BIOGRAPHICAL SKETCH

NAME: Silke Paust

eRA COMMONS USER NAME: SILKEPAUST

POSITION TITLE: Associate Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
University of Wisconsin-Madison, WI	B.S.	05/1997	Biochemistry
Washington University, St. Louis, MO	M.S.	01/2000	Biomedical Sciences
Harvard Medical School, Boston, MA	Ph.D.	06/2005	Immunology

A. PERSONAL STATEMENT

I am an Associate Professor of Immunology at The Scripps Research Institute, La Jolla, CA, a member of the San Diego Center for AIDS Research, and a leading authority in Natural Killer (NK) cell biology and NK cell-mediated immunological memory. The foundation of my research is based on my published work demonstrating that tissue-specific subsets of murine and human NK cells are long-lived and capable of immunological memory [1,2]. These data give precedence to harnessing NK cells and their effector functions to prevent or treat human disease. For the past decade, I have used my expertise to dissect NK cells' crucial contributions to mucosal immunity to viral infection and malignancy. My laboratory uses human patient samples, mouse, and humanized-mouse models we generate in-house to study NK cell responses to viruses and solid tumors for these projects. Specifically, we are pursuing three distinct but interrelated projects:

The first project aims to determine how antigen exposure shapes the subsequent NK cell response to HIV. In prior work, we determined the ability of a novel experimental oral HIV vaccine to elicit a protective Natural Killer cell memory response in the gastrointestinal tract of mice with a humanized immune system. This work was supported by R01 AI116282 (PI - Paust). Currently, we aim to identify the NK functional subsets that are most responsive to HIV and their mechanisms of host protection from HIV disease. The knowledge gained from these studies will inform future HIV vaccine design targeting memory NK cells for protection from HIV infection. This work is funded by **R01 AI161014 (PI - Paust)**.

The second project aims to develop a universal prophylactic or therapeutic agent to prevent or treat influenza A infection and associated pathologies. We first established that the vaccination of juvenile mice with inhaled gold-nanoparticles coated with influenza virus-encoded matrix protein 2 extracellular domain derived peptides (AuNP-M2e) elicits robust and lifelong protective immunity to influenza A virus (IAV) infection [3]. Building on these vital data, my laboratory and I developed several M2e-specific monoclonal antibodies and demonstrated them to be highly effective and potentially universal treatment agents for IAV infection in mice [4]. Our current focus is to develop these M2e-specific antibodies as universal and viral-escape-mutant-resistant therapies for human use. This work is supported by **R01 AI130065 (PI - Paust).**

The third project aims to develop NK cell immunotherapy products to treat difficult-to-treat solid tumors, such as lung and pancreatic cancers, and their metastatic tissues using mouse and humanized mouse models of pancreatic cancer. We developed a novel patient-derived xenograft model of lung cancer in prior work. We demonstrated that NK cells and cytotoxic T lymphocytes are required to clear solid tumors (in this case, lung adenocarcinoma) [5]. Since then, we have developed an orthotopic humanized mouse model of pancreatic cancer with metastatic potential. Based on this knowledge, we use this model to identify critical pathways NK cells require for tumor eradication and design, produce, and evaluate NK cell infusion products. This work is essential for developing new and effective immunotherapies to treat highly metastatic cancers refractory to approved therapies. Our work was initially supported by the Helis Medical Foundation Award (PI – Paust) and the Barry Stephen Smith Memorial Pancreatic Cancer Research Award (PI-Paust) and is currently supported by my **Scripps Research Seed Funds**.

During my training and career as a principal investigator, I have published peer-reviewed research papers in many top-tier journals, including *Nature, Nature Immunology, Science Immunology, the Journal of Clinical Investigation, and the Journal of Virology*. In addition to grants, patents, and peer-reviewed publications, I have also achieved substantial academic recognition, including attaining national and international distinction through awards, invited speaking engagements, and professional memberships. As the PI of many NIH and Foundation grants, I have gained significant expertise in project administration, collaborating, and mentoring.

1. Nikzad R., Angelo L. S., Aviles-Padilla K., Le D. T., Singh V. K., Bimler L., Vukmanovic-Stejic M., Vendrame E., Ranganath T., Simpson L., Haigwood N. L., Blish C. A., Akbar A. N., **Paust S.** Human natural killer cells mediate adaptive immunity to viral antigens. *Science Immunology* 2019 May 10;4(35):eaat8116. PMID: 31076527; PMCID: PMC6636344
2. **Paust S**, Gill H.S., Wang B.Z., Flynn M.P., Moseman E.A., Senman B., Szczepanik M., Telenti A., Askenase P.W., Compans R.W., von Andrian U.H. Critical role for the chemokine receptor CXCR6 in NK cell-mediated antigen-specific memory of haptens and viruses. *Nature Immunology* 2010 Dec; 11(12):1127-35. PMID: 20972432; PMCID: PMC2982944; *over 620 citations*
3. Bimler, L.H., Song, Y.H., Le D.T., Murphy-Schafer A., and **Paust S**. AuNP-M2e + sCpG vaccination of juvenile mice generates lifelong protective immunity to influenza A virus infection. *Immunity & Aeging*, 2019 (16:23) Immun Ageing. 2019;16:23. doi: 10.1186/s12979-019-0162-y. eCollection 2019. PubMed PMID: 31507643; PubMed Central PMCID: PMC6720989.
4. Bimler L, Ronzulli SL, Song AY, Johnson SK, Jones CA, Kim T, Le DT, Tompkins SM, **Paust S**. Matrix Protein 2 Extracellular Domain-Specific Monoclonal Antibodies Are an Effective and Potentially Universal Treatment for Influenza A. *Journal of Virology* 2020 Dec 2. doi: 10.1128/JVI.01027-20. PMID: 33268521.
5. Le D.T., Huynh T.R., Burt B., Van Buren G., Abeynaike S.A., Zalfa C., Nikzad R., Kheradmand F., Tyner J.J., **Paust S.** Natural killer cells, and cytotoxic T lymphocytes are required to clear solid tumor in a patient-derived xenograft. ***JCI Insight***. 2021 Jul 8;6(13) PMID: 34081628, doi: 10.1172/jci.insight.140116.

B. POSITIONS, SCIENTIFIC APPOINTMENTS, AND HONORS

Faculty Positions and Major Administrative Roles

2018 – current	Associate Professor, Department of Immunology and Microbiology, The Scripps Research Institute, La Jolla, CA
2018 – current	Member, The San Diego Center for Aids Research (CFAR), San Diego, CA
2018 – current	Member, Center for Precision Immunotherapy at the Moores Cancer Center at the University of California, San Diego, San Diego, CA
2015 – 2018	Member, The Dan L Duncan Cancer Center, TCH and BCM, Houston, TX
2014 – 2018	Member, The Texas Medical Center Digestive Disease Center, Houston, TX
2014 – 2018	Chair, Immunology Seminar Series of the Center for Human Immunobiology and Texas Children's Hospital, Houston, TX
2014 – 2018	Assistant Professor, Department of Microbiology and Molecular Virology, BCM, Houston, TX
2013 – 2018	Assistant Professor, Center for Human Immunobiology, Department of Pediatrics, Texas Children's Hospital (TCH) and Baylor College of Medicine (BCM)
2012 - 2013	Instructor, Division of Immunology, Department of Microbiology and Immunobiology, Medical School, Boston, MA

Other Professional Activities (National/International)

2021 – present	Scientific Consultant, Shoreline Biosciences, Inc., San Diego, CA
2021 – present	Scientific Advisory Board member, Shoreline Biosciences, Inc., San Diego, CA
2020 – present	Scientific Consultant, Qihan Biotechnology, Co., Ltd., Hangzhou, China
2020 – present	Scientific Advisory Board member, Qihan Biotechnology, Co., Ltd., Hangzhou, China
2020 – present	Scientific Advisory Board, Integrative Neuroscience Initiative on Alcoholism-Neuroimmune (INIA-N) Consortium, NIH, NIAAA
2020 – 2021	Scientific Consultant, Kiadis Pharma NV, a Sanofi Company, The Netherlands
2020 – 2021	Scientific Consultant, Poseida Therapeutics Inc., San Diego, CA
2019 – 2021	Guest Editor, Frontiers in Immunology, Innate Immunity
2017 - present	Associate Editor, Frontiers in Cancer Immunity and Immunotherapy
2016 - present	Review Editor, Frontiers in NK and Innate Lymphoid Cell Biology
2016 - present	Review Editor, Frontiers in Molecular Innate Immunity
2017 - 2018	Organizer - The 17th meeting of the Society for Natural Immunity, 2018. San Antonio, TX
2016 - 2018	Member of the scientific organizing committee, Immunology of Human Disease Symposium, July 28th – August 1st, 2019 Santa Fe, NM

Honors

2014	Women & Diversity Paper of the Year Award from the Society of Leukocyte Biology & FASEB, for the most highly cited publication in innate immunity of the past five years
2012-13	T32 NIH training grant, Harvard Medical School, Boston, MA
2012	Travel award from the Society for Natural Immunity (SNI): NK2012, Asilomar, CA.

2010-12	Postdoctoral Fellowship of the Ragon Institute of MGH, MIT & Harvard, Boston, MA
2010	Harvard Medical School Travel Fellowship; Harvard Medical School, Boston, MA.
2008	Keystone Symposia Fellowship: NK and NKT cell biology, Keystone, CO.
2007-09	Irvington Postdoctoral Fellowship of the Cancer Research Institute, New York, NY
2004	The Hauser Fellowship for outstanding achievements in scientific research and teaching throughout their graduate school training, Harvard University, Boston, MA

C. CONTRIBUTIONS TO SCIENCE

1. Discovery of antiviral memory NK cell responses in mice and humans. While NK cells have traditionally been considered cells of the innate immune system, mounting evidence warrants a reconsideration of the existing paradigm that T and B cells are the sole mediators of adaptive immunity. Using HIV-encoded antigens as foreign antigens in mice, as a post-doctoral fellow, I reported that murine NK cells mediate immunological memory to HIV-Gag and -Env [a,b]. Vaccine-antigen-primed hepatic, but not antigen-primed splenic or naive NK cells from either organ developed antigen-specific immunological memory of vaccines containing HIV-Env or HIV-Gag, to influenza A-encoded matrix protein, and inactivated vesicular stomatitis virus. Manipulation of NK activity through vaccination significantly enhanced host protection from infection or, in some cases, elicited cure, even in the absence of T and B cells and pathogen-specific antibodies [a-b]. Also, my laboratory recently demonstrated that human NK cells similarly displayed vaccination-dependent, antigen-specific recall responses utilizing humanized mice and human volunteers. Human NK cells isolated from livers but not from spleens of humanized mice previously vaccinated with HIV-Env protein-mediated enhanced antigen-specific recall responses to HIV-Env *in vitro* [c]. We further showed that many cytotoxic NK cells with a tissue-resident phenotype were recruited to varicella-zoster virus (VZV) skin-test-antigen challenge sites in VZV-experienced human volunteers. NK cell-mediated recall responses in humans occurred decades after initial VZV exposure, emphasizing the human memory NK cell response's longevity upon vaccination or infection [c]. These data are essential as they open the door to developing novel NK-based vaccines and therapeutics that direct potent NK antiviral functions towards host protection from infection [d].

a. **Paust S**, Gill HS, Wang BZ, Flynn MP, Moseman EA, Senman B, Szczepanik M, Telenti A, Askenase PW, Compans RW, von Andrian UH. Critical role for the chemokine receptor CXCR6 in NK cell-mediated antigen-specific memory of haptens and viruses. *Nature Immunology* 2010 Dec;11(12): 1127-35. PMID: 20972432; PMCID: PMC2982944; over 620 citations
b. **Paust S**, and von Andrian UH. Natural killer cell memory. *Nature Immunology* 2011 Jun; 12(6): 500-8. PMID: 21739673
c. Nikzad R., Angelo L. S., Aviles-Padilla K., Le D. T., Singh V. K., Bimler L., Vukmanovic-Stejic M., Vendrame E., Ranganath T., Simpson L., Haigwood N. L., Blish C. A., Akbar A. N., **Paust S.** Human natural killer cells mediate adaptive immunity to viral antigens. *Science Immunology* 2019 May 10;4(35):eaat8116. PMID: 31076527; PMCID: PMC6636344
d. **Paust S**, Blish CA, Reeves RK. Redefining Memory: Building the Case for Adaptive NK Cells. Journal of Virology. 2017 Oct 15;91(20). PMID: 28794018; PMCID: PMC5625515.

2. Discovery of novel functions and biomarkers of human NK memory cell subsets.

Whether patients with defects in NK memory exist is unknown and cannot be studied until human memory NK cell-specific biomarkers are identified. My prior published work revealed that NK cell memory of viruses depends on the chemokine receptor CXCR6 and that its' expression is required for NK cell survival but not for antigen recognition. CXCR6 is expressed on hepatic and lung-resident NK cells in mice and, interestingly, on a subset of tissue-resident NK cells in humans that co-express the transcription factor Eomesodermin (Eomes) but little T-box expressed in T cells (T-bet) [a]. Therefore, we examined the expression of these markers by human NK cells that participate in a memory response to antigen challenge. We found HIV and VZV-specific human memory NK cells to be T-bet^{-}, Eomes^{+}, and CXCR6^{+} in phenotype [a]. To better understand CXCR6^{+} and CXCR6^{-} peripheral blood (PB) NK cells' developmental and tissue-specific differences, we phenotypically and functionally evaluated these human NK cell subsets upon their *in vitro* expansion and contrasted results to bulk liver and spleen NK cells. CXCR6^{+} and CXCR6^{-} PB NK cells preserved their distinct phenotypic profiles throughout the expansion, after which phenotypically immature CXCR6^{+} PB NK cells became functionally equivalent to their CXCR6^{-} counterparts. Despite a consistent reduction in CD16 expression and enhanced expression of the transcription factor Eomes, expanded CXCR6^{+} PB NK cells had superior antibody-dependent cellular cytotoxicity than CXCR6 PB NK cells. Further, bulk liver NK cells responded to IL-15, but not IL-2 stimulation, with STAT-5 phosphorylation. In contrast, bulk splenic and PB NK cells robustly responded to both cytokines [b]. Surprisingly, CXCR6 is expressed early in human fetal immune cell development, as human fetal liver and spleen NK cells already

possess a distinct CXCR6^{+} NK cell population with discrete functional capabilities [c]. This knowledge is important. It adds significantly to our understanding of how NK functional subsets develop in humans, identifies immune memory markers on fetal and adult human NK cells for their continued analysis, and informs on the selection of superior NK cell subsets for NK cell immunotherapy.

a. Nikzad R, Angelo LS, Aviles-Padilla K, Le DT, Singh VK, Bimler L, Vukmanovic-Stejic M, Vendrame E., Ranganath T, Simpson L, Haigwood NL, Blish CA, Akbar AN, **Paust S.** Human natural killer cells mediate adaptive immunity to viral antigens. *Science Immunology* 2019 May 10;4(35):eaat8116. PMID: 31076527; PMCID: PMC6636344
b. Angelo LS, Hogg GD, Abeynaike S, Bimler L, Vargas-Hernandez A, **Paust S**. Phenotypic and Functional Plasticity of CXCR6+ Peripheral Blood NK Cells. *Frontiers in Immunol*ogy 2022 Jan 31;12:810080. PMID: 35173710
c. Angelo LS, Bimler LH, Nikzad R, Aviles-Padilla K, **Paust S**. CXCR6^{+} NK Cells in Human Fetal Liver and Spleen Possess Unique Phenotypic and Functional Capabilities. *Frontiers in Immunol*ogy 2019 Mar 19;10:469. PMID: 30941128; PMCID: PMC6433986.

3. Discovery of novel human NK cell developmental pathways and NK cell deficiencies. My laboratory has made several significant contributions to our understanding of how human NK cells develop and function. In collaboration with Dr. Orange's laboratory at Texas Children's Hospital, we first described rare immunodeficient patients with biallelic mutations in Interferon Regulatory Factor 8 (*IRF8*) [a]. We discovered that the deletion of *irf 8* in mice leads to impaired NK cell maturation and effector functions mimicking that observed in *IRF8*-deficient patients [a]. We were the first to demonstrate that MCM10 is required for terminal NK cell maturation and acquisition of their immune functions [b].

a. Mace EM, Bigley V, Gunesch JT, Chinn IK, Angelo LS, Care MA, Maisuria S, Keller MD, Togi S, Watkin LB, LaRosa DF, Jhangiani SN, Muzny DM, Stray-Pedersen A, Coban Akdemir Z, Smith JB, Hernández-Sanabria M, Le DT, Hogg GD, Cao TN, Freud AG, Szymanski EP, Savic S, Collin M, Cant AJ, Gibbs RA, Holland SM, Caligiuri MA, Ozato K, **Paust S**, Doody GM, Lupski JR, Orange JS. Biallelic mutations in IRF8 impair human NK cell maturation and function. *J Clin* Invest. 2017 Jan 3;127(1):306-320. PMID: 27893462; PMCID: PMC5199714.
b. Mace EM, **Paust S**, Conte MI, Baxley RM, Schmit M, Guilz NC, Mukherjee M, Pezzi AE, Chmielowiec, Tatineni S, Chinn IK, Akdemir ZC, Jhangiani SJ, Muzny JMD, Stray-Pedersen A, Bradley RE, Moody M, Connor PP, Heaps AG, Steward C, Banerjee PP, Gibbs RA, Borowiak M, Lupski JR, Jolles S, Bielinsky AK, Orange JS. Human NK cell deficiency as a result of biallelic mutations in MCM10. *J Clin* Invest. 2020 Oct 1;130(10):5272-5286. doi: 10.1172/JCI134966. PMID: 32865517; PMCID: PMC7524476.

4. Discovery of novel mechanisms regulating solid tumor or metastasis susceptibility to T or NK cell immune surveillance. We recently discovered a critical role for NK cell immune surveillance of metastases in a murine model, comparing the susceptibility of monoclonal vs. polyclonal metastases to NK cell cytotoxicity. We found that resistance to NK cell immunosurveillance confers a selective advantage to polyclonal metastasis [a]. These data are an important finding that may help understand differential responses of primary tumors and originally distinct metastases to NK cell-targeted therapies. Also, to investigate the roles of cytotoxic lymphocytes in solid tumor immune surveillance and to develop novel immunotherapy approaches targeting solid tumors, we use the Smad4/Pten-doubly deficient mouse model of lung cancer. To improve the translational relevance of our work, we built on our strong expertise in generating xenograft mouse models to evaluate cancer therapies [c] to develop novel pre-clinical models of lung adenocarcinoma (LUAD) and pancreatic duct adenocarcinoma (PDAC) [d]. Existing patient-derived xenograft (PDX) mouse models of solid tumors lack a tumor donor matched, syngeneic, and competent immune system. Combined treatment with interleukin-15 stimulation and immune checkpoint inhibition resulted in complete or partial tumor response in this model. Further, depletion of cytotoxic T lymphocytes and/or natural killer cells before combined immunotherapy revealed that both cell types were required for maximal tumor regression. Our TIL-PDX model provides a valuable resource for robust mechanistic and therapeutic studies in solid tumors.

a. Lo H.C., Xu Z., Kim I.S., Pingel B., Aguirre S., Kodali S., Liu J., Zhang W., Muscarella A.M., Hein S.M., Krupnick A.S., Neilson J.R., **Paust S**., Rosen J.M., Wang H., Zhang H.F.X. Resistance to natural killer cell immunosurveillance confers a selective advantage to polyclonal metastasis, *Nature Cancer* 2020;709–722.
b. Moreno-Smith M, Lakoma A, Chen Z, Tao L, Scorsone KA, Schild L, Aviles-Padilla K, Nikzad R, Zhang Y, Chakraborty R, Molenaar JJ, Vasudevan SA, Sheehan V, Kim ES, **Paust S**, Shohet JM, Barbieri E. p53 Nongenotoxic Activation, and mTORC1 Inhibition Lead to Effective Combination for Neuroblastoma

Therapy. *Clinical Cancer Research*. 2017 Nov 1;23(21):6629-6639. doi: 10.1158/1078-0432.CCR-17-0668. Epub 2017 Aug 18. PubMed PMID: 28821555; PubMed Central PMCID: PMC5959272.

c. Le D.T., Huynh T.R., Burt B., Van Buren G., Abeynaike S.A., Zalfa C., Nikzad R., Kheradmand F., Tyner J.J., **Paust S.** Natural killer cells, and cytotoxic T lymphocytes are required to clear solid tumor in a patient-derived xenograft. ***JCI Insight***. 2021 Jul 8;6(13) PMID: 34081628, doi: 10.1172/jci.insight.140116.

5. Discovery of novel central and peripheral T cell tolerance pathways regulating tissue-specific inflammation and disease. Immune dysregulation and aberrant inflammation augment tissue damage, and disease severity, and increase susceptibility to secondary infections and cancer. My Ph.D. thesis work investigated mechanisms of central and peripheral tolerance. I identified CD80 as a target of T-regulatory cell-mediated suppression [a]. Together with one of my colleagues, I identified MINK kinase as a crucial signaling molecule required for the negative selection of thymocytes [b]. As an instructor at Harvard Medical School, I investigated how neuronal triggers influenced tissue-specific autoimmune inflammation and reported that neuron-dendritic cell interactions drive autoimmune inflammation in a mouse model of psoriasis [c]. My more recent investigations into the role of soluble secreted CTLA-4 revealed its essential role in modulating T cell-mediated antiviral and anti-tumor immunity [d]. These data are important as identifying novel pathways that modulate T cell effector functions enables the development of novel therapeutic agents to treat chronic viral infections or perhaps even malignancy.

a. **Paust S**, Lu L, McCarty N, Cantor H. Engagement of B7 on effector T cells by regulatory T cells prevents autoimmune disease. *PNAS.* 2004; 101(28):10398-403. PMID:15235129. *over 482 citations*
b. McCarty N, **Paust S**, Ikizawa K, Dan I, Li X, and Cantor H. Signaling by the kinase MINK is essential in the negative selection of autoreactive thymocytes. *Nature Immunology* 2005; PMID:15608642.
c. Riol-Blanco L, Ordovas-Montanes J, Perro M, Naval E, Thiriot A, Alvarez D, **Paust S**, Wood JN, and von Andrian UH. Nociceptive sensory neurons drive interleukin-23-mediated psoriasiform skin inflammation. *Nature* 2014; 510(7503):157-61. PMID: 24759321 over 320 citations
d. Halpert M, Konduri V, Liang D, Chen Y, Wing J, **Paust S**, Levitt J, Decker W. Dendritic Cell Secreted CTLA-4 Regulates the T-cell Response by Downmodulating Bystander Surface B7. *Stem Cells and Development* 2016. PMID: 26979751.

6. Developing Matrix Protein 2 Extracellular Domain-specific monoclonal antibodies as an effective and potentially universal treatment for influenza A virus infection. Influenza virus infection causes significant morbidity and mortality worldwide. Humans fail to make a universally protective memory response to influenza A because of high mutation rates in the immune-dominant influenza epitopes. The M2-ion channel extracellular domain (M2e) is an ideal antigenic target, as it is highly conserved, has a low mutation rate, and is essential for viral entry and replication. We first reported that the vaccination of juvenile mice with inhaled gold-nanoparticles coated with influenza virus-encoded matrix protein 2 extracellular domain derived peptides (AuNP-M2e) elicits robust and lifelong protective immunity to influenza A virus (IAV) infection [a]. Vaccine-induced M2e-specific B cells are maintained, and memory is protective in old (~22 months old) influenza A challenged mice [a]. Antibodies against M2e have been evaluated in clinical trials, but none were developed into an FDA-approved therapy. Thus, using the above described AuNP-M2e vaccine and vaccination of Balb/c mice, we developed five novel M2e-specific monoclonal antibodies that protect mice at low doses and are cross-protective against circulating and highly pathogenic influenza A virus strains (H5N1, H7N9) [b]. In work currently under peer review, we demonstrate that single or cocktail antibody therapy does not elicit viral immune escape in immunocompetent or immunocompromised mice when the virus is passaged in vivo for three to four weeks. Our data are essential to public health. A universal influenza A vaccine could be administered early in life and maintain lifelong protection. Also, universally protective M2e-specific antibodies could be generated and stockpiled to protect from novel influenza A virus serotypes with pandemic potential when vaccines may not yet be available.

a. Bimler, LH, Song, YH, Le DT, Murphy-Schafer A, and **Paust S**. AuNP-M2e + sCpG vaccination of juvenile mice generates lifelong protective immunity to influenza A virus infection. *Immunity & Aeging,* 2019 Sep 2;16:23. PMID: 31507643
b. Bimler L, Ronzulli SL, Song AY, Johnson SK, Jones CA, Kim T, Le DT, Tompkins SM, **Paust S**. Matrix Protein 2 Extracellular Domain-Specific Monoclonal Antibodies Are an Effective and Potentially Universal Treatment for Influenza A. *Journal of Virology* 2020 Dec 2. doi: 10.1128/JVI.01027-20. PMID: 33268521.
c. Bimler L, Kim T, Ronzulli SL, Song, YH, Johnson SK, Jones CA, S. Tompkins SM, **Paust S**. M2e-specific monoclonal antibody cocktails against influenza A virus are superior to individual monoclonal antibody treatments, universally effective, and viral escape mutant resistant. BioRxiv preprint 04 2022: doi: https://doi.org/10.1101/2022.04.02.486847

Complete List of Published Work for Silke Paust (h-index: 21; i10index: 27, citations: >3,460):
https://www.ncbi.nlm.nih.gov/sites/myncbi/silke.paust.1/bibliography/47563634/public/?sort=date&direction=ascending