MJ Cancer Patient Advocate, LLC

Minji Jo, Ph.D., Independent cancer patient advocate consultant

My career in cancer research began with my graduate studies at the University of Pittsburgh School of Medicine, for which I earned my PhD in Cellular and Molecular biology in 2000. My graduate work focused on how the proteins, epidermal growth factor receptor (EGFR) and c-met, both of which are abundant in many cancers, interact during cancer development. Since then, I have dedicated my life to cancer research and oncology drug development.

As a research scientist in academia, I studied how the protein, urokinase (uPA) and its partner, urokinase receptor (uPAR), function in cancer development and promote cancer spread throughout the body by breaking down tissue membranes. My research expanded our understanding of how uPA and uPAR function in the spread of cancer by identifying a previously unknown mechanism of action. More than 20 publications in highly regarded peer-reviewed scientific journals are resulted from my continued research on this subject. During this time, I gained extensive hands-on experience and knowledge in the biology of different types of cancer including breast, lung, liver and brain, as well as other related disciplines such as cell and molecular biology, biochemistry and pathology.

During my thriving career as an academic research scientist, I began to look for ways I could use my skill set to help cancer patients more directly. With that in mind, I successfully transitioned to a role as a scientist in the field of oncology drug development at Ionis Pharmaceuticals, Inc. There, I successfully led several pre-clinical programs to develop drugs for patients with lymphoma, lung and liver cancer. At the same time, I led several different strategic programs to improve delivery, safety and efficacy of cancer drugs.

While I was in transition in 2019, my best friend told me that her husband had been recently diagnosed with stage IV glioblastoma, the most aggressive form of brain cancer. She and her husband were understandably shocked and overwhelmed by the diagnosis and had difficulty understanding the clinical trial options that were presented to them. At their request, I reviewed the pathology and genetic profiling reports from the doctor, as well as the clinical trial options. My extensive experience over more than 15 years in cancer research and oncology drug development gave me the tools I needed to understand and clearly explain, in plain English, what his pathology and genetic profiling reports meant and what the clinical trial would entail. More importantly, I understood the scientific details about the clinical trials to help them in making an educated decision about the clinical trial that was best-suited. I also identified a few more promising clinical trial options. For each subsequent clinical trial, I reviewed and explained them to my friend and her husband. I then tailored questions for them to ask his doctors, so that they could make a well-informed decision about each clinical trial presented to them. In addition, I was able to answer their questions regarding treatments, side effects and any problems they had to face on a daily basis. I was very glad that I was able to help to ease their burden in any way I can. I also found it to be very fulfilling to see my experiences and knowledge positively impact a cancer patient in a more direct way.

Not long after this experience with assisting my friend and her husband navigate his cancer diagnosis, treatment options and plans, a neighbor asked me for help with his father's diagnosis. His father had hairy cell leukemia, which had been in remission, symptom-free, for 10 years. Unfortunately, he had recently discovered that the cancer had returned. To complicate matters, his father was recovering

from a heart attack, which may have been caused by rituximab, a drug known to potentially damage the heart, prescribed to treat his cancer. This limited the treatment options available to him that would not further stress or damage his heart and identifying the best options in this situation was challenging, as a result. His doctors presented him with four treatment options, but my neighbor and his father were not equipped with the knowledge required to confidently select the most appropriate treatment for his special medical needs. With my knowledge and skill set, I was able to analyze previously published studies detailing the available treatments and assisted this family by explaining what each treatment would entail along with special attention to any possible side effects in terms of severity and cardiac toxicity. I am not a physician and thus cannot give them medical advice or make a medical decision for them. However, what I was able to offer allowed my neighbor and his father to identify a treatment course that would be least likely to further damage his heart based on published research studies about the potential treatments, and therefore had the highest chance of a positive outcome. However, his doctor decided to continue the treatment of rituximab, which resulted in another cardiac event. His doctor decided to switch his treatment to vemurafenib, which I had informed my neighbor and his father to be the best possible treatment with the least cardiac toxicity. Even with my limited ability to make a final decision for cancer patients, it was very satisfying to know that my advice gave my neighbor and his father confidence that the current treatment is possibly the best for him. In this case, as well, I applied my knowledge and skills to provide support by answering any questions they had during his cancer treatment.

These experiences brought to my attention the highly unmet need for someone like myself who has extensive experience and knowledge in cancer biology and cancer drug development to effectively guide cancer patients to make well-informed decisions about their treatment options. I have always been motivated in my career to make a difference in the lives of cancer patients, and my journey has brought me to a place where I know I am uniquely qualified to directly improve cancer patients' treatment and provide them peace of mind in their decision-making process. In this role, I will apply my years of cancer research and drug development experience to assist these patients and their families by educating them in a way they can understand about their diagnosis, treatment options and what those treatments entail, and what they expect from the cancer treatment experience.

What distinguishes me from other patient advocates is that I have extensive experience and a deep understanding of cancer biology and oncology drug development. This allows me to provide more informed services to patients than the average patient advocate. I am certain that physicians want to be able to devote their time to each patient, explaining all the available treatments and related issues, but it is difficult for them to do so with the high volume of patients they have to treat. Some hospitals have patient advocates, most of whom are registered nurses. While nurses are often able to answer questions from cancer patients, they may not have the experience of cancer research and drug development process and clinical trials as well as someone who worked in that industry or time to review and analyze scientific peer-reviewed publications regarding the latest cancer research and drug development.

There are many non-profit and private organizations that help patients to find clinical trials. However, the services they offer typically do not go beyond providing a list of available trials. While the service they provide is useful, the specific information for each trial that is provided is limited. In comparison, there is great value to having assistance from someone who is trained and experience in understanding the science behind current cancer therapies and clinical trials.

I have dedicated my life to the study of the biological mechanisms and genetics behind cancer for many years. I have worked on development of cancer drugs for over 15 years and proven my expertise in these fields with nearly 30 publications in highly-regarded, peer-reviewed scientific journals. Thus, I believe I am uniquely qualified to review and analyze clinical trials to determine what may be the best possible fit patients based on each patient's individual pathology and the genetic make-up of their cancer. No one can guarantee the success of a therapy or a clinical trial, no matter how much knowledge or experience one has. However, thoughtful scientific analysis of available treatments or clinical trials and guidance personalized for each patient's needs in mind, would allow them to make a well-informed decision to maximize their chance of survival and wellbeing.

Importantly, I am not beholden to any institute or organization. Dedicated as they might be, the patient advocates of hospitals or pharmaceutical companies (even physicians) have allegiance to their employers. As an independent consultant, I am able to assist and guide cancer patients without any bias or conflict of interest to ensure the maximum benefit and highest quality of service to each individual cancer patient. Lastly, I am committed to building patient relationships and will meet patients personally to discuss their treatment options, as well as following up regularly throughout their treatment to reassess their changing needs. This will allow me to provide the highest quality of service as well as peace of mind for cancer patients throughout their healing journey.

Minji Jo, Ph.D.

Education:

Postdoctoral Fellow, Department of Pathology, School of Medicine University of Virginia, Charlottesville, VA, **2000-2001**

Doctor of Philosophy, Cellular & Molecular Pathology, School of Medicine University of Pittsburgh, Pittsburgh, PA, **1994-2000**

Bachelor of Science, Department of Microbiology, Kyoung-Pook National University, Daegu, Korea, **1988-1993**

Honors and Fellowships:

1988-1993 Honor Scholarship. Kyoung-Pook National University

1998 Second Prize, 9th Annual Pathology Research Presentation, University of Pittsburgh

1999 First Prize, 10th Annual Pathology Research Presentation, University of Pittsburgh

2000 Doctoral Dissertation with honor, University of Pittsburgh

2000 Institutional Excellence Incentive, University of Virginia

Professional and Research Experience:

2021-present, Scientific Consultant, Soteria Precision Medicine Foundation, San Diego, CA

2020-present, ***Founder and Independent cancer patient advocate consultant***, MJ Cancer Patient Advocate, LLC., Vista, CA

2019-present, Senior Advisor, Biomarker Council, International Cancer Advocacy Network, Phoenix, AZ

2017-2018 Senior Scientist**,** Lead of Immuno-Oncology group, Organovo, San Diego, CA

2012-2017 Senior Scientist**,** Antisense Drug Discovery, Oncology group, IONIS Pharmaceuticals, Inc., Carlsbad, CA

2011-2012 Associate Project Scientist**,** Department of Pathology, University of California, San Diego School of Medicine, La Jolla, CA

2004-2011 Assistant Project Scientist**,** Department of Pathology, University of California, San Diego School of Medicine, La Jolla, CA

2001-04 Research Scientist*,* Department of Pathology, University of Virginia, Charlottesville, VA

Podium and Poster Presentations (Selected):

1. *Cross-talking between EGFR and c-met in transformed cells*. **Podium presentation** in Young Investigators Mini-Symposium on Growth Factor Receptor Tyrosine Kinases in Development, Tissue Regeneration, and Neoplastic Disease. "Growth Factor Receptor Tyrosine Kinases in Mitogenesis,

Morphogenesis, and Tumorigenesis", FESEB Summer Research Conference. Jul. 31- Aug. 5, 1999, Snowmass Village, CO.

2. *Apoptosis and the Liver: a Mechanism of Disease, Growth Regulation, and Carcinogenesis*. American Association for the Study of Liver Diseases' Basic Research Single Topic conference, June 17-20, 1999, Arlie, VA.
3. *Urokinase Receptor Enables EGF as a mitogen.* Poster and **Podium presentation** in the Gordon Research Conference "Plasminogen and Extracellular Proteolysis". Feb. 19-24, 2006, Ventura, CA.
4. *LRP1 Regulates Schwann cells Motility by its Effects on the Activity of the GTPases, Rac and Rho.* Neuroscience Meeting. Oct. 17-21, 2011, Chicago, IL.
5. *Characterization of GalNAc-conjugated generation 2.5 ASOs in DEN and DEN/CCL4-induced HCC tumors.* AACR, Apr. 18-22, 2015, Philadelphia, PA.
6. *Strong pharmacological activity of locally administered next generation antisense oligonucleotides (ASOs) in orthotopic lung cancer mouse models.* 4th AACR-IASLC International Joint Conference on Lung Cancer Translational Science. Jan. 4-7, 2016, San Diego, CA.

Publications:

1. Sharon C. Presnell, Donna B. Stolz, Wendy M. Mars, **Minji Jo**, George K. Michalopoulos, and Stephen C. Strom. Modifications of the hepatocyte growth factor/c-met pathway by constitutive expression of transforming growth factor-alpha in rat liver epithelial cells. **Molecular Carcinogenesis** 18(4): 244-55 (1997).
2. **Minji Jo,** Donna B. Stolz, James. E. Esplen, Kenneth Dorko, George K. Michalopoulos, and Stephen C. Strom. Cross-talk between EGFR and c-met in transformed cells. **Journal of Biological Chemistry** 275(12): 8806-8811 (2000).
3. **Minji Jo,** Tae-Hyoung H. Kim, Dae W. Seol, James. E. Esplen, Kenneth Dorko, and Stephen C. Strom. Apoptosis induced in normal human hepatocytes by tumor necrosis factor-related apoptosis-inducing ligand. **Nature Medicine** 6(5): 564-567 (2000).
4. Zhong Ma, Donna J. Webb, **Minji Jo,** and Steven L. Gonias. Endogenously-produced urokinase-type plasminogen activator is a major determinant of the basal level of activated ERK/MAPK kinase and prevents apoptosis in MDA-MB-231 breast cancer cells. **Journal of Cell Science** 114:3387-3396 (2001).
5. **Minji Jo**, Keena S. Thomas, Avril V. Somlyo, Andrew P. Somlyo, and Steven L. Gonias. Cooperativity between the Ras-ERK and Rho-Rho Kinase Pathways in Urokinase-type Plasminogen Activator-stimulated Cell Migration. **Journal of Biological Chemistry** 277:12479-12485 (2002).
6. **Minji Jo,** Keena S. Thomas, Denise M. O'Donnell and Steven L. Gonias. Epidermal Growth Factor Receptor-dependent and –independent Cell-signaling Pathways Originating from the Urokinase Receptor. **Journal of Biological Chemistry** 278:1642-1646 (2003).
7. **Minji Jo**, Keena S. Thomas, Lihua Wu, and Steven L. Gonias. **Soluble Urokinase-type Plasminogen Activator Receptor Inhibits Cancer Cell Growth and Invasion by Direct Urokinase-independent Effects on Cell Signaling Journal of Biological Chemistry**, 278: 46692 – 46698 (2003).
8. **Minji Jo**, Keena S. Thomas, Nadzeya Marozkina, Tanay J. Amin, Corinne M. Silva, Sarah J. Parsons, and Steven L. Gonias. **Dynamic Assembly of the Urokinase-type Plasminogen Activator Signaling Receptor**

Complex Determines the Mitogenic Activity of Urokinase-type Plasminogen Activator. Journal of Biological Chemistry 280: 17449 – 17457 (2005).

9. Wendy M. Mars, **Minji Jo,** and Steven L. Gonias Activation of Hepatocyte Growth Factor by Urokinase-type Plasminogen Activator is Ionic Strength-dependent. **Biochemical Journal**, 390:311-315 (2005).
10. Robin D. Lester, **Minji Jo**, W. Marie Campana, and Steven L. Gonias. Erythropoietin Promotes MCF-7 Breast Cancer Cell Migration by a ERK/MAP kinase-dependent Pathway and is Primarily Responsible for the Increase in Migration Observed in Hypoxia. **Journal of Biological Chemistry**, 280(47):39273-39277 (2005).
11. **Minji Jo**, Keena S. Thomas, Shinako Takimoto, En-Hui Hsieh, Steven L. Gonias Urokinase Receptor Signaling Enables EGF as a Mitogen. **Oncogene,** 26:2585-2594 (2007).
12. Robin D. Lester*, **Minji Jo*,** Valerie Montel, Shinako Takimoto and Steven L. Gonias. uPAR Induces Epithelial-Mesenchymal Transition in Hypoxic Breast Cancer Cells. **Journal of Cell Biology** 178:425-436 (2007) *** These authors contributed equally to this paper.**
13. **Minji Jo**, Shinako Takimoto, Valerie Montel, Steven L. Gonias. uPAR promotes cancer metastasis independently of urokinase and its role in protease activation in mice. **American Journal of Pathology**, 175:190-200 (2009).
14. **Minji Jo**, Robin D. Lester, Valerie Montel, Boryana Eastman, Shinako Takimoto, Steven L. Gonias. Reversibility of Epithelial-mesenchymal transition induced in breast cancer cells by hypoxia or by urokinase receptor over-expression. **Journal of Biological Chemistry**, 284:22825-22833 (2009)
15. Elisabetta Mantuano, **Minji Jo**, Steven L. Gonias, W. Marie Camapna Low density lipoprotein receptor-related protein (LRP1) regulates Rac1 and RhoA reciprocally to control Schwann cell adhesion and migration. **Journal of Biological Chemistry**, 285:14259-14266 (2010)
16. **Minji Jo*,** Boryana M. Eastman*, Drue L. Webb, Konstantin Stoletov, Richard Klemke, Steven L. Gonias. Cell Signaling by Urokinase-type Plasminogen Activator Receptor Induces Stem Cell–like Properties in Breast Cancer Cells. **Cancer Research,** 70:8948-8958 (2010) *** These authors contributed equally to this paper.**
17. Steve L. Gonias, Alban Gaultier and **Minji Jo**. Regulation of the Urokinase Receptor (uPAR) by LDL Receptor-related Protein-1 (LRP1). **Current Pharmaceutical Design,** 17(19):1962-1969 (2011)
18. Jingjing Hu, **Minji Jo**, Webster K. Cavenee, Frank Furnary, Scott R. VandenBerg and Steven L. Gonias. Crosstalk between the Urokinase Receptor/uPAR and EGFRvIII Supports Survival and Growth of Glioblastoma Cells. **Proceedings of the National Academy of Sciences of the United States of America,** 108(38):15984-15989 (2011)
19. Boryana M. Eastman, **Minji Jo**, Drue L. Webb, Shinako Takimoto and Steven L. Gonias. A Transformation in the Mechanism by which the Urokinase Receptor Signals Provides a Selection Advantage for Estrogen Receptor Expressing Breast Cancer Cells in the Absence of Estrogen. **Cellular Signaling**, 24(9):1847-55 (2012)
20. Jeanne M. Bristow, Theresa A. Reno, **Minji Jo**, Steven L. Gonias, and Richard L. Klemke. Dynamic Phosphorylation of Tyrosine-665 in Pseudopodium-Enriched Atypical Kinase (PEAK1) is Essential for the Regulation of Cell Migration and Focal Adhesion Turnover. **Journal of Biological Chemistry,** 288(1):123 -31 (2013)

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22. Jingjing Hu,* **Minji Jo**,* Boryana M. Eastman, Andrew J. Glider, Jack D. Bui, and Steven L. Gonias. uPAR Induces Expression of TGFβ and Interleukin-4 in Cancer Cells to Promote Tumor-permissive Conditioning of Macrophages, **American Journal of Pathology**, 184(12):3384-92, (2014) *** These authors contributed equally to this paper.**

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25. Matthew J. Reilley, Patricia McCoon, Carl Cook, Paul Lyne, Razelle Kurzrock, Youngsoo Kim, Richard Woessner, Anas Younes, John Nemunaitis, Nathan Fowler, Michael Curran, Qinying Liu, Tianyuan Zhou, Joanna Schmidt, **Minji Jo**, Samantha J. Lee, Mason Yamashita, Steven G. Hughes, Luis Fayad, Sarina Piha-Paul, Murali V. P. Nadella, Xiaokun Xiao, Jeff Hsu, Alexey Revenko, Brett P. Monia, A. Robert MacLeod, and David S. Hong. STAT3 antisense oligonucleotide AZD9150 in a subset of patients with heavily pretreated lymphoma: results of a phase 1b trial. **Journal for Immunotherapy of Cancer**. 2018 6(1):119-28, (2018)

26. Youngsoo Kim, **Minji Jo**, Joanna Schmidt, Xiaolin Luo, Thazha. P. Prakash, Tianyuan Zhou, Stephanie Klein, Xiaokun Xiao, Noah Post, Zhengfeng Yin, and A. Robert MacLeod, Enhanced Potency of GalNAc-conjugated Antisense Oligonucleotides in Hepatocellular Cancer Models. **Molecular Therapy**, 27(9):1547-57 (2019)

27. Alfred Chappell, Hans Gaus, Andres Berdeja, Ruchi Gupta, **Minji Jo**, Thazha Prakash, Michael Oestergaard, Eric Swayze, Punit Seth. Mechanisms of Palmitic Acid-Conjugated Antisense Oligonucleotide Distribution in Mice. **Nucleic Acids Research** (2019) In Submission