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Stellingen

behorende bij het proefschrift

Dynamics of TNFAlpha signaling and drug-related liver toxicity

1. Concurring signaling events induced by DILI compounds affect the final outcome of TNFα-induced NF-κB target gene expression (this thesis)

2. HepG2 cells are able to reflect many inflammatory signaling characteristics and can be used in predictive screening approaches (this thesis)

3. CDK12 polymorphisms could form a genetic basis in which certain individuals are more sensitive to specific drug-induced stress responses (this thesis)

4. The development of an ever increasing database containing detailed signaling-based features linked to chemical exposure will ultimately aid in the safety evaluation and early (DILI) prediction of new drugs and chemicals. (this thesis)

5. Neither the drug-specific nor the patient-related factors per se would typically pose a risk for DILI; much more it is the combination of both (based on Funk and Roth, Arch of Toxicology, 2017)

6. TNF is a central player within a complicated network of cytokines and it regulates not only pro-inflammatory responses but also processes as diverse as cellular communication, cell differentiation and cell death. (based on Brenner et al, Nature Review Immunology, 2015)

7. Multiple layers of gene regulation work in concert to generate a diverse set of gene programs while using only a limited number of transcription factors (based on Hao and Baltimore, PNAS, 2013)

8. Isolating hepatocytes from their physiological environment in the liver causes alterations in cell physiology resembling those in inflammatory liver diseases (based on Godoy et al, Arch. of Toxicology, 2016)

9. Cell toxicologists need more information from patients with idiosyncratic DILI, as they are the reality that our models try to approximate.

10. Full transparency of the scientific process from start to end will restore our trust in the reliability and reproducibility of published results.