

## AVAILABLE POSITIONS

<b>Principal Investigator</b>	<b>Blagoje Soskic</b>
<b>Affiliation</b>	Human Technopole, Milan
<b>Title of the proposed project</b>	Assembloid platform to model neuro-onco-immune interactions driving NF1 glioma pathology
<b>Short description of the project</b>	<p>Neurofibromatosis type 1 (NF1) is an autosomal dominant cancer predisposition disorder that primarily affects the central and peripheral nervous system. Individuals with NF1 face high risk of developing high-grade gliomas, which substantially reduce life expectancy. The condition is caused by mutations in the NF1 tumor suppressor gene. Glioma progression in NF1 is strongly influenced by interactions with the tumor microenvironment. Immune cells play a significant role in this process. Microglia, for example, support tumor growth by impairing immune surveillance and failing to clear necrotic debris. Additionally, RNA sequencing of NF1-associated gliomas has revealed infiltration by CD8+ T cells, particularly in tumors with high immune activity, suggesting a possible role for cytotoxic T cells in controlling tumor growth.</p> <p>To address key gaps in our understanding of NF1 glioma biology, the student will utilize a 3D NF1 glioma model co-cultured with immune cells. This system will enable the investigation of both tumor–neuron and tumor–immune interactions, with a focus on how the tumor microenvironment drives glioma progression. Through this approach, the project aims to uncover mechanistic insights into NF1 glioma development and establish a platform for future therapeutic screening and discovery. The student will combine various stem and immune cell culture approaches, cutting-edge genomic technologies, advanced imaging, and high-dimensional data analysis. This interdisciplinary research offers an exciting opportunity for a PhD student to gain expertise in both experimental and computational immuno-oncology.</p> <p>This project is a close collaboration between the Kalebic and Soskic labs.</p>
<b>Main research area for the project</b>	Cancer Biology
<b>Second research area for the project</b>	Immunology
<b>3 key words for project</b>	Neurofibromatosis, organoids, immune-oncology
<b>Main topic/s of the lab</b>	immunology, genomics, immunogenetics
<b>Short description of the lab activity</b>	<p>T and B cells are the two key players of adaptive immunity and their interaction and coordinated activation is critical for host defence. Their collaboration underpins the successful development of antibodies which have multiple roles in immune protection. Therefore, it is not surprising that impaired T and B cell interaction results in high susceptibility to infection and immune deficiency. On the other hand, dysregulation of T and B cell responses may lead to immune-mediated diseases such as multiple sclerosis (MS) and systemic lupus erythematosus (SLE).</p>

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	<p>The overarching aim of my group is to gain fundamental insights into the molecular and genetic control of T-B interaction, B cell activation and antibody diversification. We integrate the power of immunological and cellular readouts with cutting-edge genomic tools to dissect the pathways that regulate B cell activation, antibody production and their genetic variability.</p> <p>In this project we teamed up with the Kalebic group (neurobiology) to model neuro-onco-immune interactions in NF1 gliomas.</p>
<b>Recent bibliography</b>	<p>Demela P, Esposito L, Marchesan P, Bolognini D, Giacopuzzi E, Ricciardelli E, Ferrari P, Bombelli S, Peano C, Prati D, Valenti L, Soskic B. Competing gene regulatory networks drive naive and memory B cell differentiation. bioRxiv. 2025 Jun 8. doi:10.1101/2025.06.04.657836.</p> <p>Demela P, Pirastu N, Soskic B. Cross-disorder genetic analysis of immune diseases reveals distinct gene associations that converge on common pathways. Nat Commun. 2023 May 12;14(1):2743.</p> <p>Lynall ME, Soskic B, Hayhurst J, Schwartzentruber J, Levey DF, Pathak GA, Polimanti R, Gelernter J, Stein MB, Trynka G, Clatworthy MR, Bullmore E. Genetic variants associated with psychiatric disorders are enriched at epigenetically active sites in lymphoid cells. Nat Commun. 2022 Oct 15;13(1):6102.</p> <p>Soskic B*, Cano-Gamez E*, Smyth DJ, Ambridge K, Ke Z, Matte JC, Bossini-Castillo L, Kaplanis J, Ramirez-Navarro L, Lorenc A, Nakic N, Esparza-Gordillo J, Rowan W, Wille D, Tough DF, Bronson PG, Trynka G. Immune disease risk variants regulate gene expression dynamics during CD4+T cell activation. Nat Genet. 2022 Jun;54(6):817-826.</p> <p>Cano-Gamez E*, Soskic B*+, Roumeliotis TI, So E, Smyth DJ, Baldrighi M, Willé D, Nakic N, Esparza-Gordillo J, Larminie CGC, Bronson PG, Tough DF, Rowan WC, Choudhary JS, Trynka G+. Single-cell transcriptomics identifies an effectorness gradient shaping the response of CD4+ T cells to cytokines. Nat Commun. 2020 Apr 14;11(1):1801.</p>
<b>Group composition</b>	<p>2 Postdocs 2 PhD Students 1 MSc Student 1 Senior Technician</p>
<b>Institutional page link</b>	<p><a href="https://humantechnopole.it/en/research-groups/soskic-group/">https://humantechnopole.it/en/research-groups/soskic-group/</a></p>