PI3K signaling and adherens junctions in invasive lobular breast cancer

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Propositions

associated with the thesis

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1. Our finding that the adaptive immune system determines the efficacy of therapeutic mTOR inhibition in mouse invasive lobular carcinoma underlines the importance of immunocompetent preclinical animal models (chapter 4 of this thesis).
2. Comparing treatment-resistant tumor tissue with biopsies from tumors that are responding to therapy is essential, if we are to understand how tumors acquire resistance to treatment (chapter 4 of this thesis).
3. Tumor progression of E-cadherin-deficient cells can be driven by various events including inactivation of p53, but the classic histologic type of ILC can be much more consistently induced by hyperactivation of PI3K signaling (chapter 5 of this thesis).
4. p120 is a suppressor of breast cancer invasion and metastasis, and loss of p120 leads to anoikis resistance and hypersensitization of growth factor receptor signaling (chapter 6 of this thesis).
5. PI3K signaling is an important driver of invasive lobular breast cancer and a very druggable pathway, which increases our hope for better treatment options.
6. Artificial intelligence will be likely to support diagnosis, prediction of therapeutic response and prognosis of lobular breast cancer.
7. We do *in vivo* experiments because all organ systems are relevant in how disease works. That means we have to actively study all organ systems especially in a new animal model, and not only the tissue of our primary interest.
8. The most powerful staining method for tissues is H&E.
9. In order to optimize the efficiency and quality of experiments involving live animals, institutes should provide scientists with full-service support from experts and research facilities.
10. Medical doctors and veterinarians can do a better job if they are interested in each other's work.
11. The most underestimated task in life is to "just do your best".