CURRICULUM VITAE **Deo Prakash Pandey**

PERSONAL INFORMATION

Family name, First name: Pandey, Deo Prakash
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CURRENT AND PREVIOUS POSITIONS

2018-	Project Group Leader, Dept. of Molecular Microbiology, Oslo University Hospital, Norway
2017-2017	Researcher, Dept. of Molecular Microbiology, Oslo University Hospital, Norway
2011-2017	Postdoctoral fellow, Laboratory of Prof. Kristian Helin Biotech Research and Innovation Centre (BRIC), University of Copenhagen, Denmark

EDUCATION

2005-2010	PhD: **Disputation date:** 15.10.2010. *Supervisor: Didier Picard* Dept. of Cell Biology, University of Geneva, Switzerland
2003-2005	Master in Biotechnology. *Supervisor: Kenn Gerdes* Dept. of Biochemistry & Molecular Biology, University of Southern Denmark, Denmark
1997-2003	Bachelor in Technology, Chemical Engineering, Indian Institute of Technology, Kanpur

RESEARCH EXPERIENCE

May 2017 – present: Independent researcher at the dept. of Microbiology, Rikshospitalet, Oslo after acquiring the Helse Sør-Øst researcher fellowship. My research focus is to identify and characterize novel functional dependencies, with a focus on epigenetic and transcriptional processes, required for Glioblastoma.

Jan 2011 – March 2017: Postdoctoral fellow in the lab of Prof. Kristian at the University of Copenhagen. I setup mouse models of glioblastoma multiforme (GBM), the most prevalent and aggressive of primary brain tumors and performed in vivo and in vitro shRNA screens to identify epigenetic factors required for the tumorigenic process in GBM. After validating a number of hits, I characterized their functional requirement in GBM. I collaborated with another postdoc in the Helin group, to model pediatric gliomas and identified Ezh2 inhibition as a potential therapeutic strategy to target them, leading to a publication in Nature medicine in 2017.

Oct 2005 – Dec 2010: Graduate student in the group of Prof. Didier Picard at the University of Geneva. During my PhD, I worked on two major aspects of estrogen signaling. 1) We characterized estrogen signaling through GPR30 and identified the GPR30 signaling induced transcriptional network regulates migration and proliferation of breast cancer cells. 2) We showed that miR-22 represses estrogen signaling by directly targeting the ER α mRNA. Additionally, genetic screens in *Saccharomyces cerevisiae* were performed to identify factors that are necessary for the proper function of ERα. The resulting candidates led to publications in Molecular Cell, 2016 and in Nature Communications, 2019.