Stellingen

Behorend bij het proefschrift:

The influence of low dystrophin levels on disease pathology in mouse models for Duchenne Muscular Dystrophy

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1. It is important to develop a functional test regime that does not interfere with the disease progression in animal models (*This thesis*).

2. Motor function of *mdx/utrn* $^{+/-}$ mice is worse than that of *mdx* mice making it a better model for testing potential therapeutic compounds (*This thesis*).

3. To monitor disease progression in DMD patients, MMP-9 is a better serum biomarker than CK (*This thesis*).

4. Higher dystrophin levels are needed to prevent muscle damage than to improve vitality and muscle function (*This thesis*).

5. Even though none of the DMD mouse models exactly mimics the disease, their availability has catalyzed developments in pre-clinical research.

6. The uniformity of dystrophin expression throughout a given muscle is likely to be more important than the overall level of expression.

7. Dystrophin restoration solely in skeletal muscle may exacerbate heart pathology due to increased activity affecting the workload for the heart.

8. Combination therapies restoring both primary and secondary defects might be more successful than these only targeting the primary defect.

9. The use of the natural instincts of mice in designing experiments facilitates their cooperation thereby increasing the reliability of the outcomes.

10. Especially in animal experiments, logistics, including the timely replenishing of stock materials is essential to prevent delays in experiments of colleagues using the same protocols.

11. Interesting hypotheses arise and evolve into research projects especially when multiple disciplines mingle together during social events.