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REPLY

Inter-individual variability in the response to maximal eccentric exercise

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We thank Prof. Jones and Prof. Newham for raising useful points of discussion concerning our review on the genetic association with exercise-induced muscle damage (Baumert et al. 2016). As Jones and Newham state, the inter-individual variability in elbow flexor strength loss following eccentric exercise in the study by Newham et al. (1987) ($n = 8$) appears to be low. Other studies, however, have demonstrated large variation in strength loss following maximal eccentric exercise. For example, Nosaka and Clarkson (1996) ($n = 14$) observed a 36–74 % decrement in maximum elbow flexor strength immediately after eccentric exercise, while Miles et al. (2008) ($n = 51$) reported a 31 ± 20 % (mean ± SD) loss in strength. In our laboratory, we have observed an even greater range of responses (0–80 % strength loss) following maximal eccentric contractions in the quadriceps in over 60 untrained participants (unpublished data). Finally, in a study by Clarkson et al. (2005) ($n = 157$), the loss of elbow flexor strength immediately after eccentric exercise was ~49 ± ~2 % (SEM), i.e. apparently similar to that found by Newham et al. (1987), but the standard deviation was ~25 %. Thus, the results from these studies highlight the importance of large sample sizes to get a more complete picture of the inter-individual variability in strength loss following maximal eccentric exercise.

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As discussed in our review (Baumert et al. 2016), there is evidence for a genetic association with the initial phase of exercise-induced muscle damage, i.e. the mechanical disruption of the muscle fibre. For example, alpha-actinin-3, a structural protein linking the actin filaments to the Z-disc in type II skeletal muscle fibres, cannot be produced by *ACTN3* XX homozygotes, and these individuals appear to be more susceptible to exercise-induced muscle damage (Vincent et al. 2010), although this may be dependent on the mode of exercise (Baumert et al. 2016).

In addition to influencing the initial damage response (and therefore indirectly impacting on secondary damage responses), we agree with Jones and Newham that genetic variation is likely to have a direct influence on the inflammatory response (possibly including the expression of heat shock proteins) and muscle regeneration following eccentric exercise. If a person is genetically predisposed to produce a greater inflammatory response, they may or may not demonstrate a quicker recovery, depending on whether the inflammation is indeed increasing the rate of muscle regeneration and recovery or further degrading damaged muscle fibres and prolonging recovery. In our review (Baumert et al. 2016), we categorised the evidence for a genetic association with exercise-induced muscle damage according to the different phases of the damage response, and we believe that the secondary and regeneration phases are of equal importance to the initial damage response.

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