J Appl Physiol 102: 5–6, 2007;
doi:10.1152/japplphysiol.00952.2006.

Invited Editorial

Stretching human muscles makes them stronger

WHEN SKELETAL MUSCLE IS STRETCHED while activated, force increases significantly. After the end of the stretch, force decays rapidly and reaches steady state. This steady-state force is higher than the force produced at the corresponding length during purely isometric contractions. This increase in force observed after the stretch, referred to as steady-state force or residual force enhancement, has been observed since early 1950s (1), but its mechanisms are still a matter of debate. Residual force enhancement has been typically associated with sarcomere nonuniformities and the so-called "popping sarcomeres hypothesis" (5). However, recent studies have shown that the residual force enhancement cannot be explained uniquely by popping sarcomeres (11), and evidence suggests that it may be linked to cross-bridge kinetics and passive elements inside muscle fibers (3).

Residual force enhancement has been observed in a variety of experimental preparations, including myofibrils (11), fibers (2, 9), and isolated muscles (1). Recent studies performed with humans experimented "in vivo" suggest that this phenomenon has important functional implications. These studies show that the residual force enhancement is present in small muscle groups following maximal and submaximal electrical stimulation (4, 10), as well as voluntary contractions (4, 6). The residual force enhancement following electrical stimulation presents characteristics that are similar to those observed during experiments with isolated preparations. However, the residual force enhancement obtained after voluntary activation, and its physiological importance for human muscles, still needs investigation. One of the reasons for such limited knowledge is that the residual force enhancement following submaximal, voluntary contractions depends on the level of muscle activation, and it also varies considerably among individuals (6).

The study performed by Pinniger and Cresswell, published in this issue of the *Journal of Applied Physiology* (7), adds important information regarding the connection between the residual force enhancement and contractions performed in physiological conditions. The authors evaluated the residual force enhancement in large leg muscles of healthy individuals; in this case, the authors measured the joint torques produced around the ankle as an indicative of muscular forces. During the experiments, submaximal plantar flexion and dorsiflexion activation was maintained at ~25% of maximal effort, by providing the subjects with direct visual feedback of the electromyography signals from the soleus or tibialis anterior muscles. During activation, muscles were stretched by changing the ankle angle within their working range (corresponding to the ascending limb of their force-length relationship). Force enhancements of 7 and 12% (expressed as a function of the isometric torques at corresponding lengths) were observed for plantar flexion and dorsiflexion, respectively. For comparisons, residual force enhancement was also examined with electrical stimulation during plantar flexion, which provided an increase in force of 13%.

To gain insight into the mechanisms of force enhancement, the authors measured the torque after the muscles were deactivated, following stretch and isometric contractions. The authors evaluated whether there was a remaining force enhancement in the passive state, referred to as "passive force enhancement." Passive force enhancement has been suggested to originate from titin molecules or slow-detaching cross bridges, and it is believed to contribute to the residual force enhancement (9). Passive force enhancement was observed during dorsiflexion only, but it was not large enough to explain the residual force enhancement.

Altogether, the results presented by Pinniger and Cresswell (7) suggest that the residual force enhancement is a fundamental characteristic of human skeletal muscles contracting in physiological situations, and therefore it may influence the performance of the muscular system during everyday activities. Because the results show a greater residual force enhancement for the tibialis anterior compared with the soleus muscle, the functional implications of this phenomenon may be related to different fiber types. These results, along with results from other investigators (4, 6), open the possibility for future studies aimed at exploiting force enhancement in functional tasks and at determining how it may be affected by acute and chronic situations, like fatigue, physical training, muscle atrophy, and diseases that affect the muscular system.

Studies performed with human muscles provide the fundamental link between findings from controlled experimental situations (e.g., single fibers) and situations that may affect everyday life (e.g., voluntarily contracting human muscles). On the other hand, these studies often present limitations when dealing with cellular and molecular mechanisms. In the case of the paper by Pinniger and Cresswell (7), changes in the ankle angle during plantar flexion and dorsiflexion activation provide a valid indication if muscles are being stretched or shortened, but it makes impossible for the investigators to know the actual length of the muscles. Controlling the level of muscle activation is difficult, and evaluating the contribution of cellular and molecular structures during the experiments is virtually impossible. Therefore, conclusions regarding potential mechanisms are risky. Because the residual force enhancement was observed along the ascending limb of the force-length relationship, and it persisted for a long time after the stretch was ended, the authors conclude that it cannot be caused by popping (unstable) sarcomeres or cross bridges, respectively. Pinniger and Cresswell suggest that force enhancement is associated with an increased stiffness of titin resultant from muscle activation; the passive force enhancement may be taken as an indication that it happens in certain cases. This mechanism would be fiber-type dependent, because titin isoforms are different in slow and fast muscle fibers. Although these suggestions are well founded and carefully discussed in their paper, and are strengthened by other studies performed with single myofibrils and fibers (8, 9), they cannot be safely extrapolated from this study.

Investigation about the physiological implications of the residual force enhancement has started a few years ago. Much research will have to be performed before we understand how residual forced enhancement influences everyday life. During regular activities, contractions are produced mostly during repeated shortening-stretch cycles and with varying levels of activation, instead of prolonged isometric contractions at constant, controlled levels of activation. Nonetheless, Pinniger and Cresswell (7) have approximated their experiments to situa-

tions that may happen during regular muscle activity, and therefore their study represents an important step to understand the connection between force enhancement and muscle performance. As for the mechanism(s) of the residual force enhancement, the history of investigation is not new and has provoked insightful debate, but the question remains: what causes the increase in the steady-state force observed after stretch of activated muscles? The search will certainly continue.

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