COPD is a fourth leading cause of death and a fifth leading cause of disability worldwide. Development of cachexia syndrome and a consequential deterioration of peripheral muscle function significantly associates with increased mortality and reduced life quality in COPD.

An important step towards better management of muscle abnormalities and dysfunction in COPD is to reveal molecular alterations underlying their development and progression. Development of animal models and in-vitro platforms, such as in this study, represents a valuable strategy and a powerful tool to get a better insight into possible mechanisms and pharmacological targets, which can in turn lead towards better treatment of the human disease. This thesis might give a miniature contribution to this goal.