Overload Injury of the Knees With Resistance-Exercise Overtraining: A Case Study

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Objective: To report a joint-centered mechanism of performance decrements caused by overtraining.
Design: Case study.
Setting: Laboratory-induced overtraining.
Participants: Eleven weight-trained men, 1 (subject A) with overload injury of the knees.
Intervention: High-intensity squat resistance-exercise overtraining for 2 weeks.
Outcome Measures: 1RM lower-body strength, isokinetic and isometric knee-extension strength, and stimulated isometric knee-extension strength.
Results: Subject A's 1RM strength decreased 40.3 kg, and the other overtrained subjects (OT) exhibited significant ($P < .05$) 1RM decrements ($x = -9.3$ kg). Isokinetic knee-extension strength decreased for all subjects. For the OT group, voluntary isometric knee-extension strength did not change and stimulated isometric knee-extension strength decreased. Subject A exhibited increased values for both these variables.
Discussion: These data indicate that muscle strength was attenuated for subject A only during dynamic activity. It is theorized that subject A exhibited a joint-centered overtraining syndrome, with afferent inhibition from the affected joints impairing dynamic strength.

Improper exercise prescription can lead to a phenomenon known as overtraining. There is considerable discussion as to the exact definition of overtraining.[1,2] For the purposes of this study, overtraining is defined as a training stimulus involving an increase in training volume and/or intensity that results in decreased physical performance that is not caused by muscular overstrain.[1,3-5] Additional terms used to identify overtraining

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include *staleness, chronic overwork, burnout, overfatigue,* and *overreaching*.[1,2] Previous investigations have studied the various factors contributing to resistance-exercise overtraining. Among these factors are training volume, rest between exercise bouts, and exercise intensity.[1-6] Resistance-exercise overtraining from excessive relative intensity (percent 1-repetition-maximum [1RM]) has produced different physiological responses than has aerobic overtraining or resistance-exercise overtraining from increased training volumes.[4,5] For example, circulating hormones and catecholamines exhibit different profiles with the different types of overtraining stimuli.[1,2,5] Therefore, increased-intensity overtraining with resistance exercise appears to be a unique stimulus.

Overtraining includes such factors as inappropriate rest and recovery from training and results in increased levels of fatigue.[1] It can result in joint impairment known as overload injury, with the knee being a common site of impairment.[7,8] Problems occurring in this joint include pain from increased load factors caused by prolonged stress on knee cartilage, as well as swelling and instability.[9] Overuse problems are usually the result of increased volume or intensity in an athlete's training. The net result is often termed overtraining syndrome, which implies "an impaired state of health which is caused by overtraining."[10,p1] The purpose of this study was to observe the characteristics of an individual who was medically diagnosed with overload injury of the knee joint as a result of a 2-week, high-intensity, resistance-exercise overtraining (OT) protocol that was part of previously published research.[4,5] By studying this individual we wanted to see how this condition developed and how performance was affected by a condition we will designate a joint-centered overtraining syndrome.

Methods

Subjects

Eleven weight-trained men participated in a resistance-exercise overtraining protocol. Of these 11, 1 (subject A) was diagnosed with overload injury of the knee joint. All 11 subjects had consistently weight trained for at least 1 year prior to this study, and all were capable of a parallel barbell squat of at least 1.5 times their body weight for a 1RM. There was no history of anabolic steroid use over the previous year. Clinical knee examinations were administered to all subjects by a medical doctor (JML) before the investigation. The knee exam was performed with the subject in a supine position. The knee was flexed to 15° (a standard Lachman position). The distal femur was stabilized while anterior and posterior force was applied to the proximal tibia until the examiner felt the tautness of the respective cruciate ligaments. Millimeters of movement anterior and posterior were estimated. Medial and lateral laxity of each knee were also determined. The subject's ankle was fixed between the examiner's elbow and chest wall

Table 1 Descriptive Characteristics of Subject A and OT Group ($x \pm SE$, N = 10)

Variable	Subject A	OT group
Age (y)	21	22.1 ± 0.9
Height (cm)	174	176.9 ± 2.3
Body mass (kg)	64.5	85.3 ± 4.9
% body fat	3.9	13.1 ± 1.9

while both joint lines were palpated. The examiner exerted medial and lateral pressure on the knee and estimated the number of millimeters the joint space increased.[11-13] All subjects signed an informed consent statement as approved by The Pennsylvania State University Institutional Review Board before the study began. Characteristics for all subjects are presented in Table 1.

Training Protocol

Lower-body training was performed on a resistance-exercise machine that simulated a parallel-squat motion (Tru-Squat, Southern Xercise Inc, Cleveland, Tenn). A constant shoulder-width stance was used throughout the study, and the torso was maintained in a vertical position. This equipment has been previously described in detail.[3-5] Two familiarization sessions on the squat machine were conducted during the first week of the study. Both sessions included a 1RM assessment using a previously recommended progression.[14] Test–retest reliability for 1RM was $r = .96$. At the beginning of each training session during the 2-week overtraining period, the subjects were tested for 1RM.[14] To induce overtraining symptoms, all subjects performed 10 sets of 1 repetition at 100% of 1RM on the squat machine each day for 2 weeks. All repetitions were separated by 2-minute intervals. If there were any unsuccessful attempts, the load was decreased by 4.5 kg, allowing each successful lift to be as close as possible to the subject's maximal capacity.

Strength Tests

Test batteries were administered before, during, and after the 2-week overtraining period. Body composition was estimated from skinfold measurements.[15,16] Tester reliability for each skinfold site was $r \geq .94$. Capabilities for 1RM were determined for all subjects by using the same warm-up progression.[3-5,14] Subjects were also tested for concentric leg-extension peak torque (N · m) of their dominant leg at angular limb velocities of 0.00, 0.53, and 5.24 rad/s on a Cybex II isokinetic dynamometer (Lumex Inc,

Ronkonkoma, NY). Before each test, the dynamometer was calibrated according to the manufacturer's specifications. Isometric testing for the leg included a 5-second maximal effort with the knee at 90° flexion (0° = leg fully extended).

Stimulated quadriceps force was also assessed at 90° knee flexion. Surface stimulation was applied to the femoral nerve with a Teca Electromyograph/Synchronized Stimulator with built-in oscilloscope (model B-2) and a Teca 9523 handheld stimulator at the inguinal fold, immediately distal to the inguinal ligament.[17] Maximal muscle activation of the vastus medialis was determined by electromyographic (EMG) activity, using 2 silver–silver chloride electrodes with a built-in preamplifier and a constant electrode distance of 2 cm. Maximal activation of the quadriceps was verified by the amplitude of the EMG signal on the oscilloscope. Beginning with the single rectangular-wave stimulations of 0.05-millisecond duration, the nerve stimulation was increased in steps of approximately 60 V, followed by duration increases to a maximum of 2.0 milliseconds until the EMG signal did not increase in amplitude. To ensure complete activation, supramaximal stimulation of 30–60 V was added to maximal activation. The stimulated force tests were highly repeatable and were performed under the direction of a clinical neurologist. Isometric peak torque was determined by the Cybex dynamometer. Each muscle-force assessment was performed 3 hours postexercise and at the same time of day to help ensure acute neural recovery.[5] Training protocols were held constant before each test because short-term training can affect efficiency of electrical activity of skeletal muscles.[18] At the beginning, middle, and end of the 2 weeks of overtraining, each subject performed a maximum number of repetitions for the 70% 1RM test. After a warm-up of 3 repetitions at 32 kg, sets of 10 continuous repetitions at 70% of the most recent 1RM load were performed to fatigue, separated by 1-minute rests.

Statistical Analyses

All data are reported as $x \pm$ SE. Results for the OT group were analyzed with one-way repeated-measures analyses of variance. Post hoc analyses were performed with the Fisher least-significant-difference procedure. Significance for this study was set at $P < .05$.

Results

The subjects in the present study, as previously reported, were overtrained, exhibiting significant decreases in 1RM strength of –12.2 kg.[5] During the overtraining protocol, subject A was diagnosed with overload injury of the knee joint by a medical doctor (JML). He showed slight effusion of the knees bilaterally. Tests performed included anterior/posterior drawer, for stability of the anterior and posterior cruciate ligaments. The Lachman test

was also used to show stability of the anterior cruciate ligament alone. The McMurray test was used to diagnose any disruption of the cartilage. All tests for all subjects presented negative results. Subject A also demonstrated normal range of motion in both knees. Physical characteristics for subject A and the OT group are listed in Table 1. During the course of the overtraining, both subject A and the OT group exhibited attenuated 1RM strength (subject A = –40.3 kg, OT group = –9.3 kg; see Figure 1). Muscle endurance at 70% 1RM did not significantly change for the OT group but increased from 23 repetitions to 57 repetitions for subject A (see Figure 2). Isokinetic knee-extension strength decreased for both the OT group and Subject A at both 0.52 and 5.23 rad/s (see Figure 3). Maximal voluntary isometric knee-extension strength did not significantly change for the OT group but increased 13.7% for subject A. There was also a significant decrease in stimulated isometric knee-extension strength for the OT group, whereas this variable increased 14.0% for subject A (see Figure 4). During actual training sessions, subject A exhibited a unique phenomenon wherein the involved muscles appeared to shut down as the point in the range of motion for the squat approached the greatest torque forces.

Discussion

Both the OT group and subject A exhibited 1RM strength decrements by the end of this investigation. Additional aspects of this physiological

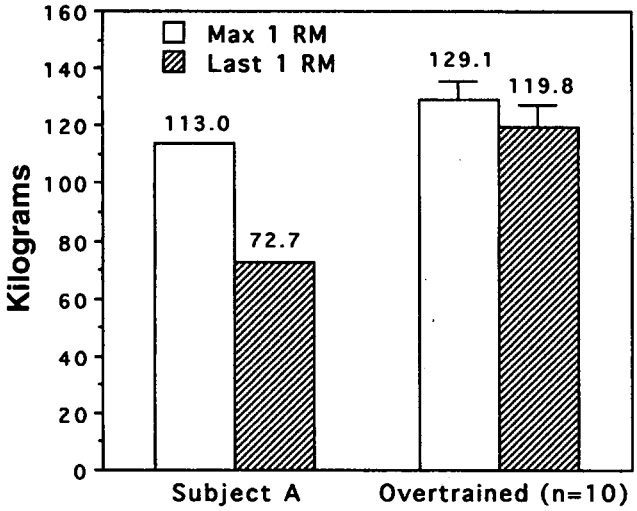

Figure 1 One-repetition-maximum (1RM) performances ($x \pm$ SE). Performances were compared between the maximum 1RM performed during the course of the study (max 1RM), and the 1RM performed on the last day of the study (last 1RM). The OT group exhibited significant decreases by the end of the study ($P < .05$).

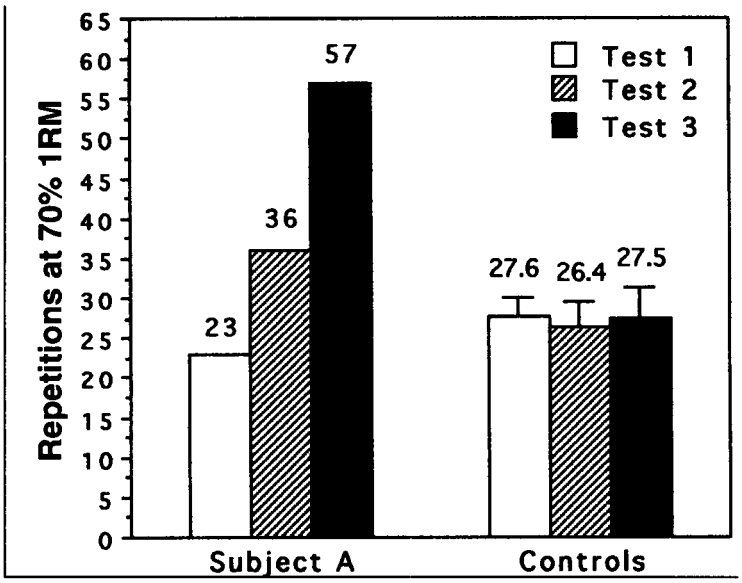

Figure 2 Repetitions at 70% of 1 repetition maximum (1RM; $x \pm SE$).

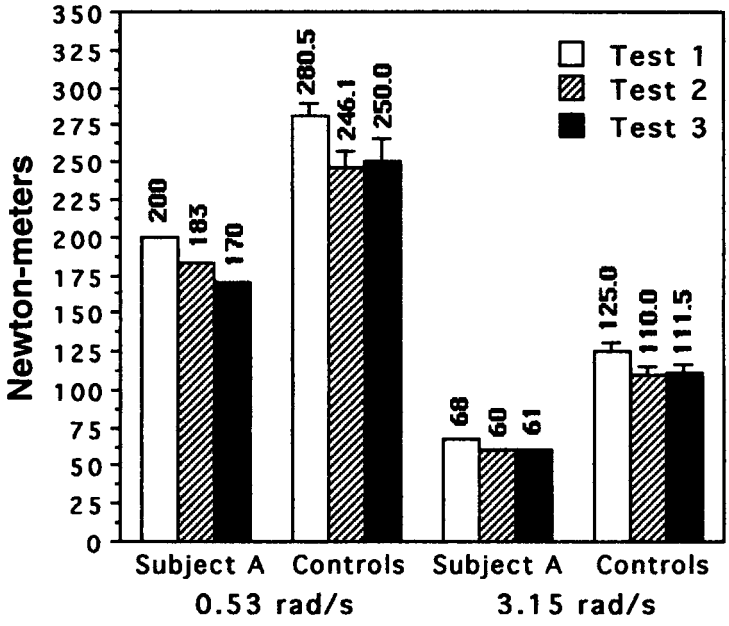

Figure 3 Isokinetic knee-extension strength (N · m) at 0.52 and 5.23 rad/s ($x \pm SE$). The OT group exhibited significant decreases at both velocities by test 2 ($P < .05$).

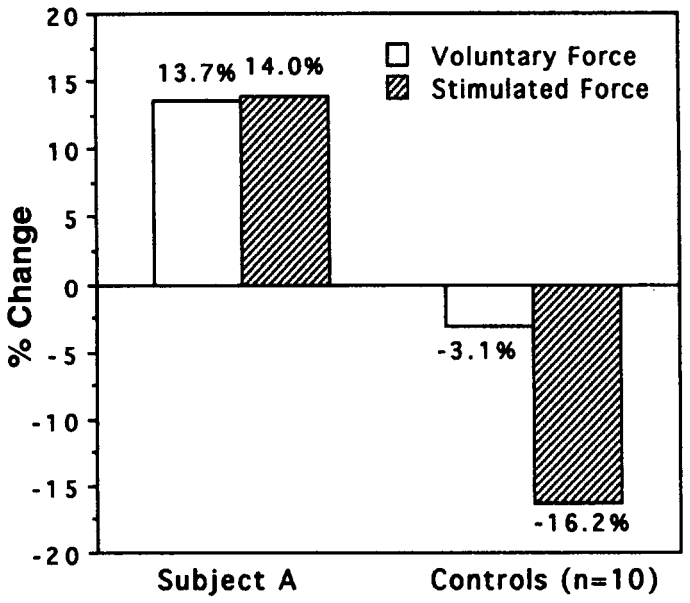

Figure 4 Percent changes for stimulated and voluntary isometric knee-extension strength ($x \pm SE$).

scenario have been previously described.[3-5] Besides 1RM decrements, isokinetic and involuntary knee-extension strength were attenuated, and a sympathetic overtraining syndrome[1] was indicated by augmented exercise-induced catecholamines. What is unique to this case study is the clinical diagnosis of overload injury of the knees for subject A, while all other subjects did not demonstrate any joint abnormalities. As indicated by the preinvestigation joint-laxity examinations, all subjects exhibited normal joint characteristics before the study.[11-13] It was only after initiating the high-intensity training protocol that subject A developed the overload injury. Although the OT group also demonstrated 1RM strength decrements, the resulting strength differences between the OT group and subject A were dramatic.

The additional strength measures used for this investigation permit a closer examination of possible physiological mechanisms that might have contributed to the strength decrements for subject A. Because relative muscle endurance (repetitions at 70% 1RM) actually increased for subject A, it appears that only maximal strength was adversely affected. Relative muscle endurance typically does not change when 1RM strength is altered.[14] In the case of subject A, the large increase in repetitions at 70% 1RM indicates that submaximal muscle forces did not decrease. It is possible that a force threshold existed for subject A, above which muscle performance decreased but below which strength was not affected.

It is also interesting to note that when maximal strength was assessed, only dynamic strength was impaired for subject A. Voluntary and stimulated isometric strength actually increased for subject A, whereas the OT group demonstrated no change (voluntary) or decreases (stimulated) for these measures. Changes for involuntary stimulated strength indicate adaptations of the peripheral musculature.[5] The distinct difference in stimulated strength between the OT group and subject A suggests that peripheral muscle maladaptation occurred for only the OT group. Such a result also suggests that the decreased 1RM for subject A is not the result of impaired intrinsic capacity of the peripheral musculature.[5]

Such results beg the question, Why was dynamic strength impaired for subject A but not for the OT group? Considerable clinical and experimental evidence exists indicating that joint impairment results in attenuated performance of the involved muscles.[9,19-22] Such phenomena are attributed to afferent inhibitory input from the affected joint. Inhibitory input of this type would serve as a very helpful protective mechanism for the impaired joint. Neural input to the musculature is very sensitive to a preceding exercise stimulus[6,18] and might have served as an immediate protective mechanism for the overload injury experienced by subject A. Severe injury to the knee, such as anterior-cruciate-ligament damage, results in attenuated knee-extensor strength,[23,24] because of not only muscle atrophy but also decreased central nervous system activation.[23,25] It should be noted, however, that even an inflammatory response of the knee will also result in attenuated knee-extensor strength[26] because of decreased central nervous system activation[23,24,27] and that such decreases in strength are readily measurable via isokinetic or gait assessments.[25,27] Whether the decreased 1RM strength exhibited by subject A is a result of afferent inhibition or decreased central nervous system muscle activation, the involved knee joints are somewhat protected from further damage during dynamic actions. From empirical observations of the training sessions, the inhibition and/or decreased activation occurred most dramatically when subject A approached the point of greatest knee torque in the range of motion. By inhibiting muscle activity at this point, the involved muscles were protected from the highest torque forces. This did not occur for those in the OT group. By avoiding these high forces, subject A's quadriceps actually were allowed partial recovery, resulting in augmented strength for tasks not involving joint movement (voluntary and stimulated isometric strength).

In conclusion, it appears that resistance-exercise overtraining resulting from excessive relative training intensity (% 1RM) might be manifested through a joint-centered mechanism. This appears to be different from the maladaptation of the peripheral muscles that has been previously described for this type of overtraining.[5] It is already evident that overtraining can exhibit a variety of physiological characteristics[1,3-5] and that different training modalities can result in different physiological characteristics.[2] In light

of these findings, it should be noted that joint- centered resistance-exercise overtraining appears to possess different symptoms than those of non-joint-centered resistance-exercise overtraining.

References

1. Stone MH, Keith RE, Kearney JT, Fleck SJ, Wilson GD, Triplett NT. Overtraining: a review of the signs, symptoms, and possible causes. *J Appl Sports Sci Res.* 1991;5:35-50.
2. van Borselen F, Vos NH, Fry AC, Kraemer WJ. Overtraining: the role of anaerobic exercise. *Natl Strength Cond Assoc J.* 1992;14(3):74-79.
3. Fry AC, Kraemer WJ, Lynch JM, Triplett NT, Koziris LP. Does short-term near-maximal intensity machine resistance training induce overtraining? *J Strength Cond Res.* 1994;8:188-191.
4. Fry AC, Kraemer WJ, van Borselen F, et al. Catecholamine responses to short-term high-intensity resistance exercise overtraining. *J Appl Physiol.* 1994;77:941-946.
5. Fry AC, Kraemer WJ, van Borselen F, et al. Performance decrements with high-intensity resistance overtraining. *Med Sci Sports Exerc.* 1994;26:1165-1173.
6. Häkkinen KA, Pakarinen M, Kauhanen AH, Komi PV. Neuromuscular and hormonal responses in elite athletes to two success strength training sessions in one day. *Eur J Appl Physiol.* 1980;57:133-139.
7. Johnson PH. Get that knee going (restoration of knee function). *J Ark Med Soc.* 1975;72:281-284.
8. Kibler WB, Chandler TJ. Musculoskeletal and orthopedic considerations. In: Kreider RB, Fry AC, O'Toole ML, eds. *Overtraining in Sport.* Champaign, Ill: Human Kinetics; 1998:169-190.
9. Renstrom P. Overuse injuries. In: Dirix A, Knuttgen HG, Tittel K, eds. *The Olympic Book of Sports Medicine.* Oxford, England: Blackwell Scientific Pubs; 1991:446-468.
10. Lehmann M, Foster C, Gastmann U, Keizer H, Steinacker JM. Definitions, types, symptoms, findings, underlying mechanisms, and frequency of overtraining and overtraining syndrome. In: Lehmann M, Foster C, Gastmann U, Keizer H, Steinacker JM, eds. *Overload, Performance Incompetence, and Regeneration in Sport.* New York, NY: Kluwer Academic/Plenum Pubs; 1999:1-6.
11. Hoppenfield S. *Physical Examination of the Spine and Extremities.* Norwalk, Conn: Appleton-Century Crofts; 1976.
12. Magee DJ. *Orthopedic Physical Assessment.* Philadelphia, Pa: WB Saunders; 1987.
13. Tory JS, Conrad W, Kalen V. Clinical diagnosis of anterior ligament instability in the athlete. *Am J Sports Med.* 1976;4(2):84-91.
14. Kraemer WJ, Fry AC. Strength testing: development and evaluation of methodology. In: Maud PJ, Foster C, eds. *Physiological Assessment of Human Fitness.* Champaign, Ill: Human Kinetics; 1995:115-138.

15. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. *Br J Nutr.* 1978;40:497-504.

16. Siri WE. Body composition from fluid spaces and density: analysis of methods. In: Brozek J, Henschel A, eds. *Techniques for Measuring Body Composition.* Washington, DC: National Academy of Sciences; 1961:223-224.

17. Delisa, JA, Mackenzie K, Baran EM. *Manual of Nerve Conduction Velocity and Somatosensory Evoked Potentials.* 2nd ed. New York, NY: Raven Press; 1987.

18. Fry AC. The effects of acute training status on reliability of integrated electromyographic activity and "efficiency of electrical activity" during isometric contractions: a case study. *Electromyogr Clin Neurophysiol.* 1992;32:565-570.

19. DeAndrade JR, Grant C, Dixon AS. Joint distention and reflex muscle inhibition in the knee. *J Bone Joint Surg Am.* 1965;47:313-322.

20. Freeman MAR. Instability of the foot and ankle. *J Bone Joint Surg Br.* 1965;47:669-677.

21. Sherman KS, Shakespeare DT, Stokes M, Young A. Inhibition of voluntary quadriceps activity after meniscectomy. *Clin Sci.* 1983;64:70.

22. Stratford P. Electromyography of the quadriceps femoris muscles in subjects with normal knee and acutely effused knees. *Phys Ther.* 1982;62:279-283.

23. Freiwald J, Reuter I, Engelhardt M. Neuromuscular and motor system alterations after knee trauma and knee surgery. In: Lehmann M, Foster C, Gastmann U, Keizer H, Steinacker JM, eds. *Overload, Performance Incompetence, and Regeneration in Sport.* New York, NY: Kluwer Academic/Plenum Press; 1999:81-100.

24. Lass P, Kalund S, LeFevre S, Arendt-Nielsen L, Sinkjaer R, Simonsen O. Muscle coordination following rupture of the anterior cruciate ligament. *Acta Orthop Scand.* 1991;62:9-14.

25. Andriacchi TP, Birac D. Functional testing in the anterior cruciate ligament deficient knee. *Clin Orthop Rel Res.* 1993;288:40-47.

26. Kniffki KD, Schomburg ED, Steffens H. Synaptic responses of lumbar alphamotoneurons to chemical algesic stimulation of skeletal muscle in spinal cats. *Brain Res.* 1979;160:549-552.

27. Schmalz T, Blumentritt S, Wagner R, Gokeler A. Ganganalytische Verlaufsuntersutung patellasehnenversorgter rupturen des vorhandenen kreuzbandes. *Phys Rehab Kur Med.* 1998;9:1-8.