Uncovering vulnerabilities in triple-negative breast cancer

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Stellingen

behorende bij het proefschrift

Uncovering vulnerabilities in triple-negative breast cancer

1. FRET-based signaling reporters not only substantially contribute to monitoring the efficacy of kinase drugs, but also to the further understanding of their mode-of-action in relation to crosstalk with well-defined signaling components in cancer, including ERK and AKT signaling. **This thesis.**
2. Src family member FYN confers TNBC resistance against EGFR kinase-targeted inhibition via negatively regulating EGFR/PI3K/AKT signaling. **This thesis.**
3. Polypharmacology-based multi-kinase targeted inhibition shuts down the crosstalk among receptors as well as mTOR pathway within signaling networks in the resistant scenario. **This thesis.**
4. ASAP1 is an amplification-dependent driver gene promoting TNBC cell proliferation. **This thesis.**
5. The cancer dependency map will facilitate the prioritization of therapeutic targets. **Tsherniak, Cell, 2017.**
6. Image-based phenotypic screening approaches can provide high-throughput in vitro readouts of key parameters that represent commonly desired properties of anticancer therapies. **Conway, Nat Rev Cancer, 2014.**
7. Combination therapy with rapamycin or rapalogs has become a promising approach, as various strategies have the potential of improving drug efficacy by inhibiting multiple targets either activated by removal of feedback loops or involved in parallel pathways. **Li, Cell Metab, 2014.**
8. Genes that are found in amplified regions and are highly expressed are likely drivers of important “hallmarks” of malignancy and represent critical candidate tumor addictions. **Patel, Nat Commun, 2018.**
9. Hardly can progress be made without facing the difficulties.
10. A banana kick bypasses, but rarely scores.

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