

MA461 2026 exams – with answer guide

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Seminar: Muscular and functional adaptations to low-load blood flow resisted exercise.

Lecturer: Olav Vikmoen

Reference: Hughes, L., Rosenblatt, B., Haddad, F., Gissane, C., McCarthy, D., Clarke, T., Ferris, G., Dawes, J., Paton, B., & Patterson, S. D. (2019). Comparing the effectiveness of blood flow restriction and traditional heavy load resistance training in the post-surgery rehabilitation of anterior cruciate ligament reconstruction patients: A uk national health service randomized controlled trial. *Sports Medicine*, 49(11), 1787–180

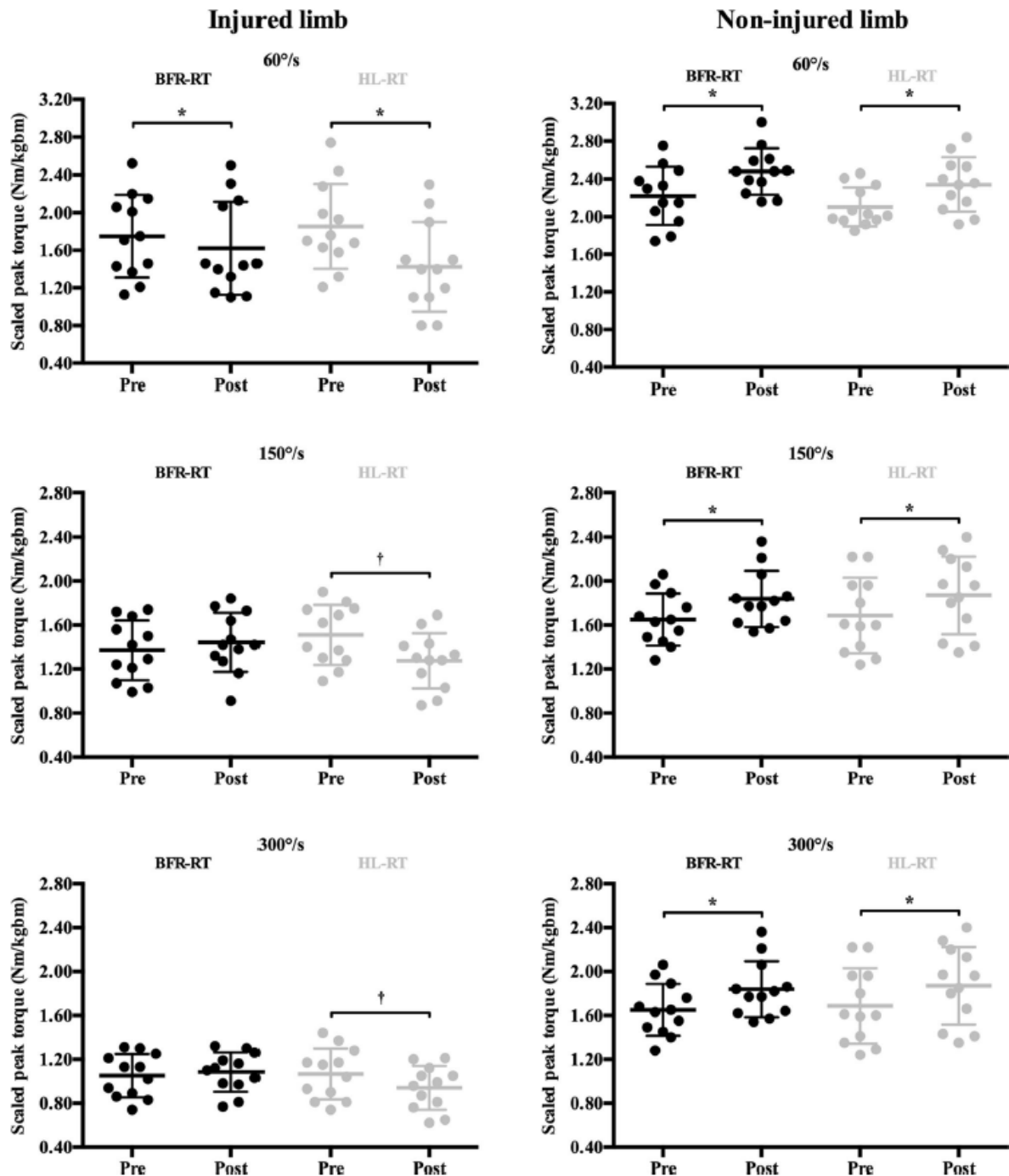


Fig.3 Change in scaled knee extensor peak torque at 60°/s, 150°/s and 300°/s for the injured and non-injured limb. Data are presented as mean±SD. "Asterisk" indicates a significant change ($p < 0.01$);

"Dagger" indicates a significantly greater decrease compared to BFR-RT ($p < 0.01$). BFR-RT blood flow restriction resistance training, HL-RT heavy-load resistance training

Context: Following anterior cruciate ligament reconstruction (ACLR) surgery, patients experience significant loss of lower limb strength due to muscle atrophy and arthrogenic inhibition. Knee extensor (KE) and flexor (KF) muscle weakness is substantial during the first 12 weeks following surgery, impairing lower limb function and quality of life. Muscle weakness can persist for years after ACLR surgery and is associated with chronic reductions in function, a high re-injury risk and joint

degeneration. Therefore, targeting muscle weakness early in the rehabilitation process is imperative. Therefore, a blood flow restriction resistance training (BFR-RT) intervention during an 8-week rehabilitation programme in anterior cruciate ligament reconstruction (ACLR) patients within a National Health Service setting was implemented with the objective of comparing the effectiveness of BFR-RT and standard-care traditional heavy-load resistance training (HL-RT) at improving skeletal muscle hypertrophy and strength, physical function, pain and effusion in ACLR patients following surgery

Methods

28 patients scheduled for unilateral ACLR surgery with hamstring autograft were recruited for this parallel-group, two-arm, single-assessor blinded, randomised clinical trial following appropriate power analysis. Following surgery, a criteria-driven approach to rehabilitation was utilised and participants were block randomised to either HL-RT at 70% repetition maximum (1RM) ($n = 14$) or BFR-RT ($n = 14$) at 30% 1RM. Participants completed 8 weeks of biweekly unilateral leg press training on both limbs, totalling 16 sessions, alongside standard hospital rehabilitation.

Resistance exercise protocols were designed consistent with standard recommended protocols for each type of exercise. Scaled maximal isotonic strength (10RM), muscle morphology of the vastus lateralis of the injured limb, self-reported function, Y-balance test performance and knee joint pain, effusion and range of motion (ROM) were assessed at pre-surgery, post-surgery, mid-training and posttraining. Knee joint laxity and scaled maximal isokinetic knee extension and flexion strength at 60°/s, 150°/s and 300°/s were measured at pre-surgery and post-training.

Questions:

- A) Describe the main differences you can identify from the figures in how BFR-RT vs. HL-RT affected knee extension isokinetic peak torque at different angular velocities!
- B) How do these results align with the expected effects of BFR-RT vs. HL-RT on muscle size and muscle fiber adaptations?

Answer guide:

- a) The main differences between the effects of BFR-RT vs. HL-RT seem to be a more beneficial effect of BFR-RT than HL-RT on the fast speeds of isokinetic knee extension evident by regained peak torque at the post measurements at 150 and 300 degrees per sec. However, for the uninjured leg, the effects seem to be identical across all 3 speeds!
- b) It is surprising that there seems to be a beneficial effect of BFR-RT in the injured limb only at faster speeds because previous studies have indicated that low load BFR-RT specifically targets type I fibre hypertrophy, whereas the HL-RT specifically targets type II fibre hypertrophy. Consequently, the opposite result could have been hypothesized because specific type II fibre hypertrophy should in theory benefit torque production at high speeds. Other effects such as reduced pain or reduced arthrogenic inhibition could potentially explain these findings. This might be supported by the fact that there is no difference between the effects of BFR-RT and HL-RT in the non-injured leg.

Seminar: Influence of sex and female hormones on performance and response to training.

Lecturer: Mette Hansen

Reference: Dam, Dalgaard, Ringgaard, Johansen, Begtsen, Mose, Lauritsen, Ørtenblad, Gravholt, Hansen. "*Transdermal estrogen therapy improves gains in skeletal muscle mass after 12 weeks of resistance training in early postmenopausal women*" *Frontiers in physiology*, Volumen 11, January 2021

Context

The figure shows data from a randomized controlled trial.

The project aim was to test the hypothesis that transdermal estrogen therapy amplifies the skeletal muscle response to resistance training in early postmenopausal women. Thirty postmenopausal women performed supervised resistance training for 12 weeks (3 times a week). The primary focus in the training intervention was on the lower extremities and included three leg exercises in resistance training machines: leg press, knee extension, and knee flexion. The three other exercises were dumbbell triceps extension, sit-ups, and back extensions. Half of the women wore estrogen patches during the 12 weeks (Estrogen), whereas the other half wore placebo patches (Placebo). Muscle cross-sectional area of the quadriceps muscle was measured by MR-scans and whole-body fat-free-mass was measured by DEXA.

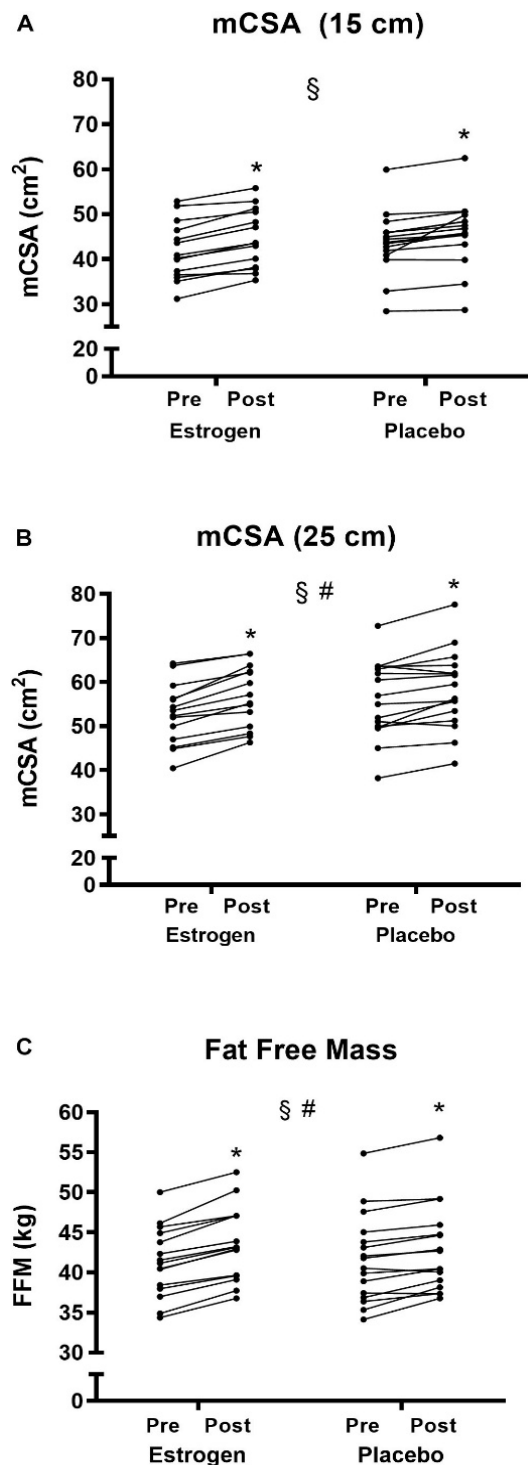


Figure 3 | (A) Muscle cross-sectional area determined 15 cm above patella plateau (mCSA 15 cm) (B) mCSA determined 25 cm measured by magnetic resonance imaging and (C) Whole-body fat-free mass measured by dual-energy-x-ray absorptiometry before and after 12 weeks of resistance training in the individual participants in the ET and PLC groups, respectively. # Group $\times$ time interaction ($p < 0.05$). § Training effect ($p < 0.05$). * Different from pre- in the individual group ($p < 0.05$).

Questions:

Based on the results shown in the figure, describe the findings of the study.

What is your interpretation relative to the aim of the study?

Further, with reference to the curriculum, discuss at least two potential mechanisms which can explain the observations.

Answer guide:

The study investigated whether transdermal estrogen therapy enhances the skeletal muscle response to resistance training in early postmenopausal women.

Figure A and B show changes in muscle cross-sectional area (mCSA) of the quadriceps measured by MRI, whereas Figure C shows changes in whole-body fat-free mass measured by DXA.

In Figure A (mCSA measured 15 cm above the patella), muscle cross-sectional area increased after the 12-week resistance training program in both the estrogen and placebo groups. However, the increase appears relatively similar between the groups, suggesting that estrogen treatment did not markedly augment muscle hypertrophy at this measurement site.

In Figure B (mCSA measured 25 cm above the patella), muscle cross-sectional area also increased in both groups, but the increase is greater in the estrogen group than in the placebo group, indicating a significant group $\times$ time interaction. This suggests that estrogen therapy enhances the hypertrophic response to resistance training at this muscle region.

In Figure C whole-body fat-free mass increased after the training intervention in both groups, but again the increase is larger in the estrogen group, supporting the finding that estrogen therapy amplifies training-induced increases in muscle mass.

Overall, the results suggest that resistance training increases muscle mass in postmenopausal women, but that estrogen therapy enhances this adaptive response, particularly when assessed as whole-body fat-free mass and at certain muscle sites.

Several mechanisms may explain this observation (at least 2 mechanisms should be mentioned).

- 1) estrogen may influence satellite cell activity. Satellite cells are muscle stem cells that are activated during resistance training and contribute to muscle repair and hypertrophy by proliferating and fusing with existing muscle fibers. Estrogen deficiency has been associated with reduced satellite cell content

and function, whereas estrogen supplementation may enhance satellite cell activation and muscle regeneration.

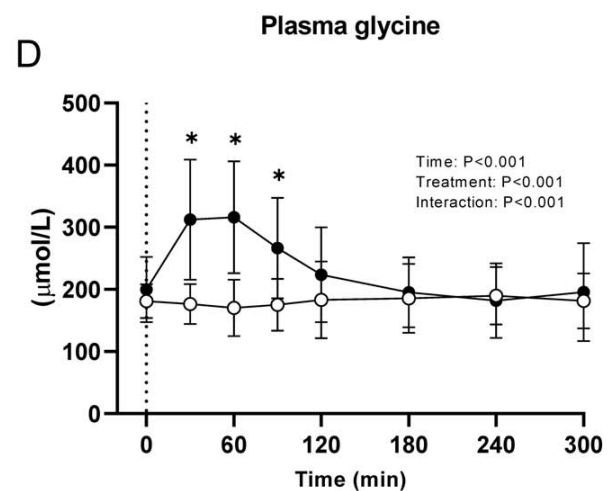
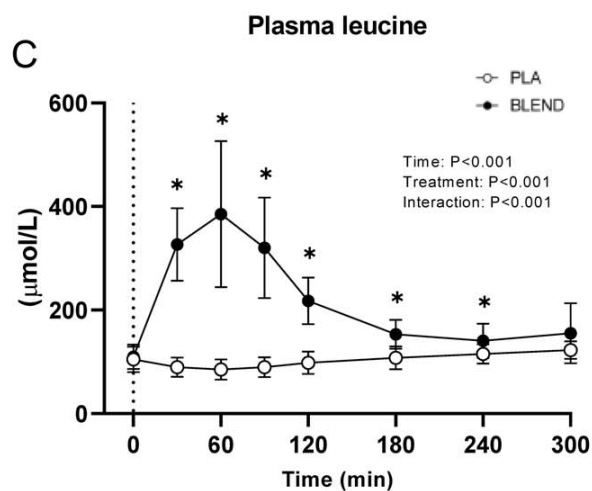
- 2) estrogen can exert direct effects through estrogen receptors (ER- α and ER- β) in skeletal muscle. Binding of estrogen to these receptors can regulate gene expression and intracellular signaling pathways involved in muscle adaptation and protein synthesis.
- 3) Third, estrogen has antioxidants and anti-inflammatory properties, which may reduce exercise-induced muscle damage and improve recovery, thereby facilitating training adaptations.
- 4) estrogen may influence muscle protein turnover. Estrogen may affect the balance between muscle protein synthesis and muscle protein breakdown. Higher estrogen levels may enhance the anabolic response to exercise, thereby promoting greater muscle hypertrophy.

In conclusion, the figures show that resistance training increases muscle mass in postmenopausal women, but that estrogen therapy enhances these adaptations, likely through mechanisms involving satellite cell regulation, estrogen receptor signaling, reduced muscle damage, and improved regulation of muscle protein turnover.

Seminar: The effect of protein intake on muscle hypertrophy with strength training.

Lecturer: Truls Raastad

Reference: Aussieker T, Kaiser J, Hermans WJH, Hendriks FK, Holwerda AM, Senden JM, VAN Kranenburg JMX, Goessens JPB, Braun U, Baar K, Snijders T, VAN Loon LJC. Ingestion of a Whey Plus Collagen Protein Blend Increases Myofibrillar and Muscle Connective Protein Synthesis Rates. Med Sci Sports Exerc. 2025 Mar 1;57(3):544-554.



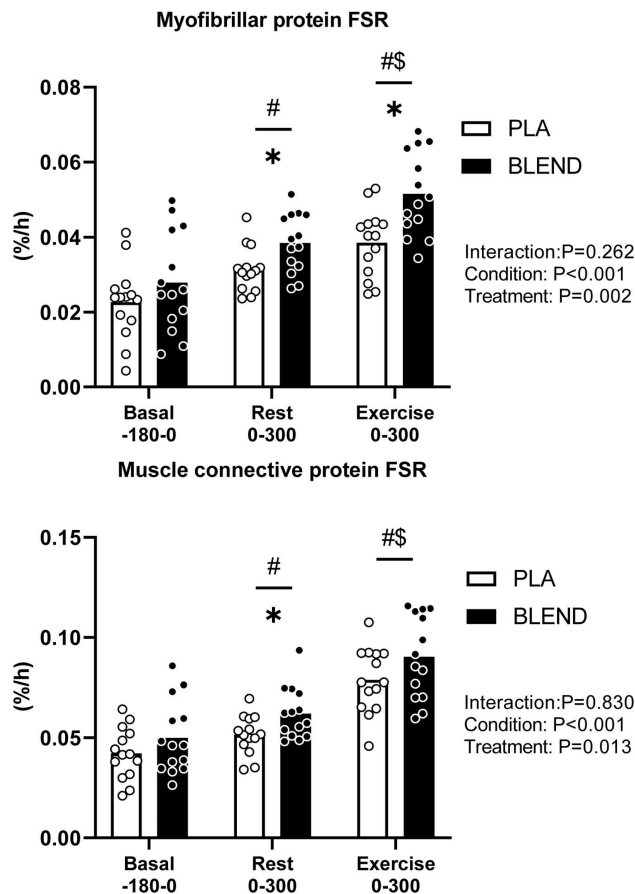


Figure text: Fractional myofibrillar (left) and muscle connective protein synthesis rates (right) (%·h⁻¹) during the post-absorptive period (basal; t = -180 to 0 min), after whey plus collagen

protein or placebo ingestion in rest and during recovery from a single bout of unilateral leg resistance exercise (t = 0–300 min). Bars represent means, and circles represent individual values; n = 14 per group. Data within one condition have been analyzed by independent-samples t-tests. Time-course data were analyzed by a two-factor repeated-measures ANOVA. Bonferroni post hoc testing was used to detect differences between groups. *Significant treatment difference within the condition, P < 0.05. #Significant difference compared with the basal condition, P < 0.05. \$Significant difference compared with the rested condition, P < 0.05. BLEND, 25 g whey plus 5 g collagen protein; PLA, placebo (water).

Context: In a randomized, double-blind, parallel design, 28 men (age: 25 ± 5 yr; body mass index: 23.6 ± 2.3 kg·m⁻²) were randomly allocated to ingest either 30 g of

protein (25 g whey/5 g collagen; BLEND, n = 14) or a noncaloric placebo (PLA, n = 14) following a single session of unilateral leg resistance type exercise. Participants received primed continuous L-[ring-13C6]-phenylalanine infusions with blood and muscle biopsy samples collection for 5 h post-prandially to assess myofibrillar and muscle connective protein synthesis rates. *We hypothesized that ingestion of a whey and collagen protein blend would increase both myofibrillar and muscle connective protein synthesis rates at rest and during recovery from resistance exercise.*

Questions:

- a) Based on what you see from results in the figures, did the authors confirm their hypothesis?
- b) If the blend had been compared to ingestion of 25 g of whey protein, how do you think the results had looked like? (short explanation on why!)

Answer guide:

- A) Partly confirmed: The blend increased myofibrillar FSR more than placebo both at rest and after exercise, whereas muscle connective protein FSR only was increased with the blend at rest.
- B) 25 g of whey would give the same increase in plasma leucine as the blend, but probably no detectable increase in glycine (or the other amino acids stimulating collagen synthesis (hydroxyproline and proline). Theoretically, the stimulation on myofibrillar FSR should be similar between the blend and 25 g of pure whey, whereas for the connective protein FSR the pure whey should be more like the placebo because previous studies have not observed any detectable stimulation of connective protein synthesis with whey protein.

Seminar: Musculoskeletal connective tissue: function and adaptation to training

Lecturer: Olivier Seynnes

Reference: Magnusson, S. P., Langberg, H., & Kjaer, M. (2010). The pathogenesis of tendinopathy: Balancing the response to loading. *Nature Reviews Rheumatology*, 6(5), 262–268.

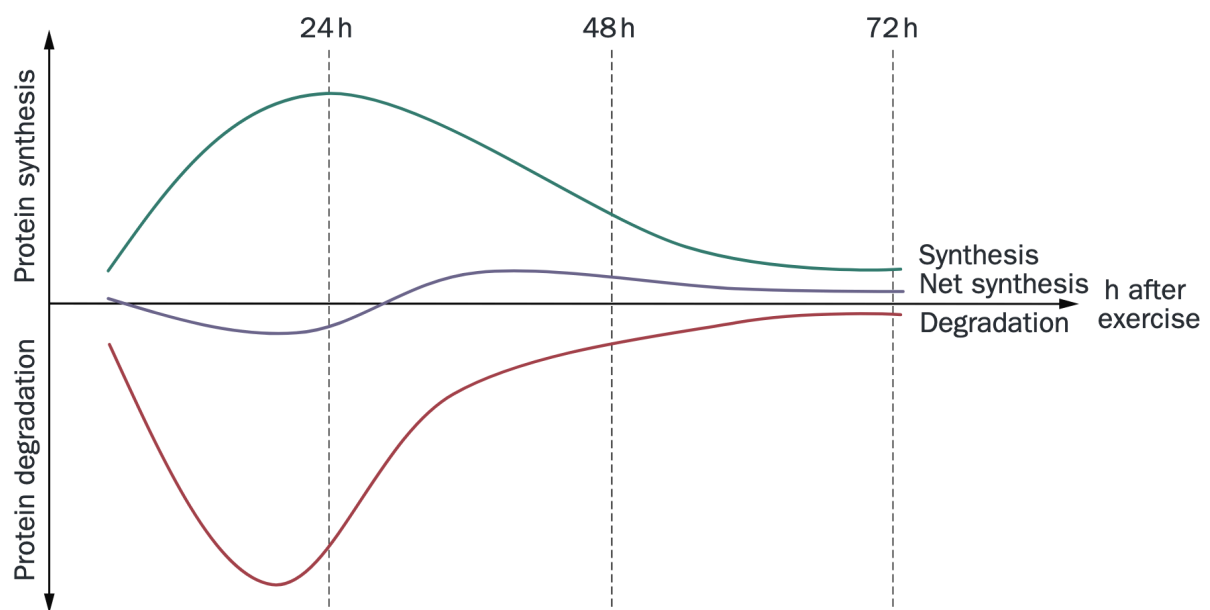


Figure 1. Schematic representation of collagen synthesis and degradation over time after exercise.

Context: This figure was published in a narrative literature review exploring the hypothesis that tendon chronic pathologies (tendinopathy) is related to a failed adaptation to training. It depicts the time course of collagen synthesis/breakdown following a bout of exercise in humans and is based on studies using microdialysis or measurements of labelled proteins.

Questions:

- Describe the relative time courses of collagen synthesis and degradation, and the resulting time course of collagen net synthesis.
- Explain why the authors relate these time courses to the possibility of overuse strain injury.
- Assuming an appropriate recovery time between loading exercises, what changes in tendon morphology and mechanical properties could be expected after a training period?
- What is the main stimulus driving tendon adaptations (e.g. collagen synthesis) to increased loading?
- Considering this stimulus and using the information from the figure, what training frequency and contraction mode (isometric, concentric, eccentric) would you recommend to obtain the largest tendon adaptations?

Answer:

- First two questions: “Over the first 24–36 h, [loading exercise] results in a net loss of collagen but is followed by a net synthesis 36–72 h after exercise. Repeated training with rest periods that are too short can result in a net degradation of the matrix and lead to overuse injury” (from the article). The asynchronous collagen synthesis and breakdown results in a negative balance up to 36 hours after exercise. Overloading before the end of this early phase may exacerbate collagen loss and render the tendon vulnerable to damage accumulation.
- With appropriate timing of exercise, resistive training could result in an increase in tendon cross-sectional area and an increase in stiffness.
- The main stimulus driving tendon adaptations is the strain that it undergoes during training.
- A training with eccentric training is likely to enable the highest production of force and therefore tendon strain. The training sessions should be timed at intervals of at least 36 hours to avoid accumulation of collagen breakdown. Ideally, 48-to-72 hours.

Seminar: Exercise and cancer.

Lecturer: May Grydeland

Reference: Friedenreich CM, Ryder-Burbidge C, McNeil J. Physical activity, obesity and sedentary behavior in cancer etiology: epidemiologic evidence and biologic mechanisms. *Mol Oncol.* 2021 Mar;15(3):790-800. doi: 10.1002/1878-0261.12772. Epub 2020 Aug 18. PMID: 32741068; PMCID: PMC7931121

Table 1. Summary of the observational epidemiologic evidence on the association between physical activity and cancer risk by cancer site. Summary evidence for the dose-response effect, biologic plausibility, and overall classification of evidence was acquired from the 2018 Physical Activity Guidelines Advisory Committee [8] and McTiernan *et al.* [10]. Risk reduction estimates were acquired from McTiernan *et al.* [10] and meta-analyses previously conducted by the authors.

Cancer site	Overall classification of evidence	Approximate range of relative risk reduction for high versus low levels of physical activity	Evidence for dose-response effect	Biologic plausibility
Bladder	Strong	19–24%	Limited	Limited
Breast	Strong	19–27%	Yes	Yes
Colon	Strong	21–27%	Yes	Yes
Endometrial	Strong	19–29%	Yes	Yes
Esophageal adenocarcinoma	Strong	19–51%	Yes	Yes
Gastric cardia	Strong	15–19%	Yes	Yes
Renal	Moderate	12–16%	Limited	Yes
Lung	Moderate/Limited ^a	27–28%	Yes	Limited
Ovarian	Moderate	2–23%	Limited	Yes
Pancreas	Moderate	9–25%	Yes	Yes
Prostate	Limited	3–13%	Limited	Limited

^aConfounding by smoking is possible.

Table 2. Summary of the observational epidemiologic evidence on the association between sedentary time and cancer risk by cancer site. Summary evidence was acquired from Jochem *et al.* [12].

Cancer site	Overall classification of evidence	Magnitude of relative risk increase for high versus low sedentary time	Evidence for dose-response effect	Biologic plausibility
Colon	Moderate	28–44%	Limited	Yes
Endometrial	Moderate	28–36%	Limited	Yes
Lung	Moderate ^a	21–27%	Limited	Limited

^aConfounding by smoking is possible.

Context: Friedenreich *et al* 2020 published a review with the main aim to summarize the epidemiologic evidence concerning the contribution of physical activity (PA), sedentary behavior (SB), and obesity to cancer etiology* and to provide an overview

of the biologic mechanisms that may be operative between these factors and cancer incidence.

*etiology: the cause, set of causes, or manner of causation of a disease or condition

Questions: Consider the information given in Table 1 and 2, give a short description of the main findings and give the most important factors that explain the differences in evidence base for PA vs. SB and cancer incidence.

Answer guide: The task is to consider the evidence base on the associations between PA, SB and cancer risk. Strong and consistent evidence exists that higher levels of physical activity reduce the risk of six different cancer sites (bladder, breast, colon, endometrial, esophageal adenocarcinoma, gastric cardia), whereas moderate evidence inversely associates physical activity with lung, ovarian, pancreatic and renal cancer, and limited evidence inversely correlates physical activity with prostate cancer. Sedentary behavior, independent of physical activity, has been shown to increase the risk of colon, endometrial, and lung cancers. The evidence base for the relationship between SB and other cancers is too low to be considered in a review, particularly because investigation of SB as an individual risk factor (independent of PA) to cancer is relatively new and therefore understudied. Accurate investigations of SB in large cohorts are methodologically difficult, and no consensus exists in how assessment of this behavior should be done. Further limitations: mainly retrospective investigations, mainly use of questionnaires prone to recall and cognitive bias, lack of consensus in settings when SB is device measured, lack of consensus regarding definitions of what is considered SB and breaks in SB.

Further info: Given that a high proportion of individuals spend the majority (~ 55%) of their time awake taking part in sedentary behaviors [16], interventions targeting reductions in sedentary time would contribute to reducing chronic disease risk, including cancer, at a population-level. For individuals with habitually high levels of sedentary time, it is expected that replacing some of that time with light intensity or ambulatory activities (e.g., breaking up sedentary time by standing or walking) would

lead to some health benefits, with the greatest benefits occurring when sedentary time is replaced with planned moderate-vigorous intensity physical activity [14]. Additional research from prospective cohort studies is needed to assess the interactive effects of physical activity and sedentary time on cancer incidence [14]. Randomized controlled trials focused on promoting reductions in sedentary by replacing it with light, moderate, and/or vigorous intensity physical activity are also needed in individuals at high risk for cancer development.

Seminar: Training load monitoring in team sports

Lecturer: Live Luteberget

Reference: Coutts, A. J., Crowcroft, S., & Kempton, T. (2018). Developing athlete monitoring systems: Theoretical basis and practical applications. In M. Kellmann & J. Beckmann (Eds.), *Sport, Recovery and Performance: Interdisciplinary Insights* (pp. 19-32). Abingdon: Routledge.

Figure:

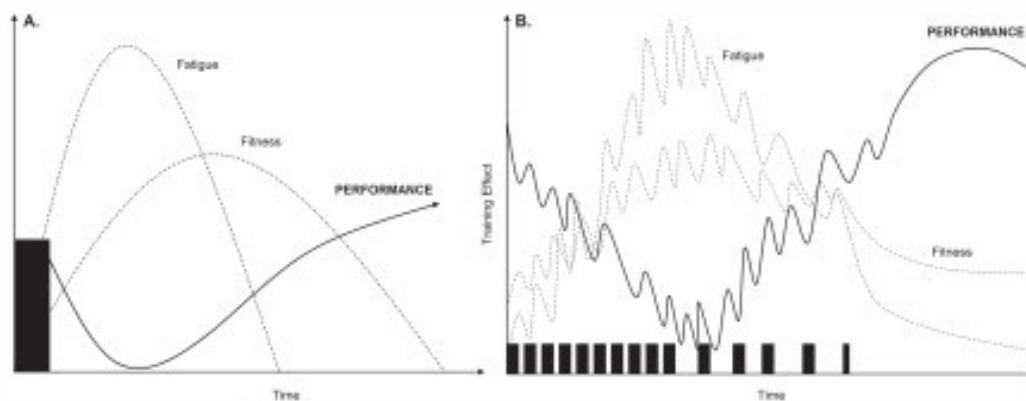


Figure 2.1 The modelled fitness and fatigue responses to (A) a single bout and (B) a sequence of training bouts.

Context: The figure above illustrates the fitness–fatigue model in response to (A) a single training bout and (B) repeated training bouts during a training program.

Questions:

- Using the figure, explain the key principles of the fitness–fatigue model and how training affects performance over time.
- Discuss how information from training load monitoring can be used to optimize this balance in practice.

Answer guide

- A strong answer should explain that training produces two simultaneous responses: fitness and fatigue. Fitness represents the positive adaptations

that improve performance capacity over time, whereas fatigue reflects the short-term reduction in performance that occurs as a consequence of training stress. These two responses follow different time courses. Fatigue typically develops quickly after a training session and dissipates relatively rapidly during recovery, while fitness develops more gradually and persists for a longer period.

Immediately after a training bout, fatigue usually outweighs the positive effects of fitness, which can lead to a temporary reduction in performance. As recovery occurs, fatigue gradually decreases while the fitness effect remains, allowing performance to return to baseline and potentially improve above previous levels. The figure should therefore be interpreted as showing how performance fluctuates over time depending on the relative contributions of fitness and fatigue.

When multiple training bouts are performed over time, both fitness and fatigue responses can accumulate. Performance at any given time reflects the net balance between these two components. If training is appropriately balanced with adequate recovery, the accumulated fitness effect will progressively lead to improvements in performance over the course of a training program. Conversely, if fatigue accumulates excessively due to high training loads or insufficient recovery, performance may stagnate or temporarily decline.

Students should demonstrate that they can interpret the key elements illustrated in the figure (i.e., link their explanation to the figure), including the short-term drop in performance following a training session, the recovery phase during which fatigue dissipates, and the potential for improved performance once fatigue has decreased. Detailed physiological mechanisms are not required.

- b) A strong answer should explain that training load monitoring can help practitioners manage the balance between fitness and fatigue described in the fitness–fatigue model. By systematically monitoring training load and athlete responses, coaches and sport scientists can gain insight into how athletes are responding to training and whether the current training program is likely to promote adaptation or lead to excessive fatigue.

Training load monitoring typically involves assessment of both external load and internal load. External load refers to the work completed by the athlete, such as distance covered, speed, or accelerations. Internal load reflects the athlete's physiological or perceptual response to that work and can be assessed through measures such as session rating of perceived exertion (sRPE), heart rate responses, or other physiological indicators. In addition, practitioners may monitor indicators of recovery and fatigue, such as wellness questionnaires, or simple performance tests.

Information from these monitoring tools can help practitioners evaluate whether fatigue is accumulating excessively or whether the training stimulus is appropriate for promoting adaptation. Based on this information, training load can be adjusted by modifying volume, intensity, or recovery periods. For example, training load monitoring may help identify when an athlete requires additional recovery, when training loads can be increased to provide sufficient stimulus, or when a reduction in training load (e.g., tapering) is needed to reduce fatigue and optimize performance before competition.

Overall, training load monitoring provides practitioners with practical tools to manage the balance between fitness and fatigue over time, helping to optimize performance improvements while reducing the risk of excessive fatigue or maladaptation.

Seminar: Stretching exercise. What is it good for?

Lecturer: Jens Bojsen-Møller

Reference: Magnusson, S. P., Simonsen, E. B., Aagaard, P., Sorensen, H., & Kjaer, M. (1996). A mechanism for altered flexibility in human skeletal muscle. *J.Physiol*, 497 (Pt 1), 291-298.

Context: It is well known that stretching exercise over time (weeks – months) increases joint range of motion. The physiological mechanism for increased ROM after habitual stretching exercise could be changes in muscle-tendon length, changes in tissue mechanical properties such as stiffness, or it could be due to increased tolerance to stretch. The purpose of the present study was in fact to examine mechanisms that underlie changes in ROM after a period (3 weeks) of unilateral stretching for the hamstring muscles.

One leg underwent stretching (2 times pr day for 20 days) while the other leg served as control.

The study had 2 different test protocols, where the hamstring muscles were passively stretched (constant joint velocity) by use of an isokinetic device to a fixed angle (protocol 1) and stretched further to the point of pain (protocol 2). In both protocols passive torque, joint angle and stored energy (area below the torque-joint angle curve) was measured. Moreover, EMG was sampled from the hamstring muscles during the stretch. Stiffness of the muscle tendon unit was calculated from the slope of the torque-joint angle curve (protocol 1).

Both the intervention leg and the control leg were examined prior to and post training, and both protocol 1 and 2 were applied to both legs.

The authors found in general (protocol 1 and 2) very low levels of EMG (approximately 3% of maximal voluntary EMG amplitude) during stretches and no differences in neural activation (EMG) were observed pre-post on both the control and stretched leg.

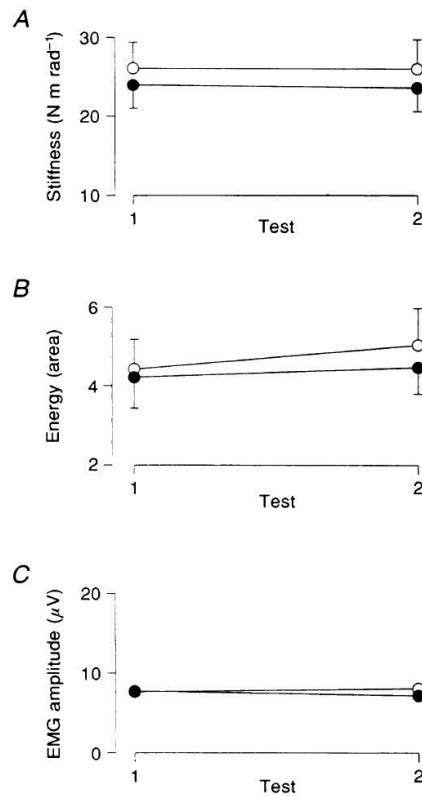


Figure 4. Results from the stretch phase of protocol 1

No significant differences existed for stiffness (A), energy (B) or EMG (C) before and after the training period on the control (○) and the stretch side (●). Note that the EMG variation was negligible. Here, and in the following figures, vertical lines and bars indicate the S.E.M.

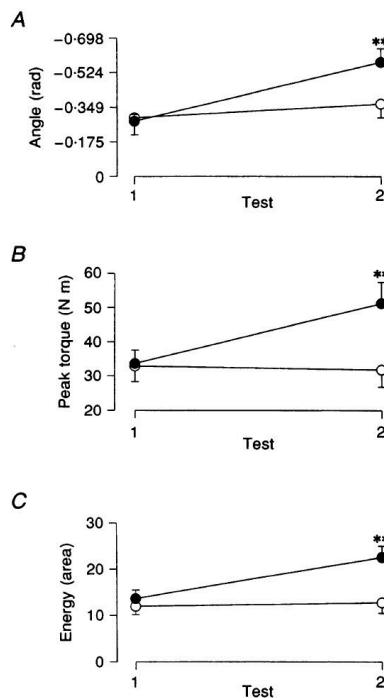


Figure 6. Results from protocol 2

The stretching regimen resulted in significant increases on the stretch side (●) in maximal angle (A), peak torque (B) and energy (C), $P = 0.018$, while the control side (○) remained unchanged.

Questions:

1. Explain the findings in the above figures (figure 4 and 6)

2. Combined with the observation, that muscle activation (EMG amplitude) was very limited during stretches, explain why the authors conclude that the increases in joint ROM after training pertain to training induced changes in “tolerance to stretch”.

Answer guide: Figure 4 presents average data from protocol 1 where it can be seen that passive stiffness of the muscle tendon unit (mechanical property) and stored energy (area below the torque – angle curve) are unchanged following a period of stretching. Moreover it is seen that neural activation is low (negligible variation) and does not change relative to intervention leg vs control leg.

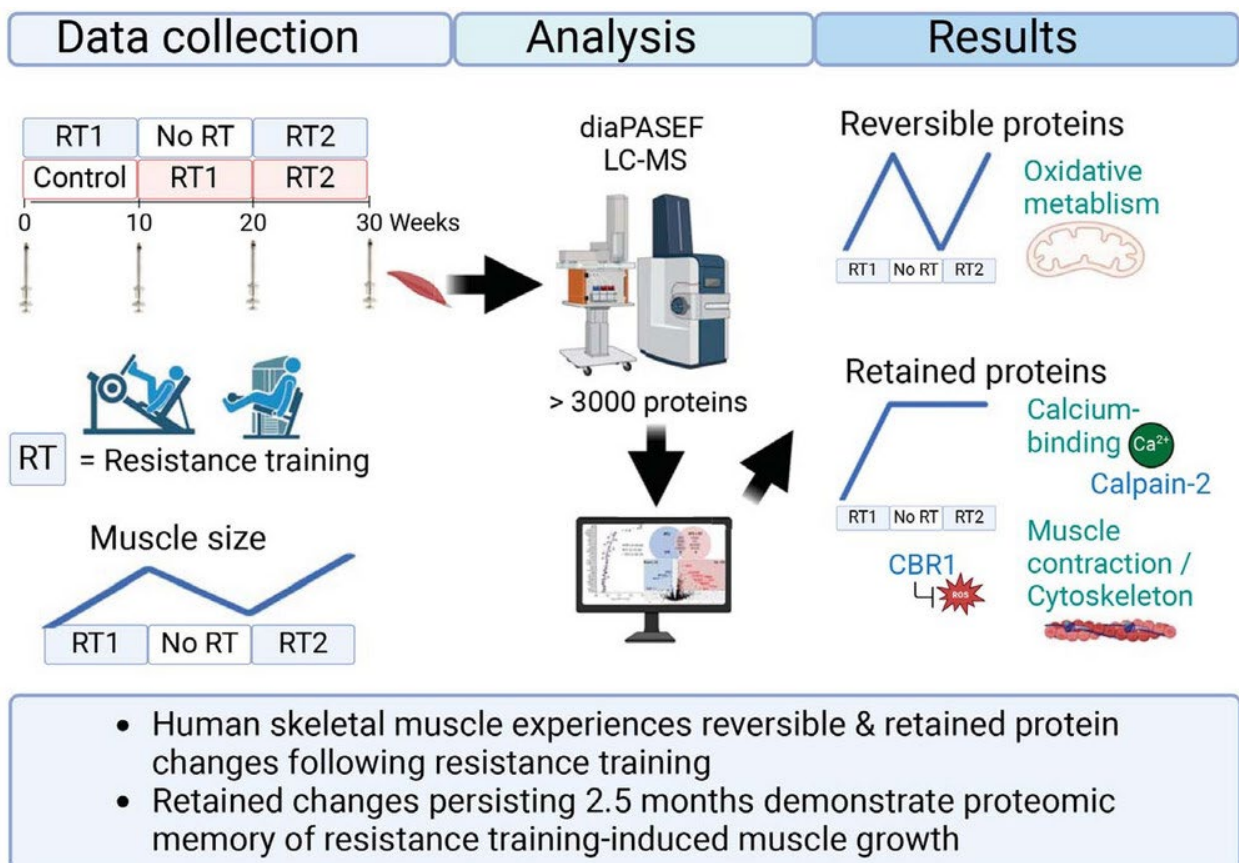
Figure 6 consists of average data on all subjects (protocol 2) and demonstrates that peak torque, joint angle at the point of pain, and stored energy during stretch increase for the intervention leg, while no changes are seen in the control leg. Combined with the observed minute muscle activation (very low EMG amplitude) the combined data suggest that the changes following the stretching intervention are unrelated to stiffness or length changes in the muscle tendon unit, and unrelated to reflex activity (low and unchanged pre-post EMG) but instead are mainly brought about by an increased tolerance to stretch for the intervention leg.

Seminar: Does Skeletal Muscle have a Memory?

Lecturer: Adam Sharples

Reference: Hulmi, J. J., Halonen, E. J., Sharples, A. P., O'Connell, T. M., Kuikka, L., Lappi, V. M., Salokas, K., Keskitalo, S., Varjosalo, M., & Ahtiainen, J. P. (2025). Human skeletal muscle possesses both reversible proteomic signatures and a retained proteomic memory after repeated resistance training. *The Journal of Physiology*, 603(9), 2655–2673. <https://doi.org/10.1113/JP288104>

Figure and Context:



The Journal of
Physiology

A schematic illustrating positive skeletal muscle memory at the proteomic level following training and detraining.

Question:

Describe, explain, and discuss positive skeletal muscle memory at the molecular level. Using the figure as a guide, outline how skeletal muscle can retain molecular signatures of earlier training across epigenetic, transcriptional, and most recently, proteomic layers, and how these retained signatures may enhance the response to later retraining. In your answer, you should draw upon resistance training studies that demonstrate retained DNA hypomethylation and gene expression as well as retained protein abundance following detraining, and you may also refer to evidence that high-intensity interval training evokes mode-specific retention of hypomethylation and gene expression. Answers should consider how these findings collectively support the concept that skeletal muscle exhibits a molecular memory of exercise training that persists through detraining and facilitates a retained capacity to respond and/or an enhanced response during subsequent retraining.

Answer guide: Positive skeletal muscle memory refers to the retained response during detraining or enhanced adaptive response during retraining that results from molecular signatures retained from an earlier training period. At the epigenetic and transcriptional level, resistance training induces hypomethylation in genes related to structural remodelling, cytoskeletal organisation and growth signalling, and at the gene loci level retain their hypomethylated state during detraining, maintaining or priming gene expression for an amplified response during retraining. High-intensity interval training produces a similar retained hypomethylated and transcriptional profile but in mode-specific pathways, particularly those governing metabolic regulation, lactate transport and calcium signalling.

Proteomic analyses show that resistance training also produces both reversible and retained protein signatures. Many metabolic proteins increase with training, return to baseline with detraining and increase again during retraining, whereas several structural, cytoskeletal and calcium-binding proteins (for example CBR1, ME2, S100A13 and CAPN2) remain elevated through detraining and retraining, demonstrating memory through to the proteomic level. Genes such as CAPN2 appear consistently across epigenetic, transcriptional and proteomic layers, illustrating a central integrative node of molecular memory.

Together, these findings show that skeletal muscle retains training-induced epigenetic marks, transcriptional profiles and protein abundance in a way that could enhance the adaptation of muscle during subsequent retraining, with resistance training and high-intensity interval training imprinting partially distinct but complementary molecular pathways.

Seminar: Modelling of Human Movement

Lecturer: Hannah Rice

Reference: Han, S., Moon, J., Lee, J., Han, S., Sterzing, T., Farris, D., & Rice, H. (2025). Influence of Manipulating Running Foot Strike Angle on Internal Loading of the Tibia. *Scandinavian Journal of Medicine & Science in Sports*, 35(5), e70066. <https://doi.org/10.1111/sms.70066>

Context

The purpose of this study was to quantify internal loading at the distal third of the tibia during running with different foot strike types (i.e. indicated by which part of the foot first contacts the ground).

Nineteen runners who naturally run with a rearfoot strike (heel contact first) ran with three foot strike conditions: 1) their preferred foot strike where they were not asked to think about foot strike; 2) with an intentional rearfoot strike and 3) with an intentional forefoot strike, in a randomised order. When asking participants to run with an intentional foot strike, they tend to exaggerate this condition, hence resulting in three distinct conditions: natural rearfoot strike (nRFS), exaggerated rearfoot strike (eRFS) and exaggerated forefoot strike (eFFS).

Force and motion capture data were collected synchronously during running, and the foot strike in the sagittal plane was identified from the kinematic data. Tibial bending moments were estimated as an indicator of internal tibial loading. These were estimated using musculoskeletal modelling and beam theory, where the tibia was represented as a hollow cylindrical beam. The results of the influence of running with different foot strike types on tibial bending moments are presented in Figure 1 and Table 1 below.

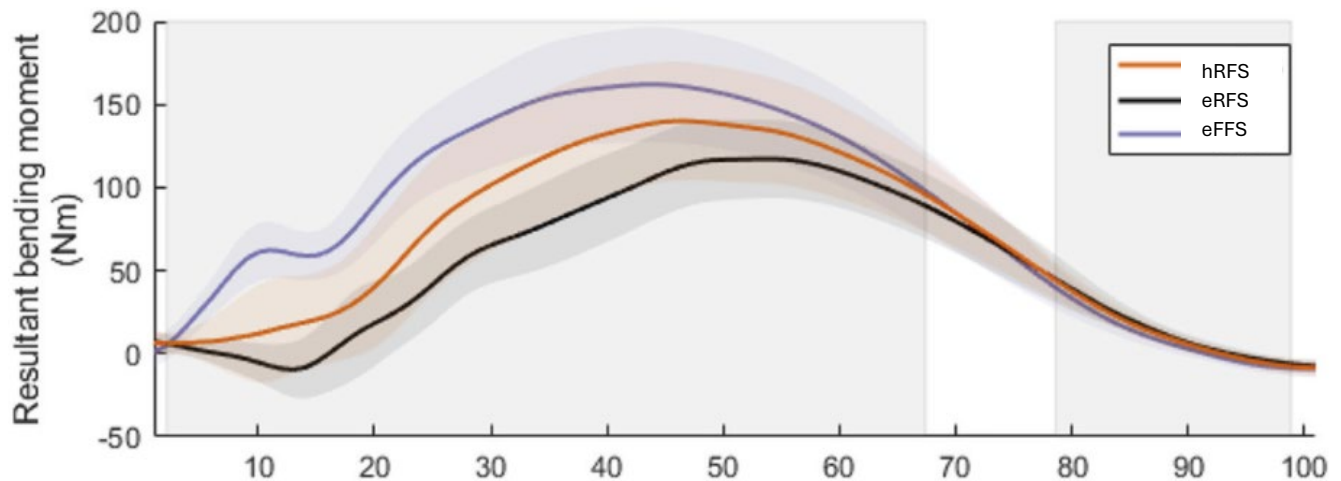


Figure 1: Mean and standard deviation tibial bending moment during stance. The horizontal axis represents the percentage of the stance phase. Vertically shaded areas indicate regions where significant differences were observed between foot strike patterns (hRFS, habitual rearfoot strike; eRFS, exaggerated rearfoot strike; eFFS, exaggerated forefoot strike).

Table 1: Peak bending moment during stance in the three conditions

	Habitual RFS	Imposed RFS	Imposed FFS	P-value
Peak Tibial Bending Moment (Nm)	142.3 (34.9)	120.5 (34.9)	163.6 (34.1)	< 0.001

Questions

- 1) What was the influence of foot strike on tibial loading during running?
- 2) Suggest two ways in which the complexity of the model could be increased?
- 3) Name one advantage and one disadvantage of increasing the model complexity.

Answer guide

- 1) Increasing foot strike angle from most rearfoot to most forefoot increased the tibial bending moment throughout the first ~two thirds of stance, including the peak.
- 2) Participant-specific bone scaling, bone geometry, finite element with generic density or participant-specific density, accounting for deformation, accounting for bone remodelling/fatigue.
- 3) Advantages: more participant-specific values; can be more valid; can allow better detection of between- participant differences; can reduce error from assumptions and oversimplifications. Disadvantages: may result in unnecessary increase in computation time, resource requirement, new error sources.

Seminar: The kinematics and kinetics of human running

Lecturer: Hannah Rice

Reference: Park, S.-K., Jeon, H.-M., Lam, W.-K., Stefanyshyn, D., & Ryu, J. (2019). The effects of downhill slope on kinematics and kinetics of the lower extremity joints during running. *Gait & Posture*, 68, 181–186.
<https://doi.org/10.1016/j.gaitpost.2018.11.007>.

Context

Fifteen runners ran on a force-instrumented treadmill with three different slope conditions to simulate downhill running [level (0°), moderate (-6°), and steep (-9°)] at 3.2 m/s during which synchronized motion capture and force data were collected. Sagittal plane joint angles, moments, power and work were reported from the ankle, knee and hip during the stance phase of running. Power was the joint moment multiplied by joint angular velocity. Results for joint power are presented in Figure 1 below.

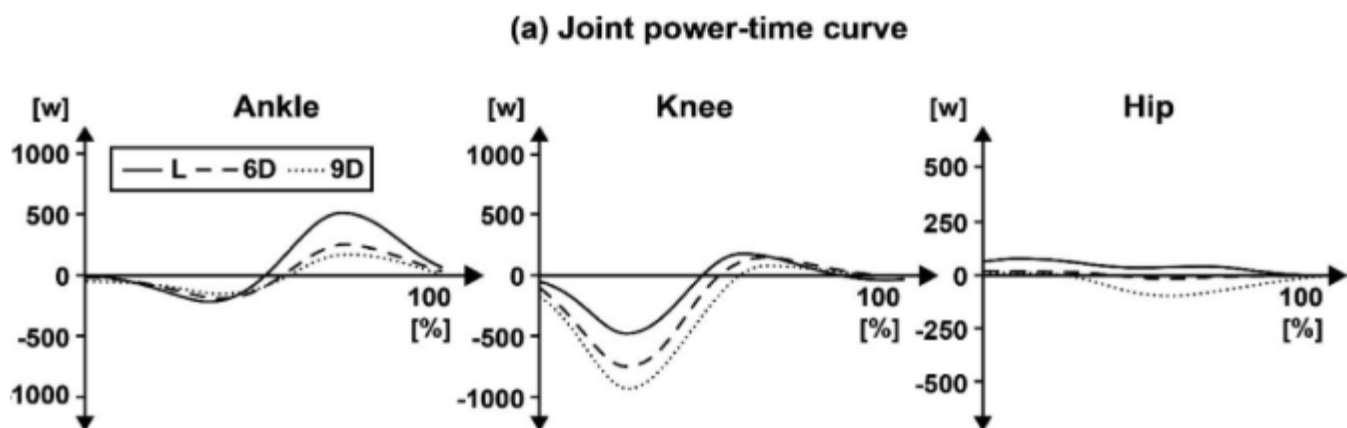


Figure 1 (a): Comparisons of joint power-time curve between different downhill slope conditions, Level (L), 6 degrees downhill (6D) and 9 degrees downhill (9D).

Questions

- 1) What was the influence of downhill running on ankle, knee and hip power?
- 2) Describe what is occurring at the ankle and knee in terms of concentric and eccentric actions during level running.

Answer guide

- 1) With an increase in downhill incline, there appeared to be a decrease in positive power at the ankle joint, an increase in negative power at the knee joint and a change to negative power during later stance at the hip joint. There was no/less visible effect of downhill running on negative ankle work.
- 2) The ankle experiences initial eccentric activity, as indicated by the negative work, followed by concentric activity during the later stance phase, where the positive work is greater than the initial negative work. The knee experiences initial eccentric activity until approximately midstance, followed by concentric activity in late stance, where this positive work is smaller than the negative work.

Seminar: The physiology of high-intensity exercise

Lecturer: Øyvind Skattebo

Reference: Calbet, J. A., Losa-Reyna, J., Torres-Peralta, R., Rasmussen, P., Ponce-Gonzalez, J. G., Sheel, A. W., de la Calle-Herrero, J., Guadalupe-Grau, A., Morales-Alamo, D., Fuentes, T., Rodriguez-Garcia, L., Siebenmann, C., Boushel, R., & Lundby, C. (2015). Limitations to oxygen transport and utilization during sprint exercise in humans: evidence for a functional reserve in muscle O₂ diffusing capacity. *Journal of Physiology*, 593(20), 4649-4664.
<https://doi.org/10.1113/jp270408>

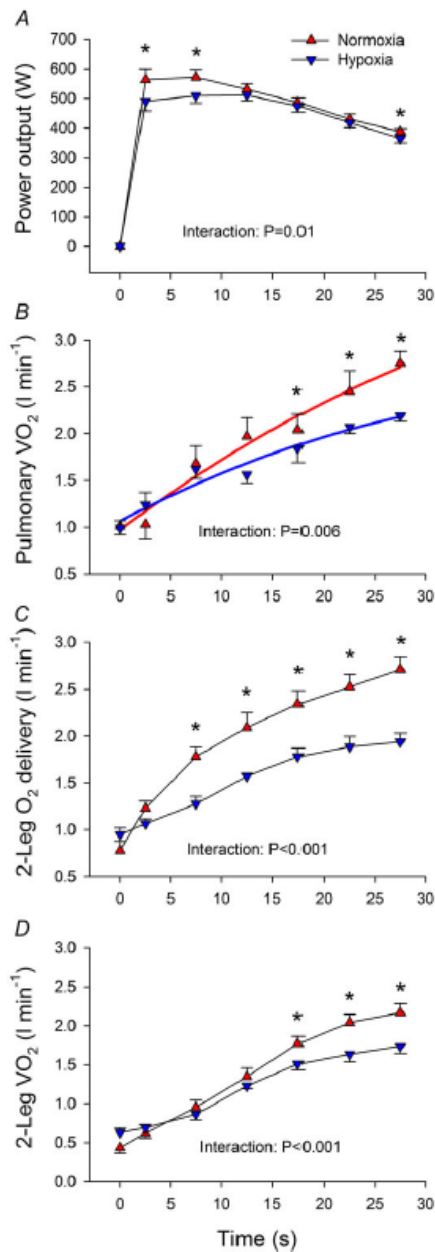


Figure 1. Power output, O_2 delivery and O_2 uptake during sprint exercise

Power output (A), pulmonary oxygen uptake ($\dot{V}O_2$) (B), two-legged O_2 delivery (C), and two-legged $\dot{V}O_2$ (D) during isokinetic Wingate tests at 80 rpm performed in normoxia ($P_{iO_2} \approx 143$ mmHg) and acute hypoxia ($P_{iO_2} \approx 73$ mmHg). * $P < 0.05$ normoxia (triangles pointing up) vs. hypoxia (triangles pointing down); $n = 10$ (one subject did not perform the Wingate test in hypoxia).

Context:

In Calbet et al. (2015), eleven young men performed 30-second all-out Wingate cycling sprints in normoxia (inspiratory O_2 pressure of 143 mmHg) and acute hypoxia (73 mmHg). Blood flow and O_2 uptake were measured by the direct Fick method in the legs, by measuring leg blood flow, O_2 delivery and O_2 extraction. Figure 1 from

the paper shows the time course of (A) power output, (B) pulmonary $\dot{V}O_2$, (C) leg O_2 delivery, and (D) leg $\dot{V}O_2$ during the sprints in normoxia and hypoxia.

Key findings:

- **Power output:** Peak power output was not statistically different between conditions ($P=0.36$), but mean power during the first 10 seconds was ~12% lower in hypoxia. Over the whole sprint, mean power was ~7% lower in hypoxia.
- **O_2 delivery & uptake:** Leg O_2 delivery was ~22–28% lower in hypoxia, especially in the last 15 seconds, leading to ~19–23% lower leg $\dot{V}O_2$ compared with normoxia.
- Despite reduced O_2 delivery and $\dot{V}O_2$, power output during the last 15 seconds was maintained at near-normoxic levels.

Questions:

1. Why was power output only slightly lower (~7%) across the whole sprint in hypoxia, even though leg $\dot{V}O_2$ was reduced by ~23%? Which energy system did and did not compensate for the reduced oxidative ATP production?
2. Why was mean power during the **first 10 seconds** already lower in hypoxia despite a similar total O_2 uptake? What mechanism may have reduced the power output?
3. In the **last 15 seconds** of the sprint, O_2 delivery and $\dot{V}O_2$ were substantially lower in hypoxia, yet power output was maintained. What does this tell you about energy system contribution during this phase?
4. If you assume a similar ATP cost per watt of cycling in both conditions, how does Figure 1 illustrate a shift in the balance between aerobic and anaerobic ATP resynthesis across the sprint in hypoxia compared with normoxia?

Answer Guide

1. Whole-sprint power vs. $\dot{V}O_2$

- Power fell only ~7% despite a ~23% drop in leg $\dot{V}O_2$.
- Compensation came from **higher anaerobic glycolysis** (lactate-producing ATP resynthesis).
- PCr contribution is mostly expended in the first 10 s, so glycolysis had to fill the gap.

2. First 10 seconds of sprint

- Leg O_2 uptake was similar between normoxia and hypoxia, yet power was ~12% lower in hypoxia.
- This suggests a **neural/central limitation** (reduced recruitment or inhibition of high-threshold motor units under hypoxia).
- Supported by evidence of hypoxia-induced activation of group III/IV muscle afferents → reflex inhibition of motor drive (this last point is not needed but is bonus- as would entail that the full article was read).

3. Last 15 seconds of sprint

- Hypoxia caused ~26–28% lower O_2 delivery, leading to ~19% lower leg $\dot{V}O_2$.
- Power was still maintained at normoxic levels → implies **greater reliance on glycolysis** during this phase.
- Anaerobic ATP resynthesis “substituted” for reduced oxidative supply.

4. Aerobic vs. anaerobic ATP balance

- Figure 1 shows that in normoxia, oxidative ATP resynthesis ($\dot{V}O_2$) rises steadily and contributes substantially after ~15 s.
- In hypoxia, this oxidative rise is blunted → larger fraction of ATP must come from **anaerobic sources** throughout the sprint.
- Hence, the **balance shifts toward glycolysis** in hypoxia, particularly in the latter half of the sprint.

Seminar: The application of sport science knowledge in elite sport practice

Lecturer: Matthias Gilgien

Reference: Fullagar HHK, McCall A, Impellizzeri FM, Favero T, Coutts AJ. The Translation of Sport Science Research to the Field: A Current Opinion and Overview on the Perceptions of Practitioners, Researchers and Coaches. *Sports Med.* 2019 Dec;49(12):1817-1824. doi: 10.1007/s40279-019-01139-0. PMID: 31214978.

Context: This text is part of the chapter “2.2 Researchers’ and Practitioners’ Perceived Importance of Research Areas” describing the advantages and disadvantages of embedded scientists in clubs / teams vs research conducted externally in a scientific institution.

“Club- or organisation-embedded research is generally considered to have greater impact on professional practice, whereas laboratory studies lay the foundation of EBP in many areas of sport science [1]. For instance, well-controlled randomised trials produce a much higher level of evidence than applied investigations conducted in the field [17]; however, the former has reduced ecological validity. Field-based investigations have been reported as difficult to implement in high-performance settings [18] due to experimental control requirements, training interference and randomisation issues [1]. Indeed, balancing the need for efficacious research (i.e. does the intervention work?) with effective research (i.e. does it work in practice?) is paramount [19]. In addition, due consideration needs to be given to the efficiency of such measures (cost vs. benefit [19]). Practitioners and researchers also need to consider that the organisation or club will prioritise a competitive advantage over research dissemination. Although difficult, it is important for sport scientists to strive for appropriate scientific design where possible when performing research in applied settings (recommendations regarding this issue are offered in Sect. 5). Such optimised design may increase the rigour and subsequent uptake of sport science research, which in turn will influence the areas researchers and practitioners view as important.

Ideally, if better-quality research is designed and conducted, this will strengthen the evidence from which applied sport science practice is based [3].”

Questions: Based on the text explain the arguments for and against the embedding of scientists in the sport practice aiming to maximize the applicability of the research findings. Implement the implications of embedding scientists and maximizing applicability in practice for the study design.

Answer guide:

1. Arguments for embedding scientists in sport practice

1.1 Increased applicability and ecological validity

Research conducted directly within sports clubs or organisations is perceived as more relevant to real performance contexts. Studies embedded in the applied environment are considered more applicable because they address practical problems faced by coaches and practitioners and therefore facilitate the translation of research findings into practice.

Embedding researchers within teams allows them to:

- Observe the real constraints of training and competition
- Develop research questions aligned with coaches’ and organisations’ needs
- Ensure the outcomes are directly usable for improving performance

This leads to greater ecological validity, meaning results better reflect real-world conditions.

1.2 Improved collaboration and knowledge transfer

Embedding scientists can improve collaboration between researchers, practitioners, and coaches. This approach provides:

- Additional scientific expertise within the team environment
- Direct communication between researchers and coaches

- Faster translation of scientific insights into training practices

Such close collaboration helps bridge the gap between research and practice, which has traditionally limited the use of sport science in elite sport.

1.3 Identification of relevant research questions

When scientists work within sport organisations, research questions are more likely to be generated from real performance needs rather than purely academic interests.

This increases the likelihood that:

- Findings will be implemented in training and competition
- Research outcomes provide competitive advantages

2. Arguments against embedding scientists in sport practice

2.1 Reduced experimental control

Field-based research often limits the ability to implement highly controlled experimental designs. Compared with laboratory studies:

- Randomisation may be difficult
- Standardisation of training loads or conditions may not be possible
- Confounding variables may influence results

Therefore, while field studies increase applicability, they may produce lower internal validity than controlled laboratory experiments.

2.2 Practical constraints in elite sport

Research conducted within teams must fit into the performance environment, which creates several limitations:

- Training schedules and competition calendars restrict research protocols
- Coaches may prioritise performance over experimental requirements
- Interventions may interfere with athletes' preparation

These constraints can make it difficult to conduct rigorous experimental studies.

2.3 Organisational and expertise challenges

Embedding students or scientists in clubs may introduce challenges such as:

- Differences in background knowledge (monodisciplinary vs. multidisciplinary)
- Limited experience of students or junior researchers
- Organisational culture and staff acceptance

Additionally, teams may prioritise competitive advantage over publication, limiting dissemination of findings.