Vitamin D deficiency in humans is associated with a reduction in maximal muscle force and atrophy of type II fibres. Furthermore, it has been shown that vitamin D deficiency changes muscle contractile properties; the half relaxation time is prolonged in rats and chickens. Further support for effects of vitamin D on skeletal muscle is the presence of the vitamin D receptor in skeletal muscle tissue.

The aim of this thesis is to obtain more insight in the effects of vitamin D on the structural and contractile properties of skeletal muscle. Elevated blood serum levels of active vitamin D (1,25D) were obtained in old rats via alfacalcidol supplementation. As a result the rats ate less, which may have contributed to a reduction in maximal force and muscle mass. The supplementation resulted in a leftward shift of the force-frequency relation. The reduction in muscle mass was caused by atrophy of type IIb and IIx fibres, which may be related to the observed increased expression of MuRF1, one of the proteins of the ubiquitin pathway.

Low vitamin D levels were obtained with a vitamin D deficient diet. The deficiency did, in contrast to our hypothesis, not result in a reduction in muscle mass and maximal force.

The finding of a negative influence of addition of 1 nM 1,25D in the medium on maximal force and 10%-relaxation time of Xenopus muscle fibres in culture and an unchanged fibre CSA is a further indication that the role of vitamin D on skeletal muscle structure and function is complex. The results suggest that vitamin D itself has not the important direct effects on muscle mass and contractile properties as was thought. Since vitamin D regulates calcium, phosphorus and parathyroid hormone serum levels, these factors might be more important in the regulation of muscle mass and function.