Program
2006-2011 Era of Hope Scholar Award, Dept. of Defense Breast Cancer Research Program
2001-2002 Gertrude B. Elion Cancer Research Award (Am. Association for Cancer Research)
2000-2002 Hellman Family Award for Early Career Faculty
2000-2003 Edward Mallinckrodt, Jr. Foundation Award for Medical Research
2000-2002 V Foundation Scholar (V Foundation for Cancer Research)
1985, 86, 88 Recognition Award, Genentech Inc.

NIH/NCI and External/Scientific Advisory or Review Board (EAB/SAB/SRB) Service *(current):*

Cell Signaling Tech. (Immunology SAB, 2021-present); HiberCell, Inc., (SAB, 2020-present); Kineta, Inc. (SAB, 2020-present); Lustgarten Fndtn, Therapeutics Working Group (member, 2019-present); CytomX Therapeutics, Inc. (SAB, 2019-present); Dana Farber/Harvard Cancer Center (P30 EAB, 2019-2025); Zymeworks, Inc. (SAB, 2019-2021); Univ. of California, San Diego Moores Cancer Center (P30 EAB, 2019-present); Verseau Therapeutics, Inc. (SAB, 2018-2022); Carisma Therapeutics Inc. (SAB, 2018-present); Breast SPORE, Dana Farber Cancer Center (P50 EAB, 2017-present); Syndax Pharmaceutical, Inc. (EAB, 2016-present); Bloomberg-Kimmel Inst. for Cancer Immunotherapy, Sidney Kimmel Comp. Cancer Center at Johns Hopkins (EAB, 2016-2022); NIH/NCI-Frederick Natl Laboratory Advisory Committee (EAB, 2016-2024); V Fndtn for Cancer Research (SRB, 2013-2021); Cancer Research Inst. (SAB, 2013-present); Koch Inst. for Integrated Cancer Research, MIT

(P30 EAB, 2012-present); STARR Cancer Consortium (SRB, 2011-2021); The Jackson Laboratory Cancer Center (P30 EAB, 2021-present); Alkermes, Inc. (SAB, 2021-present); Prostate P01, Columbia University Medical Center (P01 EAB, 2020-present); MDACC GI SPORE (P50 EAB, 2022-present); Pio Therapeutics Pty Ltd (EAB, 2022-present); PDX Pharmaceuticals, Inc. (2022–present); NextCure (2022-present)

***Consulting Activities** (current)*: Shasqi Inc. (2020-present); AbbVie Inc (2020-present); Cell Signaling Tech. (2017-2021)

C. CONTRIBUTION TO SCIENCE (Scopus: *56,570 citations, h-index 82*; Google Scholar: *83,436 citations, h-index 93, i10-index 172*)

1. *Receptor tyrosine kinases*: My passion for science and cancer research was ignited while working with Axel Ullrich at Genentech, Inc. In the days of Southern blots and Maxim-Gilbert sequencing, our team successfully cloned and characterized many of the initial growth factor receptors, and in so doing, recognized their associated activities as cellular (proto)oncogenes. Collectively, this body of research revolutionized understanding of cellular growth control and set the stage for subsequent development of therapeutic antibody- and RTK-targeted therapeutics now routinely deployed in the clinic.

- Ullrich A, **Coussens L**, Hayflick J, Dull T, Gray A, Tam A, Lee J, Yarden Y, Libermann T, Schlessinger J, Downward J, Bye J, Whittle N, Waterfield M, Seeburg P. (1984) Human epidermal growth factor receptor cDNA sequence and aberrant expression of the amplified gene in A431 epidermoid carcinoma cells. ***Nature*** 309:418-425.
- **Coussens L**, Yang-Feng T, Liao T-C, Chen E, Gray A, McGrath J, Seeburg P, Libermann T, Schlessinger J, Francke U, Levinson A, Ullrich A. (1985) Tyrosine kinase receptor with extensive homology to the EGF receptor shares chromosomal location with *neu* oncogene. ***Science*** 230:1132-1139.
- **Coussens L**, Van Beveren C, Smith D, Chen E, Mitchell R, Isacke C, Verma I, Ullrich A. (1986) Structural alteration of viral homologue of receptor proto-oncogene *fms* at carboxyl terminus. ***Nature*** 320:277-280.
- **Coussens L**, Parker P, Rhee L, Yang-Feng T, Chen E, Waterfield M, Francke U, Ullrich A. (1986) Multiple, distinct forms of bovine and human protein kinase C suggest diversity in cellular signaling pathways. ***Science*** 233:859-866.

2. *Inflammation drives solid tumor development*: Solid tumors have long been studied from tumor cell-intrinsic points of view; indeed, mutations of significant genes in would-be tumor cells drive cancer development. That said, the microenvironment in which early neoplastic cells evolve also critically regulates all aspects of cancer progression and is now recognized as a characteristic hallmark of cancer (*Hanahan and Coussens, 2012*). In early studies employing transgenic mouse models of squamous carcinogenesis, we defined roles for immune cell-derived mediators in regulating oncogene-driven cancer development; in a seminal study, we revealed that a previously unappreciated myeloid cell type, e.g., mast cells, potentiated squamous carcinogenesis by providing proteases (matrix metalloproteinase 9) and angiogenic factors necessary for early pre-cancer development (*Coussens et al., 1999; Coussens et al., 2000*). Together, these manuscripts yielded a paradigm shift by revealing that pre-cancer development involves a tissue response whereby activation of chronic inflammatory pathways promotes cancer development. Based on these findings, I co-authored a seminal review that remains one of the most highly cited *Nature* reviews to date (*Coussens and Werb, 2002*).

- **Coussens LM**, Raymond WW, Bergers G, Laig-Webster M, Behrendtsen O, Werb Z, Caughey, GH, Hanahan D. (1999) Inflammatory mast cells upregulate angiogenesis during squamous epithelial carcinogenesis. ***Genes & Development*** 13:1382-1397.
- **Coussens LM**, Tinkle CL, Hanahan DH, Werb Z. (2000) MMP-9 supplied by bone marrow-derived cells contributes to skin carcinogenesis. ***Cell*** 103:481-490.
- **Coussens LM**, Werb Z. (2002) Inflammation and cancer. ***Nature*** 420: 860-867.
- Hanahan D, **Coussens LM**. (2012) Accessories to the crime: Functions of cells Recruited to the tumor microenvironment, ***Cancer Cell*** 21: 309-322.

3. *B cells, humoral immunity and complement regulate solid tumor development*: With a mouse model of HPV16-driven carcinogenesis (*Coussens et al., 1996*), we sought to understand critical roles of adaptive immune cells on pre-cancerous cutaneous neoplasms. As such, we were first to appreciate that B cells foster tumor development by activating protumor activity of diverse myeloid cells infiltrating premalignant tissues (*deVisser et al., 2005; Schioppa et al., 2011*). Using a series of in vivo molecular genetic, biochemical and immunologic approaches, we revealed that humoral immunity potentiates cutaneous carcinogenesis via induction of Th2-type inflammatory pathways in myeloid cells following activation of FcRγ-mediated signaling (*Andreau et al., 2010*). That B cells, humoral immunity and FcγR regulate solid tumorigenesis remains a novel concept that has challenged existing paradigms in both the cancer biology and tumor immunology fields. Based on these studies, we examined B cell (CD20)-depleting antibodies as therapeutics in solid tumors and reported that B

cell depletion enhances therapeutic efficacy of chemotherapy by $CD8^+$ T cell-dependent mechanisms (*Affara et al., 2014).* By revealing similar "B cell signatures" in squamous, pancreas and head and neck (HNSCC) cancers, we identified a protumoral role for B cells in human pancreatic cancers (*Gunderson et al., 2016,*) and HNSCC. These preclinical findings and human correlations provided rationale to evaluate Bruton's Tyrosine Kinase (BTK) inhibitors in combination with gemcitabine and nab-paclitaxel in patients with locally advanced pancreatic adenocarcinomas (NCT02436668; *Tempero et al, 2021*), and in combination with PD-1 immune checkpoint blockade in patients with advanced HNSCC (NCT02454179; *Taylor et al., 2022*). More recently, we extended these studies to reveal activation of complement protein C5a and its receptor expressed on diverse myeloid subtypes, activate protumoral effector pathways, leading to macrophage-based T cell suppression; inhibition of C5aR signaling blunts these and fosters antigen-specific $CD8^+$ T cell responses that are synergistic with chemotherapy (*Medler et al, 2018*).

- Andreu P, Johansson M, Affara NI, Tan TT, Junankar S, Korets L, Lam J, Tawfik D, Pucci F, De Palma M, DeNardo D, de Visser KE, **Coussens LM**. (2010) FcRγ activation regulates inflammation-associated squamous carcinogenesis. ***Cancer Cell*** 17(2):121-134.
- Affara NI, Ruffell B, Medler TR, Gunderson AJ, Johansson M, Bergsland E, Steinhoff M, Li Y, Gong Q, Ma Y, Wiesen JF, Kulesz-Martin M, Irving B, **Coussens LM**. (2014) B cells regulate macrophage phenotype and response to chemotherapy in squamous carcinomas. ***Cancer Cell*** 25(6): 809-821.
- Gunderson AJ, Kaneda M, Tsujikawa T, Affara NI, Nguyen, Ruffell B, Gorjestani S, Liudahl SM, Truitt M, Olson P, Kim G, Hanahan D, Tempero M, Sheppard B, Irving B, Varner JA, **Coussens LM**. (2016) Bruton's tyrosine kinase (BTK)-dependent immune cell crosstalk drives pancreas cancer. ***Cancer Discovery***, 6(3): 270-285.
- Medler TR, Murugan D, Horton W, Kumar S, Cotechini T, Forsyth AM, Leyshock P, Leitenberger JJ, Kulesz-Martin M, Margolin A, Werb Z, **Coussens LM**. (2018) Complement C5a fosters squamous carcinogenesis and limits T cell response to chemotherapy, ***Cancer Cell,*** 34:561-578.

4. *T_H2 innate and adaptive leukocytes regulate mammary carcinogenesis and pulmonary metastasis*: Whereas we had appreciated that macrophages played a key role in regulating mammary cancer metastasis (Lin and Pollard, 2001, 2002), it was unclear how macrophages were "programmed" to acquire a protumorigenic phenotype. Based on our studies in squamous cancers demonstrating myeloid cells were "programmed" by adaptive lymphocytes, we investigated the hypothesis that protumoral macrophages in mammary carcinomas were similarly regulated by lymphocyte-derived paracrine factors. Using transgenic mouse models, we revealed the interleukins (IL)-4 and 13, derived from Th2-$CD4^+$ T cells induce protumor activities of monocytes and macrophages, that in turn potentiate late-stage cancer development and pulmonary metastasis (*DeNardo et al., 2009*). With the clinical potential of targeting macrophages via CSF1/CSF1R-blockade, we evaluated small molecule CSF1R antagonists and were first to report that clinical compounds targeting this macrophage survival pathway disrupt late-stage tumor progression and enhance chemo-sensitivity (*DeNardo et al., 2011; Strachan et al., 2013*) and radiation therapy sensitivity (*Shiao et al., 2015*) by $CD8^+$ T cell-dependent mechanisms. Together, these studies provided supporting data for an investigator-initiated phase Ib/II clinical trial evaluating a CSF1R inhibitor with chemotherapy in women with metastatic triple negative breast cancer (NCT01596751) funded by a Komen award (Coussens: coPI). These studies have led to identification of a rare population of intratumoral $TIM3^+$ dendritic cells, whose maturation and production of IL-12 is repressed by macrophage-derived IL-10 in mammary tumors; these are critical for cross presentation and activity of intratumoral $CD8^+$ T cells, and importantly provide an immune-based signature for identifying patients likely to respond to immune therapy (*Ruffell et al., 2014; de Mingo Pulido et al., 2018; Casetta et al., 2019*).

- DeNardo DG, Baretto JB, Andreu P, Vasquez L, Kolhatkar N, **Coussens LM**. (2009) $CD4^+$ T cells regulate pulmonary metastasis of mammary carcinomas by enhancing protumor properties of macrophages. ***Cancer Cell*** 16:91-102.
- DeNardo DG, Brennan DJ, Rexhapaj E, Ruffell B, Shiao SL, Madden SF, Gallagher WM, Wadhwani N, Keil SD, Junaid SA, Rugo HS, Hwang ES, Jirstrom K, West BL, **Coussens LM**. (2011) Leukocyte complexity predicts breast cancer survival and functionally regulates response to chemotherapy. ***Cancer Discovery*** 1(1): 54-67.
- Ruffell B, Chang-Strachan D, Chan V, Rosenbusch A, Ho CMT, Pryer N, Daniel D, Hwang SE, Rugo HS, **Coussens LM**. (2014) Macrophage IL-10 blocks $CD8^+$ T cell-dependent responses to chemotherapy by suppressing IL-12 expression in intratumoral dendritic cells. ***Cancer Cell*** 26:623-637.
- de Mingo Pulido A, Gardner A, Hiebler S, Soliman H, Rugo HS, Krummel MF, **Coussens LM**, Ruffell B. (2018) TIM-3 regulates $CD103^+$ dendritic cell function and response to chemotherapy in breast cancer. ***Cancer Cell***, 33(1): 60-74.

5. *Microenvironmental regulation of tumor progression: specificity matters*: A significant impact of our studies has been recognition that tissues vary with regards to "types" of immune programs used to potentiate

and/or control early cancer. Our seminal findings underscore the tenet that complexity and tissue-specificity of host-tumor programs must be considered for therapy (*deVisser et al., 2006; Tlsty and Coussens 2006; Coussens et al., 2013; Ruffell et al., 2015; Medler et al.,* 2015). Examples of this are illustrated by our studies described above, but also in our efforts to understand tissue-specific roles for stromal cell-derived proteases (23 publications). These studies led us into collaborative endeavors to develop in vivo imaging approaches for select proteolytic activities and/or immune cell infiltrates (13 publications), development of a 13-color polychromatic flow cytometry platform to identify the spectrum of lymphoid and myeloid cells infiltrating solid tumors that was later disseminated as an SOP by the NCI-TMEN Network (*Ruffell et al., 2012; Del Alcazar et al., 2017*). We have elucidated a monocyte and macrophage transcriptomic signature that predicts malignancy based on evaluating either tumor infiltrating macrophages or circulating monocytes (*Cassetta et al., 2019*). And recently, we developed a multiplex immunohistochemistry (mIHC) platform for single cell resolution analyses enabling quantitative and simultaneous evaluation of 12-29 epitopes in one FFPE tissue section, thus preserving regional geography to appreciate tumor heterogeneity in situ (*Tsujikawa et al., 2017; Chang et al., 2017; Banik et al., 2020*). With this platform, we recently published a pancreatic adenocarcinoma (PDAC) atlas on immune contexture utilizing ~130 surgical resection specimens revealing previously unappreciated heterogeneity of immune contexture in this disease (*Liudahl et al., 2021*), that in turn provided a baseline data set in which to evaluate impact of neoadjuvant immune therapy targeting CD40 in PDAC (*Byrne et al., 2021*). These powerful platforms enable retrospective or prospective evaluation of tumor specimens and peripheral blood at near single cell levels for preclinical and clinical patient stratification and therapy response monitoring (*Del Alcazar et al., 2016; Cooper et al., 2016; Gopalakrishnan et al, 2017; Li et al., 2018; Pennock et al., 2018; Means et al., 2019; Reddy et al., 2019; Blair et al., 2019; Hassan et al., 2019; Michaelis et al., 2019; Tsujikawa et al., 2020; Pennycuick et al., 2020; Vayrnen et al., 2021; Link et al., 2021; Gatti et al., 2021; Thomas et al., 2021; Byrne et al., 2021; Labrie et al., 2021; Yoshimura et al., 2021; Taylor et al., 2022; Kitko et al., 2022; Sinha et al., in press*). The combination of resultant single cell imaging data derived from these studies has also enabled tremendous innovation for development of higher fidelity analytic pipelines for investigating features of tumor ecosystems for application toward patient stratification (*Tsujikawa et al., 2019; Schapiro et al., 2021; Blise et al., 2022; Mi et al., 2022*)

- Tsujikawa T, Kumar S, Borkar RN, Azimi V, Thibault G, Chang YH, Balter A, Kawashima R, Choe G, Sauer D, El Rassi E, Clayburgh DR, Kulesz-Martin MF, Lutz ER, Zheng L, Jaffee EM, Leyshock P, Margolin AA, Mori M, Gray JW, Flint PW, **Coussens LM**. (2017) Quantitative multiplex immunohistochemistry reveals myeloid-inflamed tumor-immune complexity associated with poor prognosis. ***Cell Reports****,* 19:203-217.
- Tsujikawa T, Crocenzi T, Durham JN, Sugar E, Wu A, Onners B, Nauroth JN, Anders RA, Fertig EJ, Lahreru DA, Reiss KA, Vonderheide RH, KO AH, Tempero MA, Fisher GA, Considine M, Danilova L, Brockstedt DG, **Coussens LM**, Jaffee EM, Le DT. (2020) Safety, survival, and immune response with GVAX pancreas prime and *Listeria Monocytogenes*-expressing mesothelin (CRS-207) boost vaccines with and without Nivolumab for metastatic pancreatic cancer. ***Clin Can Res****.* 26(14):3578-3588
- Liudahl SM*, Betts CB*, Sivagnanam S*, Morales-Oyarvide V, da Silva A, Yuan C, Smuel Hwang S, Grossblatt-Wait A, Leis KR, Larson W, Lavoie MB, Robinson P, Costa AD, Väyrynen SA, Clancy TE, Rubinson DA, Link J, Keith D, Horton W, Tempero MA, Vonderheide RH, Jaffee EM, Sheppard B, Goecks J, Sears RS, Park BS, Mori M, Nowak JA*, Wolpin BM*, **Coussens LM***. (2021). Leukocyte heterogeneity in pancreatic ductal adenocarcinoma: phenotypic and spatial features associated with clinical outcome. ***Cancer Discovery****,* 11(18): 2014-2031.
- Byrne KT*, Betts CB*, Mick R*, Sivagnanam S, Bajor DL, Laheru DA, Chiorean EG, O'Hara MH, Liudahl SM, Newcomb C, Alanio C, Ferreira AP, Park BS, Ohtani T, Huffman AP, Vayrynen SA, Costa AD, Kaiser JC, Wherry EJ, Cheever MA, Wolpin BM, Furth EF, Jaffee EM, **Coussens LM**, Vonderheide RH. (2021) Neoadjuvant selicrelumab, an agonist CD40 antibody changes in the tumor microenvironment in patients with resectable pancreatic cancer. ***Clin. Can Res****.* 27: 4574-4586.

Published work:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1dQRhESKgaA5R/bibliography/40445210/public/?sort=date&direction=ascending