




Claudia Katharina Petritsch

Associate Professor (Research) of Neurosurgery

Adult Neurology

 NIH Biosketch available Online

 Curriculum Vitae available Online

Bio

BIO

Claudia earned her PhD (Dr. rer.nat) at the Institute for Molecular Pathology (IMP) in Vienna, where she trained in cancer signaling, and identified crucial regulators of growth factor receptor kinase signaling. Her postdoctoral studies on neural stem cells and asymmetric cell division in the Lab of Dr. Yuh Nung Jan at the Howard Hughes Medical Institute and University of San Francisco, California implied for the first time a minus-end directed myosin in the process of cell fate determination. During two years as an instructor and head of a research group in Munich, Germany, Dr. Petritsch and her team showed that cell fate determinants use a bimodal mechanism (diffusion and active capturing) for proper intracellular location. She returned to UCSF to conduct translational research, and apply her combined expertise in stem cells and signaling on the study of brain neoplasms and human stem and progenitor cells. Dr. Petritsch is an expert in oligodendrocyte progenitor cells, and cancer stem cells, and her team's emphasis is on intra-tumoral heterogeneity, in vitro and in vivo cancer model development, and tumor-immune interactions. Her research identified conserved mechanisms of cell fate determination in mammalian brain progenitors and led to a paradigm shift in understanding how brain progenitor cells self-renew and differentiate. She guided the generation and distribution of several immune competent mouse models for studies of the glioma immune microenvironment. A major effort of her team is to facilitate the use of fresh surgical tissue from neurosurgeries for research.

CURRENT ROLE AT STANFORD

Director of NCI-funded Cancer Model Development Center

ACADEMIC APPOINTMENTS

- Member, Bio-X
- Member, Maternal & Child Health Research Institute (MCHR)
- Member, Stanford Cancer Institute
- Member, Wu Tsai Neurosciences Institute

HONORS AND AWARDS

- Pediatric Cancer Model Development - Human Cancer Model Initiative, National Cancer Institute (2021)
- Cancer Research Award, Emerson Collective
- Seed Award, Women's Health & Sex Differences in Medicine (WHSMD) (01/01/2021-12/31/2021)

BOARDS, ADVISORY COMMITTEES, PROFESSIONAL ORGANIZATIONS

- Member, Society for Neuro-Oncology (2009 - present)

LINKS

- My Lab Site: <https://med.stanford.edu/petritsch-lab.html>

Research & Scholarship

RESEARCH INTERESTS

- Brain and Learning Sciences
- Research Methods
- Science Education

CURRENT RESEARCH AND SCHOLARLY INTERESTS

The Petritsch lab broadly investigates underlying causes for the intra-tumoral heterogeneity and immune suppression in brain tumors from a developmental neuro-developmental perspective. Proper cell fate decisions by neuroglia stem cells are critical for growing the cell lineages that form the brain during development and to maintain adult brain homeostasis. The mechanisms for cell fate decisions in the human brain are largely unknown. By using patient-derived cells from brain surgeries, we investigate cell fate decision mechanisms in the normal brain and in brain malignancies. We think that defective cell fate decisions fuel the intra-tumoral heterogeneity and plasticity that makes treatment of human brain tumors so challenging. We therefore work to gain an understanding of how brain cells control the fate of their progeny, whereby we unravel novel points of vulnerabilities in brain tumor cells, that could be exploited therapeutically.

Excessive proliferation, apoptotic evasion, and migratory spread are all hallmarks of tumorigenesis. However, these defects fail to explain the incredible heterogeneity and immune suppression observed in malignant brain tumors, two major hurdles to their treatment, which remains mostly palliative.

In the healthy brain, neuroglia stem cells generate progenitors, which in turn give rise to differentiating cells that will eventually acquire their final functional state. Cell fate decisions within these hierarchical brain cell lineages are tightly controlled and irreversible: e.g. cells in the state of differentiation will not turn into progenitor cells or stem cells. It is known that brain tumor cells, on the other hand, defy many general principles of neurobiology. This is especially true for malignant glioma cells, which simultaneously express markers of different lineages and states exhibiting incomplete differentiation. Tumor cell hierarchies are poorly understood, providing no explanation for why tumor cells with stem-like, progenitor-like, and differentiated features co-exist and interact with normal brain cells and immune-infiltrating cells within a single tumor entity, and how this heterogeneity relates to the lack of active immune infiltration.

Defects in cell fate control could explain many key defects present in brain tumors. Of special emphasis, we study the establishment of cell fates within normal hierarchical brain lineages for comparison to the dysregulated cell-fate hierarchies seen in brain tumors. Our lab was the first to demonstrate that normal adult oligodendrocyte progenitor cells (OPCs) undergo asymmetric divisions to make cell fate decisions, i.e. to generate OPCs as well as differentiating cells each time they divide. Drawing from these data, we investigate whether brain tumors divide along hierarchical lineages and how oncogenic mutations might affect cell fate decisions within these hierarchies. A major line of investigation in our lab focuses on whether defects in asymmetric division lead to aberrant cell fate decisions that cause the paradigm mixed lineage phenotypes and the intra-tumoral heterogeneity present across tumors. We complement our work with human cells with orthotopic and genetically engineered mouse models of gliomagenesis to conduct molecular, cellular and bioinformatic analyses

Teaching

STANFORD ADVISEES

Postdoctoral Research Mentor

Ruolun Wei

Publications

PUBLICATIONS

- **A spatially resolved human glioblastoma atlas reveals distinct cellular and molecular patterns of anatomical niches.** *Nature communications*
Sonpatki, P., Park, H. J., Xing, Y. L., Han, K. Y., Schroeder, B. A., Yu, H. J., Kim, H. J., Chowdhury, T., Hwang, J. H., Nam, S. M., Byun, Y. H., Kang, H., Lee, et al
2026
- **Unraveling transcriptomic and phosphokinomic dynamics of the tumor microenvironment during glioma progression**
Xing, Y., Bui, B., Cordero, K., Ngok, E., Magtoto, C., Panovska, D., Nirschl, J., Coppe, J., Petritsch, C.
OXFORD UNIV PRESS INC.2025: v444
- **BRAF/MEK inhibition induces cell state transitions boosting immune checkpoint sensitivity in BRAFV600E-mutant glioma.** *Cell reports. Medicine*
Xing, Y. L., Panovska, D., Park, J. W., Grossauer, S., Koeck, K., Bui, B., Nasajpour, E., Nirschl, J. J., Feng, Z. P., Cheung, P., Habib, P., Wei, R., Wang, et al
2025: 102183
- **GABAergic neuron-to-glioma synapses in diffuse midline gliomas.** *Nature*
Barron, T., Yalçın, B., Su, M., Byun, Y. G., Gavish, A., Shamardani, K., Xu, H., Ni, L., Soni, N., Mehta, V., Maleki Jahan, S., Kim, Y. S., Taylor, et al
2025
- **H3.3-G34R Mutation-Mediated Epigenetic Reprogramming Leads to Enhanced Efficacy of Immune Stimulatory Gene Therapy in Diffuse Hemispheric Gliomas.** *bioRxiv : the preprint server for biology*
Garcia-Fabiani, M. B., Haase, S., Banerjee, K., Zhu, Z., McClellan, B. L., Mujeeb, A. A., Li, Y., Tronrud, C. E., Varela, M. L., West, M. E., Yu, J., Kadiyala, P., Taher, et al
2025
- **CONCURRENT IMMUNE CHECKPOINT BLOCKADE ENHANCES THE SURVIVAL BENEFIT OF CLINICALLY RELEVANT MAPK PATHWAY INHIBITORS IN A T CELL-DEPENDENT MANNER**
Xing, Y., Park, J., Grossauer, S., Panovska, D., Bui, B., Morales, D., Nirschl, J., Feng, Z., Anderson, W., Levy, J., Schreck, K., Prolo, L., Hambardzumyan, et al
OXFORD UNIV PRESS INC.2024
- **CDC42BPA::BRAF represents a novel fusion in desmoplastic infantile ganglioglioma/desmoplastic infantile astrocytoma.** *Neuro-oncology advances*
Barros Guinle, M. I., Nirschl, J. J., Xing, Y. L., Nettnin, E. A., Arana, S., Feng, Z. P., Nasajpour, E., Pronina, A., Garcia, C. A., Grant, G. A., Vogel, H., Yeom, K. W., Prolo, et al
2024; 6 (1): vdae050
- **FIRST-IN-CLASS CANCER THERAPEUTICS TARGETING THE DNA DAMAGE RESPONSE**
Robertson, A., Prikrylova, T., Lancero, H., Wilson, C., Petritsch, C.
OXFORD UNIV PRESS INC.2023
- **Successes and challenges in modeling heterogeneous BRAFV600E-mutated central nervous system neoplasms.** *Frontiers in oncology*
Xing, Y. L., Panovska, D., Petritsch, C. K.
2023; 13: 1223199
- **LGL1 binds to Integrin $\beta 1$ and inhibits downstream signaling to promote epithelial branching in the mammary gland.** *Cell reports*
Ma, R., Gong, D., You, H., Xu, C., Lu, Y., Bergers, G., Werb, Z., Klein, O. D., Petritsch, C. K., Lu, P.
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- **Lgl1 controls NG2 endocytic pathway to regulate oligodendrocyte differentiation and asymmetric cell division and gliomagenesis** *NATURE COMMUNICATIONS*
Daynac, M., Chouchane, M., Collins, H. Y., Murphy, N. E., Andor, N., Niu, J., Fancy, S. P. J., Stallcup, W. B., Petritsch, C. K.
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- **Pan-cancer analysis of the extent and consequences of intratumor heterogeneity** *NATURE MEDICINE*
Andor, N., Graham, T. A., Jansen, M., Xia, L. C., Aktipis, C. A., Petritsch, C., Ji, H. P., Maley, C. C.
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- **Targeting a Plk1-Controlled Polarity Checkpoint in Therapy-Resistant Glioblastoma-Propagating Cells** *CANCER RESEARCH*
Lerner, R. G., Grossauer, S., Kadkhodaei, B., Meyers, I., Sidorov, M., Koeck, K., Hashizume, R., Ozawa, T., Phillips, J. J., Berger, M. S., Nicolaidis, T., James, C., Petritsch, et al
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- **Asymmetry-Defective Oligodendrocyte Progenitors Are Glioma Precursors** *CANCER CELL*
Sugiarto, S., Persson, A. I., Munoz, E. G., Waldhuber, M., Lamagna, C., Andor, N., Hanecker, P., Ayers-Ringler, J., Phillips, J., Siu, J., Lim, D. A., Vandenberg, S., Stallcup, et al
2011; 20 (3): 328-340
- **Non-stem cell origin for oligodendroglioma.** *Cancer cell*
Persson, A. I., Petritsch, C., Swartling, F. J., Itsara, M., Sim, F. J., Auvergne, R., Goldenberg, D. D., Vandenberg, S. R., Nguyen, K. N., Yakovenko, S., Ayers-Ringler, J., Nishiyama, A., Stallcup, et al
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- **Dynamic kinome reprogramming drives resistance to treatments in BRAF^{V600E} pediatric gliomas, revealing actionable therapeutic vulnerabilities**
Panovska, D., Dobler, R., Zakraoui, O., Xing, L., Baroncini, L., Caruso, J., Pirker, R., Munoz, D., Nasajpour, E., Banik, I., Nguyen, D., Hwang, Y., Lin, et al
OXFORD UNIV PRESS INC.2025: v182-v183
- **Inflammation-driven Resistance to MAPK inhibition in Pediatric Gliomas: Mechanisms and Therapeutic Strategies**
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- **COMPARATIVE PATHOLOGY OF A RAREMN1::PATZ1-FUSION NEUROEPITHELIAL PRIMARY TUMOR AND RECURRENCE DEMONSTRATING MARKED HETEROGENEITY, POTENTIAL CHANGES IN TUMOR IMMUNE COMPOSITION, AND CELL-STATE TRANSITIONS**
Nasajpour, E., Wei, R., Panovska, D., Xing, L., Dhami, T., Che, C., Rabin, L., Feng, Z., Lyle, G., Beale, H., Kephart, E., Vaske, O., Geraldo, et al
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- **LEVETIRACETAM THERAPEUTICALLY TARGETS GABAERGIC SYNAPSES IN DIFFUSE MIDLINE GLIOMA**
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- **Quantitative surfaceome profiling of high-risk medulloblastoma prioritizes the oncofetal antigen GPC2 for potent CAR-T cell therapy**
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- **Circadian rhythms in pediatric high-grade gliomas may contribute to treatment efficacy.** *Scientific reports*
Nettnin, E. A., Garcia, C. A., Sayed, F. F., Schonfeld, E., Nguyen, T., Guinle, M. I., Petritsch, C. K., Gibson, E. M., Prolo, L. M.
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- **THE ROLE OF MAP4K4 IN TUMOR CELL INVASION OBSERVED IN A 3D IN VITRO MODEL OF PEDIATRIC GLIOBLASTOMA**
Garcia, C., Nettnin, E., Arana, S., Nguyen, T., Guinle, M., Wilson, C., Petritsch, C. K., Bassik, M. C., Prolo, L. M.
OXFORD UNIV PRESS INC.2024
- **PEDIATRIC GLIOMAS EXPRESS CIRCADIAN GENES AND DEMONSTRATE CIRCADIAN REGULATION OF TEMOZOLOMIDE SENSITIVITY**
Nettnin, E. A., Garcia, C. A., Schonfeld, E., Mehl, L. C., Guinle, M., Petritsch, C. K., Gibson, E. M., Prolo, L. M.
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- **DEUTERIUM METABOLIC IMAGING OF RESPONSE TO PRECISION THERAPY IN BRAF-V600E MUTANT GLIOMAS**
Batsios, G., Udutha, S., Gillespie, A., Petritsch, C., Mueller, S., Viswanath, P.
OXFORD UNIV PRESS INC.2024
- **BIOPROCESSING OF VIABLE SURGICAL PEDIATRIC BRAIN TUMOR SPECIMENS FOR GENOME-GUIDED PERSONALIZED DRUG TESTING**
Nasajpour, E., Tran, C., Garcia, C., Xing, L., Lyle, G., Guinle, M., Bagley, B., Mahaney, K., Vogel, H., Learned, K., Vaske, O., Grant, G., Prolo, et al
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- **GLIOMA-ASSOCIATED OLIGODENDROCYTE PROGENITOR CELLS' DYNAMIC PATTERNS SUGGEST FUNCTIONAL ADAPTATIONS DURING GLIOMA PROGRESSION IN A UNIQUE MOUSE MODEL**
Petritsch, C. K., Xing, L., Bui, B.
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- **BRAF V600E in a preclinical model of pleomorphic Xanthoastrocytoma: Analysis of the tumor microenvironment and immune cell infiltration dynamics in vivo.** *Molecular therapy. Oncology*
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- **DEUTERIUM METABOLIC IMAGING OF RESPONSE TO PRECISION THERAPY IN BRAF-V600E MUTANT GLIOMAS**
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- **CDC42BPA::BRAF REPRESENTS A NOVEL FUSION IN DESMOPLASTIC INFANTILE GLIOMA**
Guinle, M., Nettnin, E. A., Nasajpour, E., Nirschl, J., Garcia, C. A., Vogel, H., Yeom, K., Prolo, L., Petritsch, C.
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- **UNDERSTANDING OLIGODENDROCYTE PROGENITOR CELL CONTRIBUTION TO GLIOMAGENESIS USING A NOVEL PHARMACOGENETIC ABLATION TOOL**
Xing, L., Lancero, H., Hambardzumyan, D., Merson, T., Petritsch, C.
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- **BIOPROCESSING OF SURGICAL PEDIATRIC BRAIN TUMOR SPECIMENS FOR GENOME-GUIDED PERSONALIZED DRUG TESTING**
Nasajpour, E., Tran, C., Garcia, C., Lyle, G., Guinle, M., Bagley, B. A., Lancero, H., Gibson, E. E., Schouten, T., Mahaney, K., Vogel, H., Learned, K., Vaske, et al
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Xing, L., Merson, T., Petritsch, C.
OXFORD UNIV PRESS INC.2023
- **THERAPY-INDUCED CHANGES BY BRAF AND MEK INHIBITORS IN BRAF V600E-MUTATED GLIOMA MODELS PROVIDE POTENTIAL NOVEL THERAPEUTIC OPPORTUNITIES**
Park, J., Lancero, H., Nasajpour, E., Garcia, C., Prolo, L., Grant, G., Petritsch, C.
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- **MICROGLIA REPLACEMENT CHANGES THE TRANSCRIPTIONAL PROFILE OF TUMOR ASSOCIATED MYELOID CELLS IN MURINE MODELS OF BRAIN MALIGNANCIES**
Mader, M., Rodrigues, A., Chernikova, S., Wong, Z., Wang, Y., Petritsch, C., Wernig, M., Gephart, M.
OXFORD UNIV PRESS INC.2022: 290
- **ANALYSIS OF EXPRESSION SIGNATURES OF MATCHED PRIMARY GASTROINTESTINAL CANCER AND BRAIN METASTASES USING NANOSTRING NCOUNTER TECHNOLOGY**
Cheko, A., Burnside, G., Vogel, H., Gephart, M., Petritsch, C.
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- **BIOPROCESSING OF SURGICAL PEDIATRIC BRAIN TUMOR SPECIMENS FOR GENOME-GUIDED PERSONALIZED DRUG TESTING**
Nasajpour, E., Lyle, G., Lancero, H., Garcia, C., Learned, K., Gibson, E., Tran, C., Schouten, T., Vogel, H., Mahaney, K., Prolo, L., Vaske, O., Grant, et al
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- **COMBINATION OF MAPK PATHWAY INHIBITORS AND IMMUNE CHECKPOINT BLOCKADE IN BRAF-MUTANT HIGH-GRADE GLIOMA**
Park, J., Grossauer, S., Wang, W., Daynac, M., Pitteri, S., Monje, M., Grant, G., Petritsch, C.
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- **Clinically relevant MAPK pathway inhibition reverses stem cell fate defects and sensitize BRAF mutant glioma to immune modulatory therapies.**
Petritsch, C. K., Grossauer, S., Wang, W., Barrette, A., Park, J.
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- **MAPK PATHWAY INHIBITION SENSITIZES TO IMMUNOTHERAPY IN BRAF-MUTANT GLIOMAS**
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- **GENERATION OF NOVEL MOUSE MODELS FOR BRAF V600E MUTANT GLIOMAGENESIS TO GAIN MECHANISTIC INSIGHTS INTO TUMOR FORMATION AND PROGRESSION**
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- **THE IMMUNE MICROENVIRONMENT IN LOWER GRADE GLIOMAS**
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- **A NOVEL BRAF V600E LOW-GRADE GLIOMA MOUSE MODEL HIGHLIGHTS EXOMIC AND TUMOR IMMUNE ALTERATIONS AND DIFFERING THERAPEUTIC RESPONSES IN LOW- AND HIGH-GRADE GLIOMAS**
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- **DETERMINING THE NEUROANATOMICAL AND CELLULAR ORIGIN OF BRAEV600E MUTANT CDKN2A DELETED GLIOMAS AND MECHANISMS OF TRANSFORMATION BY BRAEV600E EXPRESSION IN "FRANSGENIC MICE**
Sugiarto, S., Lerner, R. G., Reichhoff, B., McMahon, M., James, C., Petritsch, C. K.
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- **GAINING A MECHANISTIC UNDERSTANDING OF THERAPY EVASION FROM DUAL MAPK PATHWAY INHIBITION IN A SYNGENIC BRAFV600E MUTANT CDKN2A DELETED MOUSE MODEL TO PREEMPT RESISTANCE IN PATIENTS WITH BRAFV600E MUTANT E PEDIATRIC GLIOMA**
Wang, W., Grossauer, S., Koeck, K., Simonds, E., Lu, E., Bergers, G., Weiss, W., Petritsch, C.
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