



High resolution dissection of non-coding determinants of disease

Fact Sheet

Project Information

B-ALLeLes

Grant agreement ID: 101061151

DOI

[10.3030/101061151](https://doi.org/10.3030/101061151)

Project closed

EC signature date

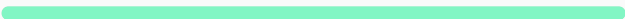
8 August 2022

Start date

1 November 2022

End date

31 October 2024



Funded under

Marie Skłodowska-Curie Actions (MSCA)

Total cost

No data

EU contribution

€ 183 600,96

Investment in EU policy priorities

Digital agenda	☐	Clean air	☐
Artificial Intelligence	☐	Climate action	☐
Biodiversity	☐		

Coordinated by

ST. ANNA
KINDERKREBSFORSCHUNG
GMBH
 Austria

Objective

While mutations at protein-coding sequences have been extensively characterized, the functional role of disease-associated non-coding variants remains largely elusive.

In fact, germline non-coding variants are often associated with increased risk of childhood cancers. In B-cell derived acute lymphoblastic leukemia (B-ALL), the most common type of cancer in children, non-coding sequence variation at lineage-specific genes, such as IKZF1, GATA3 and CEBPE is associated with disease. Interestingly, the strongest risk factor maps at ARID5B, a DNA-binding protein described to promote the removal of repressive histone marks. Still, the role of ARID5B in leukemia and haematopoiesis remains largely uncharacterized. Thus, we hypothesize that non-coding variants associated with B-ALL affect the activity of distal regulatory elements, modulating the expression of ARID5B and other B-cell lineage-specific genes (objective 1). We further postulate that ARID5B promotes the de-repression of lineage-specific genes (objective 2), playing a crucial role in B-cell development and B-ALL initiation and progression (objective 3). To test these hypotheses, I will develop a novel CRISPR screen approach to dissect ARID5B enhancers at single nucleotide resolution (work package 1). I will use a combination of genomics and transcriptomics methods after acute and prolonged degradation of ARID5B to elucidate the molecular function of ARID5B in B-ALL (work package 2). Finally, I will use mouse models and focus on the physiological relevance of ARID5B in B-cell development and leukemia initiation and progression, in vivo. (work package 3). Together, I will dissect the role of non-coding variants at leukemia-associated loci, characterize the molecular function of ARID5B and elucidate the pathophysiological relevance of ARID5B.

Fields of science (EuroSciVoc)

[natural sciences](#) > [biological sciences](#) > [biochemistry](#) > [biomolecules](#) > **[proteins](#)**

[natural sciences](#) > [biological sciences](#) > [genetics](#) > **[mutation](#)**

[medical and health sciences](#) > [clinical medicine](#) > [oncology](#) > **[leukemia](#)**



Keywords

[non-coding variants](#)

[epigenetics](#)

[CRISPR screens](#)

[hematopoiesis](#)

[leukemia](#)

[enhancers](#)

[GWAS](#)

Programme(s)

[HORIZON.1.2 - Marie Skłodowska-Curie Actions \(MSCA\)](#)

MAIN PROGRAMME

Topic(s)

[HORIZON-MSCA-2021-PF-01-01 - MSCA Postdoctoral Fellowships 2021](#)

Call for proposal

[HORIZON-MSCA-2021-PF-01](#)

[See other projects for this call](#)

Funding Scheme

[HORIZON-TMA-MSCA-PF-EF - HORIZON TMA MSCA Postdoctoral Fellowships - European Fellowships](#)

Coordinator



ST. ANNA KINDERKREBSFORSCHUNG GMBH

Net EU contribution

€ 183 600,96

Total cost

No data

Address

ZIMMERMANNPLATZ 10

1090 Wien

 **Austria** 

Region

Ostösterreich > Wien > Wien

Activity type

Research Organisations

Links

[Contact the organisation](#) 

[Participation in EU R&I programmes](#) 

[HORIZON collaboration network](#) 

Partners (1)



PARTNER 

CEMM - FORSCHUNGSZENTRUM FUER MOLEKULARE MEDIZIN GMBH

 Austria

Net EU contribution

€ 0,00

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Region

Ostösterreich > Wien > Wien

Activity type

Private for-profit entities (excluding Higher or Secondary Education Establishments)

Links

[Contact the organisation](#)  [Website](#) 

[Participation in EU R&I programmes](#) 

[HORIZON collaboration network](#) 

Total cost

No data

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