VALIDATION OF THE FORCELOSS FRAMEWORK FOR THE DIFFERENTIAL DIAGNOSIS OF DYNAPENIA

Francesca Bottin (1,2), Giorgio Davico (1,2), Domitille Princelle (1,2), Marco Viceconti (1,2)

1. *Department of Industrial Engineering, Alma Mater Studiorum - University of Bologna, Italy;*
2. *Medical Technology Lab, IRCCS Istituto Ortopedico Rizzoli, Italy*

Introduction

The loss of muscle force, or dynapenia, is a natural consequence of aging that may be accelerated or aggravated by ongoing pathological processes [1]. Dynapenia is typically associated with a reduced quality of life, and an early diagnosis is important. However, the identification of the primary cause of dynapenia, and the consequent clinical management of the patient, is challenging. Complementing current clinical measures with computer modelling and simulations (CM&S) techniques may facilitate this process. Personalized musculoskeletal (MSK) models and simulations, informed by experimental data, may be used as a falsification tool to test different clinical hypotheses, and – by exclusion – to identify the primary cause of dynapenia. To this purpose, we developed the ForceLoss framework, which we presented at the last ESB congress in Porto. In this abstract, we present the validation of the ForceLoss framework, which has so far been conducted using a public dataset [2].

Methods

Data from three editions of the Knee Grand Challenge competitions [2] were used to perform the study. Dynamometry and electromyography (EMG) data recorded while the subjects performed a knee extension task (maximal voluntary isometric contraction, MVIC) were processed to extract the maximal torques and the EMG linear envelopes. Image-based MSK models were then developed, following the INSIGNEO pipeline [3], using the available bony geometries. The models were placed in a sitting position (with hip and knee flexion angles set to 80° and 90° respectively) and simulations were run hypothesizing optimal muscle control (classical static optimization approach), in OpenSim. An external knee flexion torque was imposed, and iteratively increased till failure. To test different clinical scenarios, the level of personalization of the models was gradually increased, first by scaling the maximal isometric force values of the quadriceps muscles with the physiological cross-sectional areas extracted from medical images, then by setting the maximal activation level of the vastii and the rectus femoris muscles to the corresponding EMG linear envelopes (maximal value).

Results

For one subject, the first level of model personalization was sufficient to estimate maximal knee extension values within a 10% margin from corresponding experimental MVIC data (157 vs 146 Nm). For the other two cases, further limiting the maximal activation levels to those observed *in vivo* was necessary to achieve a good agreement between experimental and simulated data (substantial reduction from 126 to 69 Nm, comparable to the experimental values, 69 Nm).

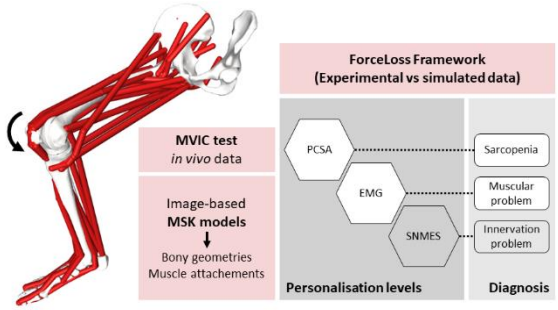

Figure 1: The ForceLoss framework: image-based musculoskeletal (MSK) models are employed to predict the maximal knee extension torques. At each step, the level of personalization of the models is increased. The last step, requiring electrical stimulation (SNMES) data, was not performed. MVIC = maximal voluntary isometric contraction, PCSA = Physiological cross-sectional area, EMG = electromyography.

Discussion

Based on these results we may conclude that all subjects were affected by a form of sarcopenia, which was accompanied by some neuromuscular problem in two cases. This suggests that the proposed framework may be suitable to conduct the differential diagnosis for dynapenia, through the combination of experimental measures and CM&S methods. To confirm these findings, we will test the ForceLoss framework on a dataset currently under construction. The dataset will be representative of a healthy adult population and of patients with osteoarthritis who will undergo a total knee arthroplasty.

References

1. Clark and Manini, J Gerontol A Biol Sci Med Sci, 63:829-34, 2008.
2. Fregly et al, J Orthop Res, 30:503–513, 2011.
3. Modenese et al, J Biomech, 73:108–118, 2018.

Acknowledgements

This study was partially supported by various grants, including the EU H2020 Mobilise-D project (topic IMI2-2017-13-07, grant ID 820820), the EU H2020 In Silico World project (grant ID 101016503), and the ForceLoss project that has received funding from the Italian Ministry of Health (Ricerca finalizzata RF-2019-12369960).