Propositions to the thesis:

Molecular and clinical implications of *IDH1* and *EGFR* mutations in gliomas

1. Lower grade IDH-mutated gliomas, both astrocytomas and oligodendrogliomas, can be kept alive in culture and may become an option as model systems for personalized medicine (this thesis).

2. CNS-specific expression of IDH1 mutant alone cannot initiate gliomagenesis in the zebrafish model system (this thesis).

3. IDH1 inhibits a putative tumor suppressor MUL1 and leads to a deficiency in the TNFα-induced growth arrest (this thesis).

4. Different EGFR mutations have distinct oncogenic function therefore should be treated differently (this thesis).

5. *EGFRvIII* status can vary between primary and recurrent glioblastomas (this thesis).

6. Decreasing D2HG levels by an IDH inhibitor did not affect the tumorigenic properties of chondrosarcoma cell lines (Suijker et al., Oncotarget, 2015).

7. Instead of relying exclusively on big data measurements of initial conditions, we should also acquire highly actionable functional information by perturbing viable primary tumor cells from patients with cancer (adapted from Letai et al., Nature Medicine, 2017).

8. There is not, and will not be, a magic bullet or penicillin equivalent for cancer (Greaves et al., BMC Biology, 2018).

9. Molecular characterization based on genomic, proteomic as well as transcriptomic analysis will provide clinicians the ability to rationally select drugs with actionable targets for each patient (Bush and Butowski, Curr Oncol Rep, 2017).

10. Recent advances in single-cell RNA-seq have led to novel insights in cancer development, progression, metastasis, and drug-resistance, that were previously “veiled” by the mixing of cells intrinsic to standard bulk-sequencing experiments (Müller and Diaz, Front. Genet. 2017).

11. All models are wrong, but some are useful (Box and Draper, 1987).