
Downloaded from: http://insight.cumbria.ac.uk/id/eprint/2685/

Usage of any items from the University of Cumbria’s institutional repository ‘Insight’ must conform to the following fair usage guidelines.

Any item and its associated metadata held in the University of Cumbria’s institutional repository Insight (unless stated otherwise on the metadata record) may be copied, displayed or performed, and stored in line with the JISC fair dealing guidelines (available here) for educational and not-for-profit activities provided that

• the authors, title and full bibliographic details of the item are cited clearly when any part of the work is referred to verbally or in the written form
  • a hyperlink/URL to the original Insight record of that item is included in any citations of the work
  • the content is not changed in any way
  • all files required for usage of the item are kept together with the main item file.

You may not

• sell any part of an item
• refer to any part of an item without citation
• amend any item or contextualise it in a way that will impugn the creator’s reputation
• remove or alter the copyright statement on an item.

The full policy can be found here.
Alternatively contact the University of Cumbria Repository Editor by emailing insight@cumbria.ac.uk.
Circulating myostatin is reduced with aging in humans but not altered by short-term, high intensity training

B. T. Elliott, Z. B. Shinwari, Z. Altayar, L. Barrios, G. A. Chaudhary, E. Hanifa, M. Parnell, T. Xenofontos, N. Sculthorpe, P. Herbert, F. Grace, L. Hayes


Poster Communications

Introduction: Ageing involves a loss of muscle mass and function. The rate of decline is associated with negative health outcomes and increased mortality (1). Muscle atrophy is observed at a predictable rate from 30 years of age (2), however maintenance of function is seen in masters athletes > 60 years of age (3). Myostatin acts as a negative regulator of muscle mass (4) and underlies hypertrophy with chronic resistance training (5) and atrophy in chronic conditions (4). Experiment 1: Declared healthy participants (n = 83, 18 - 75 years of age, 36 male, 47 female) were recruited. Body composition, metabolic rate, grip strength and 6-minute walk test were recorded. Venous blood was collected and total myostatin concentration (herein referred to as myostatin) quantified by enzyme-linked immunosorbent assay. Total myostatin was lower in females compared with males (2176.1 [135.3] vs. 2788.7 [180.2] pg.mL\(^{-1}\) [p = 0.007]). Stepwise regression observed that myostatin concentration is best predicted firstly by gender, then by age (r = 0.399, p = 0.02), and was not further improved by the addition of measures of metabolism, muscle mass or function. Experimental 2: A cohort of aged sedentary (SED) males (n = 14; 63.9 [5.6] years of age) and masters athletes (lifelong exerciser [LEX]; n = 10, 61.1 [5.8] years of age) completed 6 weeks of high intensity interview training (HITT). Two way ANOVA suggested no group (SED, LEX) × time (pre, post) interaction on myostatin concentration (p = 0.649), nor a main effect of time (p = 0.757), however there was a trend towards increased myostatin in the LEX group relative to SED (p = 0.083). Discussion: Loss of muscle mass and function occurs at a predictable rate from ~30 years of age, however the rate of loss differs between active and inactive populations. Here we demonstrate that total circulating myostatin decreases as age increases, and differs significantly between males and females. Total circulating myostatin negatively correlates with increasing age, however alterations in myostatin do not appear after short term training interventions. Longer term activity may alter myostatin, thus our next work will follow up experiment 2 with a 3 year longitudinal analysis.