

## Stellingen behorende bij het proefschrift

### 'Bone Aging in DNA Repair Deficient Trichothiodystrophy Mice'

The finding that the bone phenotype of trichothiodystrophy mice is not due to a developmental defect (this thesis) argues that the same phenotype in extremely short-lived DNA repair mouse models is in fact also not a developmental defect per sé.

The trichothiodystrophy mouse mutant is an excellent model for the study of bone fragility as well as periosteal apposition.

*This thesis*

It is most likely that a defect in nucleotide excision repair is responsible for the accelerated aging phenotype observed in the long bones of trichothiodystrophy mice.

*This thesis*

Trichothiodystrophy mice exhibit segmental aging even within the skeleton.

*This thesis*

In contrast to the normal aging phenotype, trichothiodystrophy mice do not show accumulation of fat in the liver or abdomen.

*This thesis*

The study of nature's experiments is of special value; and many lessons which rare maladies can teach could hardly be learned in other ways.

*Dr. William Harvey (1578-1657) paraphrased by Sir Archibald Garrod in the Lancet, May 26, 1928*

Distinguishing between innocent variants and pathogenic mutations is a growing problem in DNA-diagnostics.

Commercial genetic testing through the internet raises concerns about the access to reliable and unbiased health information.

*Community Genetics, 2003*

Several important successes in treating severe inherited diseases suggest that gene therapy is making a come back.

*Science, November 6, 2009*

Familial adenomatous polyposis may be a ciliopathy.

*The Lancet Oncology, July 1, 2009*

Bicarbonate of soda has an explosive effect on the rising of carrotcake.