Letters to the Editor

Muscle Strength and Oestrogen Status

SIR—The results described by Taaffe et al. [1] concerning muscle strength and oestrogen status of elderly women appear to contradict a study of ours, which they quote, in which we found a 23% decline in adductor pollicis force per cross-sectional area (F/CSA) in women during the first five years after the menopause, who were not taking hormone replacement therapy (HRT), and no such decline in HRT users [2].

We certainly agree with Taaffe et al. that it is important to see if the apparent protective effect of HRT extends to other subjects than those that we studied, and also whether our observations apply to other muscles. In this context it is surprising that they do not cite Rutherford and Jones’s study of 216 women in which a 27% decline in quadriceps F/CSA was found between the third and eighth decades with ‘a particularly large and significant’ decline around the time of the menopause [3].

Taaffe et al. correctly point out the differences between their methods and ours but do not seem to understand the full implications of our differing approaches. We measured maximal isometric force of adductor pollicis using an adaptation of a very well established method [4], and compared it with an anthropometric estimate of the muscle’s CSA which we validated against CT and MRI scan images [5]. Taaffe and co-worker’s measurement of strength, the maximum weight an individual can lift, is indirect and notoriously hard to interpret owing to a necessity for the co-ordinated activation of other muscles to stabilize the body, and the large learning component involved in weight-lifting [6]. Both of these factors are likely to vary between individuals. Their measurement of muscle size is also indirect since they rely on an estimate of total muscle mass (lean body mass). The main problem here is the implicit assumption that the amount of age-related atrophy that has occurred is the same in all muscle groups. That this is probably not the case is one of the main reasons for wishing to see if the effect that we have observed in adductor pollicis is as important in large lower limb muscles where atrophy due to decline in activity may be a relatively more significant cause of weakness [7]. Inter-individual variability of this factor is likely to be particularly problematic in the subjects described by Taaffe et al. because of their wide ranges both of age (65–82 years) and customary activity (‘sedentary to moderately active’).

Taaffe et al. have certainly failed to demonstrate an effect of HRT on muscle strength but we do not believe that their methods are sufficiently rigorous to conclude that the perimenopausal decline in F/CSA in quadriceps, shown by Rutherford and Jones, is not due to oestrogen deficiency, far less to invalidate our observations on adductor pollicis.

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The above letter was referred to Dr Taaffe and colleagues who offer the following reply:

SIR—We thank Bruce et al. for their interest and comments regarding our paper [1], and wish to respond to them.

It was not our intention to attempt to invalidate the findings of Phillips et al. [2], that a perimenopausal decline in adductor pollicis force per cross-sectional area (F/CSA) is prevented by taking hormone replacement therapy. Instead, we found their results intriguing and wished to examine if this effect was also evident for larger muscle groups of the lower body, that are of functional importance for ambulation, in older postmenopausal women. For this purpose we examined maximal isotonic muscle strength by use of the one-repetition maximum (1-RM) method, a common method for muscle strength assessment and one which we and others have used frequently [3–6]. When stringent experimental procedures are employed, as in our laboratory, the results are reliable. As pointed out in our paper, we are cognizant of the differences and implications of our method and that employed by Phillips et al. [2], and together with subject age and years post-menopause, may account for different results obtained between our studies.

Bruce et al. question our measurement of muscle size and state that it is ‘indirect and only an estimate of total muscle mass (lean body mass)’. Obviously, our determination of body composition, by dual-energy X-ray absorptiometry (DXA), and hence lean body mass (LBM) is an indirect method, as are all other methods, including MRI and CT, except for carcas analysis. It should be noted that the DXA measurement of bone-free LBM includes not only muscle mass but also other non-adipose soft tissues. DXA has repeatedly been demonstrated to be an accurate and precise assessment method of regional and total body lean, fat, and bone mineral mass [7–10]. Possibly, Bruce et al. misunderstand the inclusion of this measurement and the way we use LBM. We in no way attempt to relate strength or force to muscle size or CSA, and