

THE PATHOBIOLOGY OF PEDIATRIC B-CELL PRECURSOR ACUTE LYMPHOBLASTIC LEUKEMIA

1. *JAK2* mutated acute lymphoblastic leukemic cells are sensitive to JAK inhibitors, but a clinical implementation of these inhibitors is unlikely. (*This thesis*)
2. High expression of *STAP1* is a hallmark of *DUX4*-rearranged acute lymphoblastic leukemia cases, but not a likely target for precision medicines. (*This thesis*)
3. The oncogenic driver determines the relation between copy number alterations in B-cell development genes and drug resistance in acute lymphoblastic leukemia. (*This thesis*)
4. The increased survival of acute lymphoblastic leukemia induced by mesenchymal stromal cells is not affected by the source of these stromal cells. (*This thesis*)
5. Acute lymphoblastic leukemia cells enforce gene expression changes in mesenchymal stromal cells, which coincide with an increased survival of acute lymphoblastic leukemia cells. (*This thesis*)
6. Genome-wide association studies identify additional inherited polymorphisms that are not only associated with the response to treatment, but also with the risk of leukemic transformation, opening the way for the development of potential preventive measures. (*Pui, Seminars in hematology, 2013*)
7. It is likely that *IKZF1* deletions, which are secondary abnormalities, are also a “secondary” marker of poor outcome rather than being a key independent prognostic biomarker. (*Moorman, Haematologica, 2016*)
8. Drugs that fail in animal trials will not necessarily fail in human trials. (*Vogelstein, Science, 2013*)
9. Pathway functions are different, depending on the organism, cell type, and precise genetic alterations in that cell. (*Grueneberg, Proceedings of the National Academy of Sciences of the United States of America, 2008*)
10. Recognition of the complexity of the molecular alterations underlying the development of resistance to targeted therapeutics is necessary to understand the basis of tumor cell survival and clinical progression during therapy, as well as to design combinatorial and non-cross resistant therapeutic strategies. (*Rotow, Nature cancer reviews, 2017*)
11. Research is to see what everybody else has seen, and to think what nobody else has thought. (*Albert Szent-Gyorgyi*)