Propositions

1) Toxicity is the inability of cellular defense and adaptation mechanisms to repair damage and consequently restore homeostasis. (this thesis)
2) Quantitatively monitoring the dynamics of a limited set of cellular stress response pathways will allow the prediction of compound-induced toxicity. (this thesis)
3) There will never be one single perfect model for toxicity prediction but rather a large set of models each optimized for its own specialized purpose. (this thesis)
4) Single cell analysis allows fine tuning of sensitivity versus dynamic range. (this thesis)
5) Evolution defines that there are a limited number of cellular damage types and thus a limited number of evolutionary-optimized unambiguous repair pathways. (Cipponi et al. 2014)
6) Due to the fact that upregulation of Nrf2 in tumors is a relatively common phenotypic change and upregulation of Nrf2-regulated proteins of more than 2-fold with respect to primary human hepatocytes was found in HepG2 cells (Sison-Young et al. 2015), HepG2 cells are an excellent reporter cell line for oxidative stress.
7) The optimal in vitro model for safety testing depends on the scientific question being asked. (Godoy et al. 2013)
8) Extensive animal testing does not provide a mechanistic understanding of toxicity, and knowledge concerning adverse risks to humans is still inadequate. (Shukla et al. 2010).
9) Complex dose-response curves and large cell-to-cell variability one frequently observes reinforce the utility of unbiased multidimensional characterization of drug effects over wide ranges of doses. (Perlman et al. 2004)
10) The science of toxicology is a difficult one; it is based on compounds we only understand based on empirical observations and on biology we barely scraped the surface of.
11) Science would benefit greatly from a complete depository of publications of failed experiments.