

Modeling ageing in skeletal muscle tissue

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Abstract

One of the important changes associated with human ageing is the so called *sarcopenia*, which is defined as the progressive loss of skeletal muscle mass and strength. According to [2], this syndrome affects more than 50 millions people today and it will affect more than 200 millions in the next 40 years. There is still no generally accepted test for its diagnosis and many efforts are made nowadays by the medical community to better understand this syndrome. Having this in mind, a mathematical model of sarcopenic muscle can be a valuable resource.

Skeletal muscle tissue is one of the main components of the human body. The muscular fibres are organized in fascicles, containing a concatenation of millions of sarcomers, which are the fundamental unit of the muscle which produces force and movement (i.e. *activation*). Connective tissue, which is essentially isotropic, fills the spaces among the fibres.

Focusing our attention only on steady properties, we model the skeletal muscle tissue as a continuum hyperelastic material [5]. The alignment of the muscular fibers and the high water content of the tissue suggest the assumptions of transverse isotropy and incompressibility. In the model that we propose, there are two main constitutive prescriptions: one for the hyperelastic energy when the tissue is not activated (*passive energy*) and one for the activation. Moreover, in order to describe sarcopenia, the loss of performance and the loss of mass are obtained by two different methods.

As far as the passive part is concerned, we assume an exponential stress response of the material, which is customary in biological tissues [3].

Coming to the activation, we follow the *active strain* approach [1], where the extra energy produced by the activation mechanism is encoded in a multiplicative decomposition of the deformation gradient in an elastic and an active part.

In addition, the loss of activation is represented by a damage parameter d which reduces the active part of the stress by a given percentage; while the loss of mass is encoded in

the model by a further multiplicative decomposition of the deformation gradient, where a parameter g measures the fraction of muscle fibers which are not active anymore [4].

Finally, we present some numerical results. We show that the experimental data of [6] on the passive and active stress-strain curves, obtained *in vivo* from a tetanized *tibialis anterior* of a rat, can be well reproduced by our model in the healthy case. Moreover, we discuss the behaviour of aged tissue when g and d increase.

Keywords: Hyperelasticity, activation, ageing, active strain, biomechanics.

References

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