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Summary / Sumario

Low Back Pain and sport; what role the pelvic ring?

Dolor lumbar y deporte: ¿cuál es el papel del anillo pélvico?

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The twentieth century epidemic of low back pain has continued unabated into the 21st century, Up to 20% of the Australian population will experience low back pain at some stage of their lives[1]. Causes of low back pain remain protean and obscure to the point where 85% of patients[2] will be classified as having 'non-specific low back ain' (NSLB). It is a nihilistic exercise and ultimately, an admission of the inability to establish an accurate or specific clinical diagnosis. The world of sport has not escaped the problem: a review of the literature suggests that in the context of sport up to 15% of injuries involve the spine[3] regardless of the type of sport: soccer[4], sailing[5], hockey[6,7], golf[8,9], swimming[10], gymnastics and dancing[11], among others. These studies do not include injuries involving muscle attachments to the pelvic ring (hamstrings, adductors, etc.), which is technically part of the lower back[12] and has evident biomechanical and functional connections with the spine.

Extensive research into hamstring and groin injuries has yielded increased knowledge and consensus statements, but frustratingly poor results in terms of primary prevention and avoidance of recurrences[13]. Several years ago Mendiguchia *et al*[14] had already raised the obvious question in their insightful editorial: "are we heading in the right direction?" It is difficult to achieve meaningful results by looking at single parameters, when the origin of these pathologies is multifactorial. A different approach is warranted to remove the sports medicine community from this frustrating scenario. There is now emerging evidence that a multifactorial rehabilitation algorithm appears to yield better results[15].

Since the 1980's there has been a growing interest in the role of the sacro-iliac joint (SIJ) in the biomechanics of the lumbar spine and as a source of pain. The three yearly World Congresses on Low Back and Pelvic Pain have witnessed a dialogue between clinicians and researchers that has delivered much of the progress made in the last 25 years. From the 6th World Congress in Barcelona (2007) the sports medicine community has been an integral part of this dialogue. The dual mechanical role of load transmission and absorption of torsional stresses led to the proposed integrated model of function and the concepts of force and form closure[16], a model that could greatly assist researchers in the sports medicine field.

The early work of Mens[17] and colleagues established that in 40% of footballers with groin pain the cause of the problem was poor load transfer through the SIJ. This basic understanding of pelvic biomechanics has facilitated the establishment of validated clinical examination standards. The European Guidelines - COST ACTION B13 "Low back pain: guidelines for its management" was issued by the European Commission, Research Directorate-General, Department of Policy, Coordination and Strategy. It included a Working Group B4 to work on the European guidelines for the diagnosis and treatment of pelvic girdle pain[18]. These evidence-based guidelines stated that pelvic girdle pain is a group within the general classification of low back pain, and that the SIJ is a contributor to both. Diagnostic and treatment guidelines have become available for the practicing clinician to alleviate the burden of disease to what has been estimated 20-25% of patients diagnosed with "low back pain". This has shown success in approximately 80% of cases with directed physiotherapy[19].

The traditional imaging of the SIJ (X-rays, CT scan, scintigraphy and more recently magnetic resonance imaging) has proved its success in the diagnosis of many conditions, from trauma (fractures) to infection, tumours and inflammatory arthropathies. Only in recent years has the combination of scintigraphy with low-dose x-ray computed tomography (CT) – single photon emission computed tomography SPECT/CT been able to confirm the biomechanics of the SIJ both in a disease-free population and in those with mechanical failure of the joint[20].

The term sacroiliac joint incompetence was coined to encompass both the post-partum variant of the pelvic girdle pain syndrome and localised trauma to the joint or pelvis. This is a relatively common condition that may account for over 20% of low back pain, especially after repeated pelvic micro-trauma (overuse due to falls, dismounts, jumps, in the sporting field), very low speed motor vehicle accidents or in women in the peri-partum period or in the puerperium. Many of these patients have previously been classified as either NSLBP or worse, as malingerers or manifestations of psychiatric disease. The clinical diagnosis requires meticulous attention to detail and expertise in physical examination that may be problematic in general usage. The majority of patients in one study had reportedly normal MRI studies, adding to the difficulty in identification by the standard medical paradigms. More recently, in a cohort of 1200 patients with the clinical diagnosis of SIJ incompetence and radiological confirmation (with SPECT/CT significant enthesopathies

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were identified: hamstringss), adductors in over 70% of patients BEFORE they had developed clear symptoms of tendinitis, tendinosis or frank muscular tears[21]. Gluteus medius tendinopathy and hip impingement paralleled these findings. A significant small group of elite athletes (n=23) were part of this large cohort as their presenting pathology was a hamstring strain or tear rather than low back pain. It is therefore reasonable to think that many of these hamstrings (which have a predominance of Type II fibres) are forced on to a dual function of core stability in addition to fast movement, and the injuries the result of overuse.

In the context of sport, notably soccer, Nordic eccentric strengthening of hamstrings has been advocated as an effective strategy for the rehabilitation of hamstring injuries. Mendiguchia, *et al.* have argued however that this requires eccentric strengthening of knee flexors with the hip in a fixed position. Furthermore, this requires a stable pelvis, i.e. a sacro-iliac joint that transmits loads correctly, in other words, appropriate core stability. This begs the question: are Nordic hamstring exercises effective as a result of a stable pelvis (i.e. with adequate dynamic neuromotor control)? An interesting question that warrants further research.

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Original article

Hypertrophy training improves glycaemic and inflammatory parameters in men with risk factors

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Summary

Background and aims: A close link between metabolic syndrome (MS), insulin resistance, chronic low-grade inflammation and cardiovascular diseases has been highlighted in the literature. However, resistance training (RT) has shown interesting results on inflammatory mediators, adipokines, and insulin-related parameters in this population, although results are still contradictory. This study aimed to investigate the effects of hypertrophy RT on glycaemic, cytokines and adipokines levels in men with MS risk factors.
Methods: Twenty-one untrained men (57.8 ± 7.74 years old) underwent a RT for 15 weeks (3 times per week), comprised of nine exercises performed predominantly in the hypertrophy zone. Blood samples were drawn for analysis of glycaemic, inflammatory and hormonal parameters. Subjects were encouraged to maintain their habitual dietary intake during the intervention and dual-energy X-ray absorptiometry was used to assess body composition.
Results: Levels of interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interleukin-18 (IL-18), tumor necrosis factor alpha (TNF-α), interferon-gamma (IFN-γ), resistin, ghrelin and leptin decreased, while interleukin-10 (IL-10) and adiponectin concentrations increased after RT. Moreover, the intervention improved glycaemic and insulinemic parameters, besides body composition. Body mass, abdominal and waist circumferences, besides total cholesterol and triglycerides levels remained unaltered.
Conclusion: Positive modulation of glycaemic, insulinemic and inflammatory parameters are found in men with MS risk factors after 15 weeks of hypertrophy resistance training, parallel with improvements on body composition and independent of weight loss.

Key words:
Strength training. Inflammation. Health. Diabetes Mellitus. Exercise.

El entrenamiento de hipertrofia mejora los parámetros glucémicos e inflamatorios en hombres con factores de riesgo

Resumen

Antecedentes y objetivos: Se ha destacado en la literatura un estrecho vínculo entre el síndrome metabólico (SM), la resistencia a la insulina, la inflamación crónica de bajo grado y las enfermedades cardiovasculares. Además de varios beneficios, el entrenamiento de resistencia (ER) ha producido resultados contradictorios en citoquinas, citoquinas derivadas de tejido adiposo y niveles de parámetros relacionados con la insulina. Este estudio tuvo como objetivo investigar los efectos del ER de hipertrofia como una sola intervención en los niveles de glucemia, citoquinas y adipoquinas en hombres con factores de riesgo de SM.
Métodos: Veintiún hombres sedentarios (57,8 ± 7,74 años) se sometieron a ER durante 15 semanas (3 veces por semana), compuesto de nueve ejercicios realizados predominantemente en la zona de hipertrofia. Se tomaron muestras de sangre para el análisis de parámetros glucémicos, inflamatorios y hormonales. Los sujetos fueron alentados a mantener su ingesta dietética habitual durante la intervención y se utilizó la absorciometría de rayos X de energía dual para evaluar la composición corporal.
Resultados: Los niveles de interleucina-1 beta (IL-1β), interleucina-6 (IL-6), interleucina-18 (IL-18), necrosis tumoral alfa (TNF-α), interferón gamma (IFN-γ), resistina, grelina y leptina disminuyeron, mientras que las concentraciones de interleucina-10 (IL-10) y adiponectina aumentaron después del ER. También, la intervención mejoró los parámetros glicémico e insulinémico, además de la composición corporal. La masa corporal, la circunferencia abdominal y la cintura, además del colesterol total y los triglicéridos permanecieron inalterados.
Conclusión: La modulación significativa y positiva en los parámetros sistémicos glicémicos, insulinémicos e inflamatorios ha sido encontrada en los hombres con factores de riesgo de SM después de 15 semanas de entrenamiento de resistencia a la hipertrofia, paralelamente con mejoras en la composición corporal e independiente de la pérdida de peso.

Palabras clave:
Entrenamiento de fuerza. Inflamación. Salud. Diabetes Mellitus. Ejercicio.

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Introduction

The metabolic syndrome (MS) comprises insulin resistance, dyslipidemia, hypertension and abdominal obesity, and it is associated with a lifestyle encompassing excessive energetic intake and low physical activity levels[1]. In this regard, it is estimated that 25% of the worldwide' adults have MS[2]. In Brazil, MS prevalence is higher in middle-aged men than aged-matched women, with a prevalence ranging from 34% up to 79%, depending on overweight or obesity status, respectively[3]. Moreover, cardiovascular disorders such as abdominal aortic aneurysm, coronary heart disease, peripheral arterial disease and cerebrovascular diseases are closely related with MS prevalence[4].

One of the main factors related to MS development is abdominal obesity[1]. Adipose tissue is recognized not only as a passive fat storage, but also an active metabolic and endocrine organ that secretes several peptide hormones responsible for energy balance, appetite modulation and inflammation, such as leptin, adiponectin, resistin, interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α)[5]. In this regard, an imbalanced chronic inflammatory status is closely linked to abdominal obesity, atherosclerosis, age-related sarcopenia and type 2 diabetes mellitus (T2DM)[6]. In fact, an infiltration of immune cells in adipose tissue, muscle, liver and pancreas has been associated with a shift from an anti-inflammatory to a pro-inflammatory frame that may disrupt insulin signaling in peripheral tissues and induce β-cell dysfunction[7].

Recent studies have also linked MS and obesity to poorer cancer outcomes including increased risk of recurrence and overall mortality[8]. Considering that higher levels of muscular strength are associated with lower cancer mortality risk in men[9] and in order to avoid the progression of obesity, subclinical inflammation[10] and insulin resistance[11] in middle-aged men, resistance training (RT) has been indicated. However, RT has produced conflicting results on inflammatory cytokines, adipose-derived cytokines (adipokines) and insulin-related parameters levels[12,13]. In fact, most studies concerning RT and high risk populations have utilized training intensities below 80% of one repetition maximum (1RM)[10-12], leaving aside possible benefits of hypertrophy RT programs on inflammatory profile. Therefore, the aim of this study was to investigate the effects of hypertrophy resistance training on glycaemic, cytokines and adipokines levels in men with metabolic syndrome risk factors.

Material and method

Subjects

After advertisements of the study and fully informed about the protocol, twenty-five men were recruited. The following inclusion criteria were considered: untrained[14] men aged between 40 and 65 years, that had at least two MS risk factors, such as triglycerides (TG) levels ≥150 mg/dL or specific drug treatment, high-density cholesterol (HDL) levels ≤40 mg/dL or specific drug treatment, fasting glucose levels ≥100 mg/dL or specific drug treatment, systolic blood pressure ≥130 and/or diastolic ≥85 mmHg or specific drug treatment and waist circumference (WC) ≥90 cm[1]. Moreover, volunteers were instructed to maintain their habitual food intake during the protocol. This study was approved by the Ethics Committee of the Federal University of Santa Maria (UFSM) (permit number: 0032.0.243.000-07), followed the statements of the Declaration of Helsinki and all participants signed a written informed consent.

Anthropometric Measurements

Subjects were weighted in a scale (Plenna, São Paulo, Brazil) and heighted with a stadiometer (Cardiomed, Curitiba, Brazil). The abdominal circumference was measured with a spring-loaded metal tape (Cardiomed, Curitiba, Brazil). Body composition was determined using dual-energy X-ray absorptiometry (DXA) with a densitometer machine (Hologic QDR Discovery, Waltham, USA) with the software "Body composition with sub regional analysis". Briefly, after 12 h fasting and 24 h without exercises and wearing only a light coat, subjects were laid in the designed corrected position on the DXA table and were instructed to remain still throughout the scanning procedure.

Functional Assessments

All tests described below were performed at same time of day, before and after the RT. A submaximal test was used to estimate 1RM in the bench press, rower machine, leg press and knee flexion machines. This test was utilized to estimate the largest load that an individual can move in a single maximal effort, and thus, to prescribe the training load[15,16]. Resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels were measured with a digital sphygmomanometer (Omron, Kyoto, Japan). Furthermore, flexibility of lumbar and hamstring muscles was assessed by the sit-and-reach test[16] and the longest distance reached on the measuring board was registered after three attempts. The cardiorespiratory fitness was assessed by Bruce's modified protocol[17] in a treadmill.

Resistance Training

The supervised RT was performed three days per week during 15 weeks, with 48-72 h of recovery between sessions. The RT protocol was briefly adapted from a previous study[18]. Sessions started with a low-intensity indoor walking for 10 min and was followed by the performance of alternating upper and lower limbs, and trunk exercises. Volunteers performed nine exercises: chest press, leg press, rower machine, leg curl, triceps extension, leg extension, biceps curl, trunk extension and abdominals[19]. The first two weeks of RT consisted of two sets of 15 repetitions at 55% of one repetition maximum (1RM). In weeks 3 and 4, subjects performed three sets of 12 repetitions at 65% 1RM. During weeks 5 to 8, the intensity ranged between 70-75% 1RM, and three sets of 10 repetitions were performed. During the last seven weeks, subjects worked out with three sets of 8 repetitions at 80% 1RM, designed to induce muscle hypertrophy[20]. There were rest periods of 1-2 min between sets and exercises[21]. After training sessions, volunteers performed stretching exercises: upper and lower back, shoulders, arms, chest, abdomen, thighs (back, front, inner and outer) and calves.

Biochemical Assays

Blood samples were drawn in the morning (07:00-08:30 a.m.) from a vein of the antecubital region after 12 h of fasting and 72 h without exercise. Samples were collected into 4-mL serum separator or EDTA tubes (BD Diagnostics, Plymouth, UK), centrifuged at 1500 g for 15 min and supernatants were frozen at -80 ºC until analysis. Total cholesterol and HDL concentrations were determined using commercially available assay kits (Bioclin, Belo Horizonte, Brazil) on a Cobas MIRA® (Roche Diagnostics, Basel, Switzerland) automated analyzer. Serum TG and glucose levels were determined using commercial kits (Bio Técnica, Varginha, Brazil). The levels of low-density cholesterol (LDL) were estimated[22].

Serum levels of cytokines IL-1β, IL-6, IL-10, IL-18, TNF-α and interferon-gamma (IFN-γ) were determined by enzyme-linked immunosorbent assay (ELISA) using commercial kits (eBIOSCIENCE, San Diego, USA), according to manufacturer's instructions. IL-1β, IL-6 and IL-10 were sensitive to 2 pg/mL. TNF-α and IFN-γ were sensitive to 4 pg/mL and 4 µg/mL, respectively, while IL-18 was sensitive to 37 pg/mL. Plasma adiponectin (R & D Systems, Minneapolis, USA) and resistin (R & D Systems, Minneapolis, USA) were performed by ELISA, which was sensitive to 0.25 ng/mL and 0.023 ng/mL, respectively. Serum leptin and ghrelin (Diagnostic System Laboratories, Leawood, USA) were also analyzed by ELISA, which was sensitive to 0.05 ng/mL and 0.07 ng/mL, respectively. Insulin levels were also measured by ELISA using commercial kits (eBIOSCIENCE, San Diego, USA). Insulin resistance (IR) and beta cell function (BF) indexes were calculated using homeostasis model assessment (HOMA), where HOMA-BF: (fasting insulin [mU/L] x 20) / (fasting glucose [mmol/L] – 3.5) and HOMA-IR: (fasting insulin [mU/L] x fasting glucose [mmol/L]) / 22.5[23].

Nutritional Data

To minimize a possible bias, subjects were encouraged to maintain their habitual dietary intake during intervention and filled in a 3-day diet record before and after the RT. A specific software (Dietwin, São Paulo, Brazil) was used to determine total caloric intake and the amount of macronutrients ingested.

Statistical Analysis

Shapiro-Wilk test was carried out to verify data distribution. Afterwards, Student's t test or Wilcoxon Rank Test were used to determine significant differences between pre and post-training results. Statistical Package for Social Sciences (SPSS 14.0, Chicago, USA) was used and statistical significance was set at $p < 0.05$. Data were expressed as mean ± standard deviation of the mean (SD).

Results

Twenty-one men (57.8 ± 7.74 years old) concluded the RT and were considered in the statistical analysis. Furthermore, the sample comprised three smokers and 18 nonsmokers, 39% of men took antihypertensive agents, 19% took lipid-lowering agents and 4.75% took oral hypoglycemic agents. Table 1 shows the results of submaximal strength test before and after RT. Increases in the load lifted/moved in the bench press (p <0.001), leg press (p < 0.001), rower machine (p < 0.001) and knee flexion (p < 0.001) exercises were registered.

Furthermore, Table 2 demonstrates that RT resulted in significant improvements in hip circumference (p = 0.028), body fat (p = 0.011), lean mass (p = 0.018), and SBP (p = 0.023) levels, besides HDL reduction (p < 0.001). Moreover, the stretching performed before and after exercise sessions could have improved flexibility (p = 0.001). However, body mass, BMI, VO_{2max}, DBP, TG and total cholesterol levels remained unchanged.

It is observed in Table 3 that RT did not change insulin levels, while it decreased glucose levels (p < 0.001), HOMA-IR (p = 0.003) and increased HOMA-BF (p = 0.004).

No significant differences were found in total ingestion of calories and macronutrients, demonstrating the maintenance of habitual intake during the intervention (Table 4).

Changes in cytokines are given in Figure 1. Serum levels of IL-1β (p < 0.001), IL-6 (p < 0.001), IL-18 (p < 0.001), TNF-α (p < 0.001) and IFN-γ (p < 0.001) decreased after RT. Moreover, participants showed higher levels of IL-10 (p < 0.001) after intervention.

As shown in Figure 2, RT decreased resistin (77.8 ± 5.56 *vs.* 58.57 ± 8.11 ng/mL; p < 0.001), ghrelin (49.47 ± 5.7 *vs.* 40.23 ± 7.45 pg/mL; p < 0.001) and leptin (140.57 ± 7.76 *vs.* 83.9 ± 10.94 ng/mL; p < 0.001)

Table 1. Load moved in the strength test along intervention (n=21).

Exercises	Before	After
Bench Press (kg)	65.12 ± 16.79	74.11 ± 10.05**
Rower machine (kg)	49.93 ± 6.51	63.38 ± 8.36**
Leg Press (kg)	100.50 ± 14.57	119.03 ± 21.25**
Knee Flexion (kg)	18.46 ± 2.83	22.98 ± 3.42**

Values expressed as mean ± SD. * p < 0.05 and ** p < 0.001 after *vs.* before the resistance training.

Table 2. Effects of resistance training on anthropometric, functional and biochemical parameters of men with metabolic syndrome (n=21).

Parameters	Before	After
Body Mass (kg)	86.69 ± 13.82	86.32 ± 12.90
BMI (kg/m^2)	28.98 ± 4.43	28.86 ± 4.17
Abdominal Circumference (cm)	105.60 ± 13.60	104.53 ± 13.10
Waist Circumference (cm)	101.30 ± 12.07	100.30 ± 12.18
Hip Circumference (cm)	107.07 ± 10.33	105.31 ± 9.45*
Body Fat Mass (%)	32.51 ± 5.02	31.90 ± 5.15*
Body Lean Mass (%)	64.12 ± 4.73	64.68 ± 4.87*
Systolic Blood Pressure (mmHg)	131.95 ± 16.29	124.23 ± 17.67*
Diastolic Blood Pressure (mmHg)	78.76 ± 9.66	75.52 ± 9.28
Flexibility (cm)	17.73 ± 11.56	21.08 ± 10.97*
VO_{2max} (mL.kg^{-1}•min^{-1})	37.61 ± 7.66	38.41 ± 9.48
Total Cholesterol (mg/dL)	206.61 ± 46.95	208.85 ± 40.96
Triglycerides (mg/dL)	174.87 ± 82.62	176.71 ± 58.62
HDL (mg/dL)	52.04 ± 14.17	43.47 ± 8.78**
LDL (mg/dL)	119.59 ± 43.21	130.03 ± 39.85

Values expressed as mean ± SD. BMI: body mass index. VO_{2max}: maximal oxygen uptake. HDL: high-density cholesterol. LDL: low-density cholesterol. * p < 0.05 and ** p < 0.001 after vs. before resistance training.

Table 3. Effects of RT on glycaemic control parameters (n=21).

Exercises	Before	After
Glucose (mg/dL)	121.61 ± 34.28	96.09 ± 29.82**
Insulin (mU/L)	11.47 ± 5.96	10.42 ± 5.62
HOMA-BF (%)	87.25 ± 52.86	188.88 ± 174.7**
HOMA-IR index	3.54 ± 2.65	2.42 ± 1.36*

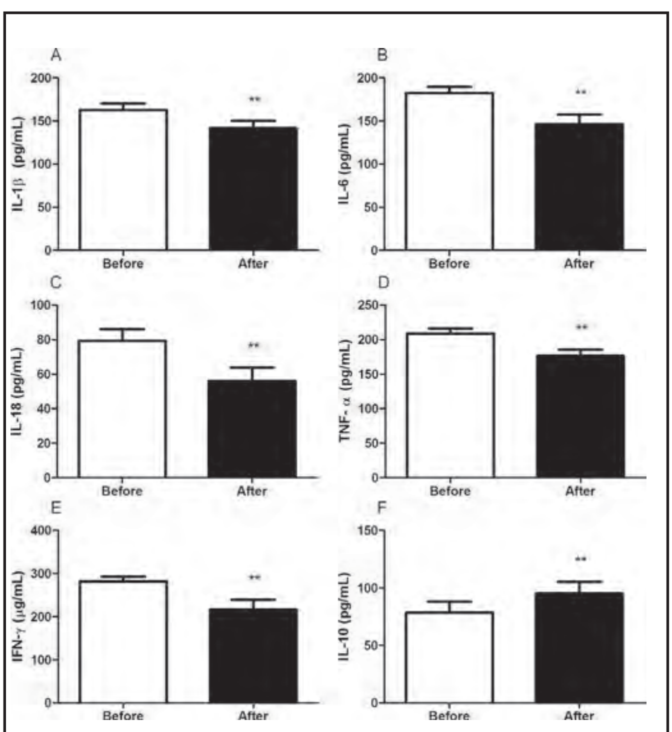
Values expressed as mean ± SD. *p < 0.05 and ** p < 0.001 after ***vs***. before the resistance training. HOMA-BF: homeostasis model assessment insulin resistance β cell function. HOMA-IR: homeostasis model assessment insulin resistance.

Table 4. Total calorie and macronutrients ingested before and after training (n=21).

Variables	Before	After
Total Caloric Intake (kcal)	2,731.19 ± 262.07	2,719.37 ± 220.97
Carbohydrates (g)	317.84 ± 29.57	314.83 ± 26.25
Proteins (g)	109.81 ± 15.22	113.04 ± 12.92
Lipids (g)	113.63 ± 17.85	111.98 ± 14.79

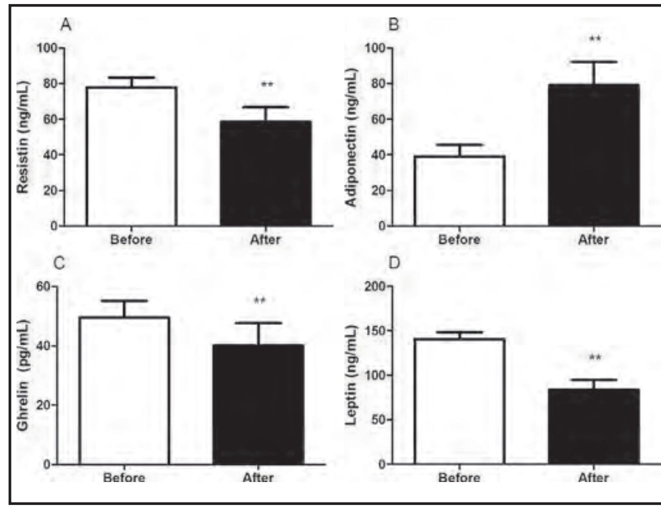
Values expressed as mean ± SD.

Figure 1. Effects of 15 weeks of hypertrophy resistance training on interleukin-1 beta (IL-1β) (A), interleukin-6 (IL-6) (B), interleukin-18 (IL-18) (C), tumor necrosis factor alpha (TNF-α) (D), interferon-gamma (INF- γ) (E) and interleukin-10 (IL-10) (F) levels in 21 men with metabolic syndrome risk factors.



Data are expressed as mean ± SD. * p < 0.05 and ** p < 0.001 after *vs*. before training.

Figure 2. Effects of 15 weeks of hypertrophy resistance training on resistin (A), adiponectin (B), ghrelin (C) and leptin (D) levels in 21 men with metabolic syndrome risk factors.



Data are expressed as mean ± SD. * p < 0.05 and ** p < 0.001 after *vs*. before training.

levels, while it resulted in increased levels of adiponectin (39.09 ± 6.41 *vs*. 79.14 ± 12.98 ng/mL; p < 0.001).

Discussion

This study aimed to investigate the effects of a supervised RT on glycaemic parameters, inflammatory and hormonal profile in men with MS risk factors. The main findings are that 15 weeks of hypertrophy RT reduced several pro-inflammatory cytokines, fasting glucose levels and HOMA-IR, together with improvements in body composition, even in the absence of weight loss. Moreover, RT increased loads moved during 1RM test, indicating a functional efficacy in the stimulus generated from training sessions. RT also resulted in modulation of resistin, ghrelin, leptin and adiponectin concentrations, independently of maintenance of total calorie and macronutrients ingested along the intervention.

Regarding criteria for the MS classification (SBP, DBP, WC, TG, HDL and glucose levels)[1], only fasting glucose concentrations and SBP were positively altered with the RT program. Indeed, a review with meta-analysis concerning the effect of RT on the treatment of MS characteristics and others variables showed no statistically significant effect of RT on HDL, TG and DBP[13]. Nevertheless, in the 13 interventions included in the aforementioned review, RT reduced resting SBP by 6.2 mmHg, similar with our findings. This SBP reduction is more prominent in RT programs with high volume (9 sets weekly per muscle group) than interventions with low volume (4-6 sets weekly per muscle group), and more pronounced in hypertensive patients at baseline[13]. This reduction of SBP induced by RT is independent of weight loss and probably linked with decreased catecholamine levels and systemic vascular resistance, with involvement of sympathetic nervous system and the renin-angiotensin system[13,24].

Changes in fasting glucose levels, HOMA-IR and HOMA-BF were observed after the hypertrophy RT. The improvements of insulin sensitivity and β-cell function in men with MS risk factors are in accordance with results of another study involving a similar protocol of hypertrophy RT with sedentary, however, young men[18]. Several mechanisms have been proposed to explain reductions in glucose concentrations and insulin resistance after a RT program. Considering that exercise training increases both transporters GLUT-4 messenger RNA (mRNA) and protein expression, it is noteworthy that the expression of GLUT-4 at the plasma membrane of myocytes is associated with increased fiber volume in both slow and fast fibers[25]. Moreover, improvement of insulin-stimulated glucose uptake after exercise training has been attributed to enhanced intracellular postreceptor signaling via phosphatidylinositol 3-kinase (PI3K) activity and/or its phosphorylation[26]. It has also been demonstrated increased protein content of protein kinase B (Akt), *Akt* substrate of 160 kDa (AS160), GLUT4 and hexokinase, besides elevated activities of *Akt* and glycogen synthase in basal and in insulin-stimulated glucose uptake conditions, respectively, both following exercise training in healthy men[27]. Considering that insulin resistance over time leads to T2DM and its secondary complications, an attenuated insulin resistance after RT in men with MS risk factor is of major importance. It may be, therefore, hypothesized that improved β-cell function is due to decreased hepatic gluconeogenesis, attenuated insulin resistance in muscles and slowly wakening of β islets to secrete insulin, together with modulation of cytokines released by myocytes and adipocytes[28].

Furthermore, exercise training may enhance muscular glucose uptake via insulin-independent mechanisms. After six weeks of RT with one leg while the other remained rested, it was reported increased protein content of AMP-activated protein kinase (AMPK) isoforms in trained compared with untrained muscles in healthy and T2DM patients, showing that RT results in an up-regulation of AMPK[29]. In addition, AMPK phosphorylates AS160 in response to muscle contraction, may result in muscle GLUT 4 expression, biogenesis and translocation[30]. Since disturbances in fatty acid metabolism and the consequent accumulation of diacylglycerol and ceramide impair insulin signaling in skeletal muscle, AMPK activation results in the up-regulation of fatty acid oxidation[26].

Following RT, there were reductions in leptin, resistin and ghrelin, as well as elevation in adiponectin levels. Leptin is a hormone released from adipose tissue that affects satiety and energy balance, may trigger the growth of several cancer cells, and when it signals directly to their receptors on the surface of mononuclear white cells (MNC), the syntheses of TNF-α and IL-6 is stimulated[31]. In this regard, IL-10 is an important physiological contributor to the central leptin action mediated by exercise[32]. Adiponectin is another mainly adipose tissue-derived protein inversely correlated with body fat levels and known by improving insulin sensitivity and increasing fat oxidation, presenting anti-atherogenic and anti-inflammatory properties[12,33]. Adiponectin binds to adiponectin receptors AdipoR1 and AdipoR2, producing beneficial on insulin sensitivity, glycaemia and lipid profile via activation of AMPK, peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PPAR-α) and p38 mitogen-activated protein kinase (P38 MAPK) pathways in skeletal muscle, adipose tissue and liver[34,35]. This link between improved glucose metabolism and adiponectin levels, as observed after our RT protocol, highlights a fine crosstalk between the different markers measured.

In an interesting study, the impact of three different intensities of RT on adipokines levels in sedentary elderly subjects was compared. Low (45-50% of 1RM), moderate (60-65% of 1RM) and high (80-85% of 1RM) intensities of training decreased leptin and increased adiponectin levels, however, the greater changes in both adipokines were found in the high-intensity group, showing an intensity-dependent effect[33]. Authors attribute the greater decline in leptin levels induced by the higher intensity due to augmented sympathoadrenal discharge and caloric expenditure, glycogen depletion and acidosis in the repeated sessions, besides long-term decreased body fat stores responsible by leptin secretion[33]. Moreover, only four weeks of intensive aerobic training increased the expression of AdipoR1 and AdipoR2 in skeletal muscle and subcutaneous adipose tissue and circulating adiponectin levels of individuals with normal or impaired glucose tolerance or T2DM[36].

Ghrelin is synthesized and secreted from the stomach and small intestine, being responsible for appetite-stimulating and anti-inflammatory functions[37]. Most investigations have demonstrated no effects of exercise training in the absence of weight loss on ghrelin levels[37]. In this regard, the intensity of our RT protocol may explain this change. A recent study showed that an intervention combining aerobic and resistance exercises produced increased levels of ghrelin and concomitant reductions in CD14+/CD16+ monocytes, possibly via interaction with its receptor, the growth hormone secretagogue receptor[37]. In addition to the discussed above, ghrelin, leptin and adiponectin may lead to the production of several cytokines from MNC[37].

In the present study, hypertrophy RT also positively modulated several cytokines levels, lowering the subclinical low-grade inflammatory status presented in patients with MS. According to the literature, RT has produced discrepant results on cytokines[12], depending on age of subjects, basal levels of cytokines, influence of the last exercise session, biomarkers assessed, differences in subject populations, variation in frequency, duration and intensity of RT, among others. Evidences have shown that TNF-α is the first cytokine produced by the inflammatory cascade, is related to lower muscle mass and it causes insulin resistance by triggering different key steps instead of the normal insulin signaling pathway, while IL-6 is a marker of the MS[38]. Still, IL-18 is closely related to the development of MS[39].

It is important to distinguish the effects of chronic elevated levels of IL-6 (released by adipocytes and/or infiltrated MNC) from the acute and drastic several fold IL-6 augmented levels provoked by muscle contractions (released by myocytes). Contrary to severe infections, exercise-induced IL-6 activation is independent of previous activation of TNF-α[38], since intramuscular IL-6 is regulated by calcium/nuclear factor of activated T cells, AMPK and glycogen/ P38 MAPK[38,40]. Moreover, studies have demonstrated that IL-6 released from myocytes is an essential regulator of skeletal muscle hypertrophy mediated by satellite-cells[41], stimulates glucose uptake, IL-10 production and inhibits TNF-α production[38]. The cumulative effect of transitory increases on IL-6 levels promoted by sessions with resistance exercises is responsible for an important part of the anti-inflammatory effect of RT. Furthermore, taking into account that adipose tissue is an endocrine organ[38], a reduction in the adipose tissue content may influence the production and releasing of pro-inflammatory markers and several adipokines, as confirmed in the present study. Lastly, it has also been shown that RT leads to reduced mRNA expression of toll-like receptor (TLR4) and mRNA TNF-α in monocytes[42].

In conclusion, significant and positive modulation in systemic glycaemic, insulinemic and inflammatory parameters are found in men with MS risk factors after 15 weeks of hypertrophy resistance training.

These findings are parallel with improvements on body composition and independent of weight loss. Thus, the present findings demonstrate that hypertrophy resistance training programs may serve as a strategy for treatment of populations at high cardiovascular risk. Limitations in the current study comprise the absence of a control group.

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Original article

Strategies to reduce pre-competition body weight in mixed martial arts

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Summary

Introduction: In Mixed Martial Arts (MMA), just like other combat sports categorized by body weight, some athletes use rapid weight loss techniques to have certain advantages in the competition.
Objective: Therefore, this study aimed to analyze the frequency of rapid body weight reduction in the period of 12 months before a competition and the methods used to achieve it, in a group of athletes from the City of Curitiba, Paraná, Brazil, from state and national competitions.
Materials and methods: Twenty-five fighters, mean age of 24.4 ± 4.1 years, participated in the study. To analyze the strategies used in body weight reduction, a validated 3-section questionnaire for weight loss in fighters was applied before weighing.
Results: Of total 25 volunteers, all 25 (100%) said that they have already used weight loss techniques before a competition. Most of them reported the loss of 1 to 18 kg three to sixty days before a competition, up to 10 times a year, using the following methods: increased physical activity, gradual diet, training in heated areas, and reduced fluid intake. The coach, training colleagues, and older athletes influenced the athlete's decision to adopt rapid weight loss methods as a supposedly competitive advantage.
Conclusions: In conclusion, rapid weight loss was frequent among all Mixed Martial Arts fighters investigated in this study, and the most commonly used methods were increased physical activity, gradual diet, training in heated areas, and reduced fluid intake.

Key words:
Weight loss. Athletes. Fighters.

Estrategias para la reducción de peso corporal en competición de artes marciales mixtas

Resumen

Introducción: En las artes marciales mixtas (MMA), al igual que en otros deportes de combate en los que existen categorías por masa corporal, es habitual el uso de estrategias de pérdida de masa corporal en fechas próximas a las competiciones con el fin de obtener algún tipo de ventaja.
Objetivo: El presente estudio tuvo como objetivo verificar la frecuencia de la reducción del peso corporal en los últimos 12 meses antes de la competición y los métodos utilizados para lograrlo, en un grupo de competidores a nivel estatal y nacional de Curitiba, Paraná, Brasil. Los participantes fueron 25 combatientes con una edad media de 25,4 ± 4,1 años.
Material y métodos: Para comprobar las estrategias empleadas en la reducción de la masa corporal, se aplicó a los deportistas un cuestionario validado, completado antes del pesaje.
Resultados: Los 25 sujetos (100%), indicaron que utilizaron técnicas para reducir su masa corporal para competir, con la finalidad de hacerlo en una categoría inferior. La mayoría indicó una pérdida de 1 a 18 kg en el período anterior a la competición, de una duración entre 3 a 60 días, de 1 a 10 veces al año a través de los siguientes métodos: Restricción de la ingesta de líquidos, sesiones de sauna, dieta gradual, aumento de la duración del entrenamiento y entrenamiento en lugares con altas temperaturas. La orientación para escoger un método de reducción de masa corporal proviene del entrenador, el compañero habitual de entrenamiento o atletas mayores.
Conclusiones: La pérdida rápida de masa corporal fue común entre los luchadores de artes marciales mixtas estudiados. Los métodos más utilizados fueron: restricción de la ingesta de líquidos, sesiones de sauna, dieta gradual, aumento de la carga de entrenamiento por encima de lo habitual y entrenar en lugares con temperatura elevada.

Palabras clave:
Reducción de peso. Atletas. Luchadores.

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Introduction

Mixed Martial Arts have gained attention in the sporting scene and among people in general, leading to the worldwide popularization of this sport category[1]. It should be noted that this type of fighting is considered complex, as it combines different techniques and styles of varied fights, of domain or percussion style[2].

When determining the temporal structure, to help understand the physiological aspects of the predominance of each energy system and its changes with the stimuli, the fights have in general three to five rounds, 5-minute duration and 1-minute intervals, showing that it is an intermittent sporting practice, whose energy metabolism is anaerobic with aerobic demand[2-4].

Then, pure strength, strength resistance and muscular power are important neuromuscular variables to be developed, so that the athlete is successful in the fight[5]. However, one characteristic of combat sports is the categories according to the athlete's body weight. Therefore, in order to balance the competitions, the athletes fight opponents of similar weights, and many athletes use aggressive methods to reduce their body weight and have a supposed advantage in relation to their opponent[6].

However, rapid weight loss is said to impact the athlete's physical performance during the fight[7]. The harmful physiological effects caused by sudden weight loss include: smaller blood and plasma volume and lower muscle glycogen utilization rate[8], loss of muscle mass[9], which in turn directly affects the variable of skeletal muscle strength[10], a prerogative for fighters who wish to win[7].

Regarding the effects of weight loss on strength, the results found in the literature are divergent[11]. When evaluating 20 judo athletes, Degoutte *et al.*[12] observed that, after seven days of weight loss, the fighters presented a considerable loss of grip strength of the left arm. In another study, Ratamess *et al.*[13], when analyzing 16 wrestling fighters who had weight loss 10 days before the competition, did not report any change in the variable of grip strength for these athletes.

Although weight loss is well documented for other types of fights, the literature has insufficient national studies on weight loss in MMA athletes. Therefore, this study aimed to analyze the frequency of rapid reduction of body weight in the period of 12 months before a competition and the methods used to achieve it, in a group of state and national level competitors in the city of Curitiba, Paraná, Brazil.

Material and method

This is a cross-sectional study that analyzed a sample of 25 male Mixed Martial Arts (MMA) athletes, mean age of 24.4 ± 4.1 years, training 6 times a week for 3 hours a day and participating in state and national competitions, in the City of Curitiba, Paraná. Of these athletes, 11 were from the 52-66 kg categories, 13 were from the 70-93 kg categories, and 1 athlete was from the 100 kg category.

The study included athletes who: (1) were between 18 and 40 years old; (2) were participating, at the time of data collection, in regional tournaments or more important competitions; (3) had practiced MMA for at least two years. The study excluded: (1) athletes who, at the moment of data collection, decided not to participate in the study; (2) athletes who, after data collection, chose to withdraw their informed consent, so that their data could not be used in the study.

All athletes, after being informed of the procedures to which they would be submitted, signed an informed consent term. Then the questionnaire on weight loss was applied at the training site of the athletes. This study was approved by the ethics and research committee of the Faculdade Dom Bosco, in Curitiba, Paraná, under protocol n° 1.124.722.

Questionnaire on pre-competition rapid weight loss

A questionnaire was applied to obtain information on rapid weight loss. The questionnaire has 21 closed-ended questions on weight loss in the pre-competitive period of MMA athletes, and it is an instrument that has been validated for judo[14]. It had to be adapted to MMA, since there is no validated instrument for this sport. This instrument has three sections: the first collects general data of the participants, the second collects data on weight and diet history of every participant, and the third refers to the weight loss methods used by the athletes in the last 12 months.

Several items were evaluated with this instrument, such as current MMA category; whether the fighter has moved to higher categories; the participant's weigh on his last vacation; whether he had already lost weight for the competition; the highest amount of weight he has ever lost for a competition and how many times it happened in the last 12 months; how many kilos he usually loses before a competition; how much time before a competition the athlete usually measures his weight; at what age he started to lose weight for a competition; how much weight he usually gains the week after a competition; degree of influence exerted for weight loss[14].

Statistical analysis

Data were inserted in a Microsoft Excel® spreadsheet and processed by software Bio State 5.0, year 2007, for data analysis. Shapiro-Wilk normality test was applied, which indicated a symmetrical distribution. The values of mean, standard deviation, minimum and maximum amplitudes, and standard error were calculated. In addition, absolute and relative frequencies and chi-square test were used to test different proportions with the answers provided. The statistical significance level considered in this study was $p \leq 0{,}05$.

Results

Table 1 shows the values for the anthropometric and general characteristics of all 25 athletes who comprised the sample. The total body mass of MMA fighters presented values of 79.5 ± 12.7 kg and mean height of 176.4 ± 7.5 cm. The age at which the athletes began to practice MMA presented mean values of 16.0 ± 4.8 years in a range of 7–28 years. Regarding the number of competitions the athletes participated in 2015, the mean values were 3.0 ± 1.4 competitions in a range of 1 - 6 competitions. For the number of victories in 2015, the athletes presented 2.2 ± 1.1 victories, in a range of 1 - 6 victories.

Table 1. Anthropometrical variables and general data of Mixed Martial Arts athletes.

Variables (n=25)	Mean±SD	Min.–Max.	Standard error
Age (years)	25.4±4.1	18-35	0.82
Total body weight (Kg)	79.5±12.7	60-104	2.53
Height (cm)	176.4±7.5	154-188	1.50
Age (years) when started practicing	16.0±4.8	7-28	0.95
Age (years) when started competing	19.6±3.7	12-28	0.74
Number of competitions	3.0±1.4	1-6	0.27
Number of victories	2.2±1.1	1-6	0.22

Table 2. Historical body weight in Mixed Martial Arts fighters.

Variable (n=25)	Mean±SD	Min.–Max.	Standard error
Weight during vacation (kg)	82.2±12.1	65-105	2.47
Higher weight loss (kg)	13.9±4.2	3-21	0.83
How much weight lost (kg)	9.3±3.2	1-18	0.63
How many times the athlete lost weight	3.3±1.9	1-10	0.37
How long to lose weight (days)	24.5±11.5	3-60	2.29
Weight recovered after a competition (kg)	9.5±4.4	3-20	0.88

Table 3. Techniques of rapid weight loss used by Mixed Martial Arts athletes.

Techniques (n=25)	Always n (%)	Sometimes n (%)	Almost never n (%)	I don't use anymore n (%)	Never n (%)	Total n (%)	P-value
Gradual diet*	14(56)	9(36)	-	2(8)	-	25(100)	0.0001
Reduced fluid intake*	18(72)	6(24)	1(4)	-	-	25(100)	0.0001
Exercise more than usual*	13(52)	7(28)	4(16)	-	1(4)	25(100)	0.0001
Training intentionally in heated rooms*	8(32)	13(52)	3(12)	-	1(4)	25(100)	0.0001
Sauna*	15(60)	8(32)	-	-	2(8)	25(100)	0.0001
Training with rubber/plastic suits	11(44)	11(44)	1(4)	-	2(8)	25(100)	1.00

*Chi-square test with significant level lower than 0.05 for the calculation of differences between methods

Table 2 shows the values of body weight history for the 25 athletes who comprised the sample. The weight of the fighters during the vacation period was heterogeneous 82.9 ± 12.1 kg due to the fact that they compete in different weight categories. The highest amount of weight the athletes lost was 13.9 ± 4.2 kg and they usually lose 9.3 ± 3.2 kg on average before a competition. The same athletes lost weight 3.3 ± 1.9 times in the last 12 months, and it takes approximately 24.5 ± 11.5 days. When observing the post-competition weight gain, the mean values were 9.5 ± 4.4 kg, in a range of 3 - 20 kg. The mean age at which the fighters started these weight loss cycles presented average values of 20.7 ± 2.9 years, in a range of 15 - 28 years.

Table 3 shows the most frequent weight loss techniques used by the MMA fighters who comprised the sample. Reduced fluid intake was the most representative technique (72%); other methods included sauna (60%), gradual diet to lose weight in two weeks (56%), exercise more than usual (52%), and training in hot places (32%).

Table 4 presents the percentages of individuals who influenced the MMA athletes of this study for rapid weight loss before a competition. The coach (72%) was the person who most encouraged the fighter to lose weight, followed by training colleagues and older athletes (both 64%). Relatives (72%) and the doctor (56%) were the people who showed no influence on the fighters.

Discussion

MMA is a recent sport, categorized by body weight, in which many fighters compete in categories whose weight limit is below their actual weight[15]. Regarding the anthropometric aspects, the Brazilian fighters of Curitiba, Paraná, are relatively larger and heavier than the athletes studied by Del Vecchio and Ferreira[16], of the City of Pelotas, Rio Grande do Sul, which presented 170 ± 6 cm and 76.0 ± 10.27 kg versus 176.4 ± 7.5 cm and 79.5 ± 12.7 kg obtained in this study. As the MMA fight

Table 4. People who influenced the Mixed Martial Arts fighters to lose weight.

People (n=25)	Influence						P-value
	No influence n (%)	Little influence n (%)	Unsure n (%)	Some influence n (%)	High influence n (%)	Total n (%)	
Training colleague*	1(4)	4(16)	1(4)	3(12)	16(64)	25(100)	0.0001
Fellow fighter*	-	1(4)	2(8)	6(24)	16(64)	25(100)	0.0001
Doctor*	14(56)	-	5(20)	2(8)	4(16)	25(100)	0.0001
MMA coach*	2(8)	1(4)	-	4(16)	18(72)	25(100)	0.0001
Parents*	18(72)	2(8)	-	2(8)	3(12)	25(100)	0.0001

*Chi-square test with significant level lower than 0.05 for the calculation of differences between the influencers.

is categorized by body weight, such anthropometric measures would not be a differentiation in the fight[17].

Regarding the general characteristics of the fighters in this study, they are older, begin to practice and compete later when compared to the study conducted by Mazzoccante *et al.*[18], who analyzed 18 Brazilian judo senior fighters, aged 22.7 ± 3.9 years, and who presented 13.8 ± 4.8 years of practice and 10.8 ± 2.1 years of competition. However, when correlating this study with the study conducted by Matthews and Nicholas[19], who analyzed the weight loss in 7 MMA fighters from the United Kingdom aged 24.6 ± 3.5 years, the fighters presented body weight of 69.9 ± 5.7 kg and 3.1 ± 2.2 years of competition, values that are lower than those for the Brazilian fighters, which shows this sport has been practiced for a longer time in Brazil[1].

Regarding the pre-competition rapid weight loss behavior, most athletes of combat sports reduce their body weight few days before the competition to have a competitive advantage over lighter opponents[20]. This study found that 100% of the fighters lost weight for a competition, with the athletes reporting weight loss of 1 to 18 kg in the period of three to sixty days before a competition, up to 10 times a year.

A study with taekwondo black belt fighters from the State of Rio Grande do Sul obtained 91.3% of fighters who lose weight for a competition, which is very close to the value obtained in this study. Also in the same study, the athletes reported that they lost 1 to 3 kg three to four days before a competition, up to twice a year, which are lower values when compared to this study[21]. However, in another study with jiu jitsu fighters, Ribas *et al.*[6] reported weight loss of 5.5 ± 4.3 kg about three to ninety days before a competition, up to four times a year.

When studying the pattern of Iranian Wrestlers, Kordi *et al.*[22] obtained pre-competition weight losses of 3.3 ± 1.8 kg. When evaluating Brazilian judo athletes, Fabrini *et al.*[23] observed losses of 4.5 ± 3.5 kg. Then, regardless of the type of fight, the athletes use weight reduction techniques; however, these methods may impact the performance and endanger the health of the athlete[6]. It should be noted that this cycle of weight loss and gain does not observe the Code of the World Anti-Doping Agency (WADA)[24]. In 2013, a Brazilian MMA fighter tried to lose 15 kg in seven days, but he died in a sauna the day before his fight[25].

In terms of rapid weight loss methods, the taekwondo (TKD) fighters from Rio Grande do Sul reported that they skipped meals, practiced more exercises, and trained with warm, plastic or rubber clothes[21]. In the study conducted by Matthews and Nicholas[19], 86% of MMA athletes adopted gradual diet, 86% reduced their fluid intake, 71% exercised more than usual, 43% used a sauna, 71% used a salt water hot tub, and 43% trained with plastic clothes. In the study of Ribas *et al.*[6], performing more exercises than usual was the most representative technique 86.36%, followed by gradual diet to lose weight in two weeks 63.63%, reduced fluid intake 54.54%, and training in heated areas 54.54%. When investigating judo athletes, Artioli *et al.*[26] observed dehydration in 68.4% of them, reduced energy intake in 63.1%, reduced intake of sweets and fats in 47.4%, practice of more exercises in 26.3%, and total or partial restriction of food intake at dinner as the most frequent techniques used by these athletes.

Another study conducted by Perón *et al.*[27] with Brazilian Olympic boxing athletes, the authors found that 83.3% of the athletes had a strategy to increase sweating, and the same percentage of 83.3% used fasting or semi-fasting to reach the category weight. In Muay Thai fighters, Ribas *et al.*[28] observed that diet 28% and dehydration 34% were the main rapid weight loss methods. These values agree with those obtained in this study, but the techniques used by MMA athletes seem to be more aggressive and promote higher weight loss when compared to other combat sports[19].

However, two situations should be taken into account: first, the techniques that promote dehydration and the methods of extreme diet can cause reduced aerobic and anaerobic performance. Second, the fighters who use these methods do not recognize the harmful effects or do not realize the negative impact on the body[26]. If they could understand that, they would not use sauna or wear plastic clothes during their training sessions, as these techniques have caused a fatality in MMA in 2013, according to the literature[19].

The negative impacts on the fighter's body include: reduced performance, reduced power, smaller blood and plasma volume, reduced venous return, lower efficiency of the myocardium, reduction in maximum oxygen consumption, weakening of the thermoregulatory process and increase of central temperature during rest and exercise, increased GH and reduced testosterone levels, low immune function, and temporary interruption of growth[11].

Regarding the persons who encouraged the MMA fighters to use weight loss techniques, when investigating boxing fighters, Lucena *et al.*[29] found that the coach 42% as the person who most influenced the athlete to lose weight, followed by another fighter – boxing or another type of fight 21%. When investigating jiu jitsu fighters, Ribas *et al*.[6] observed that the coach 63.6% was the person who most encouraged the fighters to lose weight, followed by training colleagues 40.9%, and older athletes 36.3%. When analyzing TKD athletes, Diniz *et al*.[23] pointed out that physicians has almost no influence on the athlete, and training colleagues had a reasonable to high influence 62.5%. The person who most influenced was the TKD instructor, in 77.7% of the cases.

Although the studies have reported that the person who most encouraged weight loss of the athletes was the coach, Juzwiak and Ancona-Lopez[30] suggest that this professional in the various types of fight many times does not have proper knowledge of weight loss strategies to conduct this process with the fighters.

Conclusions

In conclusion, this study observed that rapid weight loss is frequent among the investigated Mixed Martial Arts fighters, who tend to lose 1 to 18 kg during the competition season, and the most frequent methods were increased physical activity, gradual diet, training in heated areas, and reduced fluid intake. It also observed that the MMA coach is the person who most encourages such practice, followed by training colleagues and older athletes. However, this study presents a limitation regarding its method, as the questionnaire has been validated for judo, not for MMA. However, it has been used for different combat sports and fulfills the study objectives.

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Preventing injuries using a pre-training administered rated perceived exertion scale

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Summary

The objective of this study was to develop an injury prevention protocol based on Rated Perceived Exertion (RPE) before and after training sessions was measured using the CR-10 Borg scale. Measuring pre-training exertion allows players to inform their coach about their state before initiating any activity, which helps the coach to adjust the training load. A total of 12 players from the Spanish first-division "Hormigoneras Umacón" futsal team were followed-up during the 2013/2014 season. Data were collected for 40 weeks in 225 training sessions. The injuries sustained and pre-training RPE obtained were recorded for each player. A RPE value of "6" was considered a "warning sign" that indicated that the player might not be in optimal conditions to support the planned training load. The results reveal that the incidence of injuries was lower (p<0,05) among the players showing a lower number of warning signs. In addition, in the months with a higher training volume, warning signs were useful in reducing the number of injuries sustained by the players. In conclusion, "warning signs" indicate alterations in the physical state of players before initiating any activity, which allows the coach to modify the training load and reduce the risk of injuries.

Key words:
Futsal. Training load. Prevention of injuries. Subjective perception.

Prevención de lesiones usando la escala de percepción subjetiva del esfuerzo

Resumen

El objeto de estudio fue desarrollar un protocolo de prevención de lesiones basado en la Percepción Subjetiva de la Fatiga antes del entrenamiento. De acuerdo con autores que utilizan la Percepción Subjetiva (RPE) mediante la Escala CR-10 de Borg para evaluar la fatiga del jugador antes y después del entrenamiento, analizamos la fatiga previa considerando que esta variable permite al deportista informar al entrenador de sus sensaciones antes de iniciar la actividad, posibilitando variar las cargas. Participaron 12 jugadores del equipo "Hormigoneras Umacón" de Primera División española de fútbol sala durante la temporada 2013/2014. Se recogieron datos durante 40 semanas en 225 sesiones de entrenamiento. Se registraron las lesiones producidas y los valores de Percepción Subjetiva de la Fatiga previa de cada jugador estableciendo que un RPE de 6, denominado "señal de alerta", mostraba condiciones no óptimas para soportar las cargas planificadas. Los resultados muestran que los jugadores que menor número de señales tuvieron fueron los que mayor incidencia lesional reflejaron y viceversa (p<0,05). Además en los meses con mayor volumen de entrenamiento se consiguió que el número de lesiones no fuese mayor que el resto gracias a las señales de alerta obtenidas. Concluimos que la "señal de alerta" informa de cualquier alteración del estado del deportista antes de iniciar la actividad permitiendo modificar la carga disminuyendo el riesgo de lesión.

Palabras clave:
Fútbol sala. Carga de entrenamiento. Prevención de lesiones. Percepción subjetiva.

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Introduction

In any sport and, specifically, in team sports, one of the most common causes of athletes' poor adaptation is the lack of communication with the coach[1]. Mutual athlete-coach trust and open communication allow the coach to assess the state of the athlete and avoid athlete's exposure to high-risk situations for their physical and mental state. As a consequence it is necessary that players and athletes get involved in the control of the training loads and the data provided to the coach are crucial in designing an individualized and successful training plan[1].

One of the main qualitative methods used in the control of training is Rated Perceived Exertion (RPE)[2] defined by Borg en late 1982[3] as "an individual's rating of exercise intensity or the level of exertion experienced by the athlete".

Fóster *et al.* (2001)[4] suggestet that overall RPE allows to quantify exercise intensity and make calculations using a single number that represents the combined intensity of each of the drills included in the training session. Foster this reason, Foster stated that RPE is a simple and valid method for team sports.

Most studies on team and individual sports used overall RPE to control and assess the intensity of the effort made by the athletes[5-11]. However, other authors[12, 13] used measured RPE using the CR-10 scale[14] to gauge athlete's perceived exertion before and after the training session, and also to collect information useful to injury prevention before the training session is useful in collecting full information on the physical and mental state of the athlete in order to prevent injuries[12,13]. Player's feedback will be useful for the coach to assess the impact of previous training loads and evaluate player's state before initiating the training session. The coach compares the information provided by the player with his own observations made throughout the training process and with the planned training load values, which will allow him to adjust the training plan, as necessary. In this way, the object of the Study is to use perceived exertion prior to the training session (pre-training RPE) to detect improper adaptation to previous training loads and prevent injuries.

Material and method

Study design

An observational, longitudinal, repeated-measures, 40-week study including 225 training sessions in the 2013-2014 season.

Participants

The study included players from the first team of "Hormigoneras Umacón Zaragoza" of the Spanish First Divison Futsal League (n=12).

The methods employed were approved by the Ethics Committee of the University of Zaragoza, Spain, according to the guidelines of the Declaration of Helsinki regarding human experimentation, which was approved in 1974 and modified in 2008. Informed consent from all players was obtained. Participants were free to withdraw from the study at any time.

Characteristics of the sample

Age 27.00±5.12 years; height 1.75±0.05 m; weight 73.97±6.13 kg.

Inclusion criteria

Being a player of the first team or the youth team and attending training sessions every day.

Exclusion criteria

Not attending training sessions every day, having long-duration injuries (more than two months) and not having completed half of the season (twenty weeks).

Data Collect

We developed a method to inform the coaching team about player's risk of injury before the training session, so that they could adjust the training load at individual or collective level. For this purpose, we established a RPE threshold value that indicated to the coaching team that the state of the player was compromised. Due to the scant literature available on pre-training RPE, we used the values obtained in a pilot study carried out during the 2012-2013 season with 13 players (age 26.34±4.11 years; height 1.82±0.09 m; weight 71.77±6.86 kg.) along with those reported by Del Campo (2004)[12], which followed-up 10 basketball teams over 21 training sessions. In our pilot study, the mean RPE value obtained was 5.36±1.30, and was 4.89±1.12 for the Del Campo study. Basing on these data, we established that a player reporting a pre-training RPE value exceeding 6 –which corresponds to a feeling of "exertion" in the CR-10 scale– without any apparent reason is not in the optimal condition to bear the planned training load, which is considered a "warning signal". This value of 6 is above the means obtained in both studies, which is why we considered that it was a value from which the player could suffer a poor adaptation to the training loads and suffer risk of injury.

- Daily log of RPE as measured using the *CR-10 scale* (Figure 1) to assess player's level of exertion before initiating the training session. The physiotherapist and the players recorded player's physical and mental state. The physical trainer would distribute the individual registration form to each player before starting the training and verify that all will register correctly.
- The following variables were also measured: duration of training (in minutes), missed training time, injuries sustained and training sessions missed due to injury. Injury data were recorded according to the guidelines of the Injury Consensus Group through the Fédération Internationale de Football Association Medical Assessment and Research Centre (F-MARC)[15]. This way, we could compare the results obtained with those reported in other studies that used the same methodology[16-20]. Injury is understood as physical injury resulting from sports during a match or training regardless of having to receive medical attention or loss of training or match time[16]. The team doctor was the person in charge of recording and analyzing all the injuries produced.

Figure 1. CR-10 Scale.

CR-10 Scale	
Well-rested	1
	2
Rested	3
Little tired	4
	5
Tired	6
Pretty tired	7
Very tired	8
Exhausted	9
	10

Analysis of pre-training RPE

Since a warning sign is a subjective indicator, we analyzed them from three perspectives:

- Difference between normal and unusual exertion;
- Differences in players' response;
- Detection of warning signs.

Difference between normal and unusual exertion

Player's perception of pain, discomfort or hard exertion was essential for warning signals to be effective in preventing a potential injury. It was necessary that the player could distinguish between normal exertion during the training process and other unusual negative feelings. It is crucial that players learn to listen to their body in order to minimize potential injuries[21]. Therefore, it was very important to help players learn to distinguish between the pain and "normal" discomfort caused by regular training and the pain caused by an injury.

Thus, we achieved that players recorded perceived exertion consciously. Players adapted to this change and started to devote some time to reflect on and record their training routine. This way, players learned to know themselves better and understand the response of their body to different training modalities. The RPE scale became a sports educative instrument for players.

Differences in players' response

The experience and knowledge the coaching team had of the players were essential to identify differences in perceived exertion rates. Weinberg and Gould (1999)[22] highlighted the relevant role of the coaching team and, more specifically, the role of coaches in the incidence of injuries, since it is the coach who determines the time devoted by each player to play and rest. In this study we observed that as the season progressed coaches progressively knew the players better. This allowed them to analyze the RPE of each player differently according to their characteristics so that their response could be analyzed individually.

Detection of warning signs

Warning signs were analyzed according to the previous, planned and pursued training drills. It was not a serious problem when a player showed a warning sign during a regenerative session, since the planned drills for that session were intended to accelerate players' recovery and were beneficial to them. The preseason after the holidays involves a hard training process aimed at making players attain an optimal fitness state. Thus, some of the warning signs identified during the preseason may have been expected by the coaching team, who had foreseen and assumed the risk of injury[23].

Once the warning sign was detected, we tried to identify the cause. The rate reported by the player, their observations and the reports prepared by the physiotherapist before the training session provided the coaching team with complementary information that helped them to find the causes of the warning sign. According to the criteria established by García *et al.* (1996)[20], the most common causes of warning signs were:

- Fast increase in the training load;
- Insufficient time for recovery between sessions;
- Social and affective conflicts;
- Toxic, sexual and dietary excesses;
- Psychic disturbances;
- Illness.

When hard exertion was caused by psychological factors, the player received moral support from the coach, who listened to them sympathetically. Extreme cases such as depression and associated disorders were not reported. Such a case would have required the intervention of the medical staff. Poorly controlled, stressing psychological problems derived from family conflicts, disputes with team mates or relationship problems may increase the risk of injury[23] although they cannot cause an injury directly[15,22]. We agree with these authors that mutual player-coach trust and open communication allow the coach to evaluate the state of the player and avoid player's exposure to situations that are deleterious for their physical and mental state.

When the cause of hard exertion was physical, in most cases the reason was that the player had not assimilated the efforts made. In theses cases, the coaching team adjusted training intensity and/or volume or modified the drills included in training sessions. According to Anderson *et al*. (2003)[21] "altering or modifying training programs may be the response to reducing player's susceptibility to injuries." Piggot (2008)[23] used a similar methodology with Australian football players. Thus, once the risks of injury had been identified, the coaching team modified the training program. Piggot affirmed that if an early intervention had not been made, the incidence of injuries would probably have been higher. This statement supports our hypothesis that adjusting training plans before they are initiated is effective in preventing injuries. The measures adopted were:

- Reducing training volume and/or intensity;
- Modifying or removing specific drills and/or actions;

Table 1. Warning signs, injuries, missed sessions and injury incidence for each player.

Player	Warning signs	%Warning signs	Injuries	Missed sessions	Volume (hours)	Injury incidence
8	43	36.75%	2	2	372.17	5.37
3	22	18.81%	4	5	377.43	10.60
7	20	17.09%	2	3	383.98	5.21
10	14	12.00%	2	5	250.08	8.00
1	6	5.12%	3	14	350.30	8.56
5	4	3.41%	0	0	357.85	0.00
6	4	3.41%	1	2	385.47	2.59
2	2	1.71%	4	11	363.17	11.01
11	2	1.71%	0	0	297.18	0.00
12	2	1.71%	0	0	283.25	0.00
4	1	0.85%	4	16	357.88	11.18
9	1	0.85%	4	4	318.38	12.56
Team	117	100%	26	62	4.097.15	

- Increasing rest periods during the training session;
- Respecting the healing process of injuries before resuming training with the team;
- Introducing preventive programs to strengthen individual weaknesses.

Statistical analysis

Descriptive and inferential statistical analysis of the different variables was performed using the SPS, version 19, software package (License property of the University of Zaragoza) and Excel.

- The descriptive statistical analysis was performed using mean values and standard deviation for quantitative variables, and percentages for qualitative variables.
- We calculated the point-biserial correlation coefficient to assess the correlation between a quantitative variable (number of injuries) and a dichotomous variable (the team's pre-training RPE)(r_{bp}).

Results

We observed that the players who showed a higher number of warning signs (players 3, 7 and 8) missed few training sessions and minutes of training. However, when comparing the number of warning signs and the incidence of injuries, we found that players 4 and 9 –who only showed one warning sign– were the players who sustained the highest number of injuries (Table 1).

The following values were noticeable:

- Player 8: 43 warning signs, 2 injuries, 2 training sessions not completed.
- Player 4: 1 warning sign, 4 injuries, and 2 training sessions and 1,320 minutes missed.

In August, the volume of minutes missed was 3,990, a total of 55 warning signs were observed, three injuries were recorded and the incidence of injuries was 3.7 injuries/1,000h. From September, the training volume decreased and was maintained constant throughout the season. In September, the number of warning signs decreased to 26. Nevertheless, this figure is higher than in the other months. Conversely, the number of injuries was maintained. April was the month with the highest number of injuries: 4; while December and March were the months with the lowest number of injuries: 1 (Table 2).

Table 2. Warning signs, injuries and injury incidence of each month.

Month	Warning signs	Injuries	Volume	Injury Incidence
August	55	3	3.990	3.76
September	26	3	2.860	5.24
October	8	3	2.600	5.77
November	9	3	2.605	5.76
December	3	1	2.640	1.89
January	7	3	1.675	8.96
February	4	3	2.920	5.14
March	4	1	2.180	2.29
April	1	4	2.255	8.87
May	0	2	2.185	4.57
Total	117	26	24.665	5.57

At the end of the season, to examine if there was a correlation between pre-training RPE and the number of injuries, we used a biserial correlation formula, considering that there were no injuries in 21 weeks, and injuries were detected in 19 weeks. There were no significant differences between means (r=0.09), which shows that a higher RPE did not involve a higher number of injuries (Table 3).

Table 3. PSF previous relationship - number of injuries.

Average perceived fatigue in weeks = 0 injuries	**3.20**
Average perceived fatigue in weeks ≠ 0 injuries	3.29
Standard desviation perceived fatigue	0.50
% weeks = 0 injuries	0.52 (21 weeks)
% weeks ≠ 0 injuries	0.48 (19 weeks)
Point biserial corelation = 0.09	

Discussion

Identifying warning signs, sessions missed due to injury and the incidence of injuries during the season

We observed that the players who showed a higher number of warning signs (Table 1) missed few training sessions and minutes of training. Conversely, the players who showed a lower number of warning signs were the ones with a higher incidence of injuries. We concluded that warning signs were a useful indicator of the risk of injury, which helped the coaching team to prevent them. Players were trained in the identification of warning signs. The results obtained in this study are consistent with those of Piggot (2008)[23]. In his 15-week study of an Australian football team Piggot concluded that the low number of injuries (only five) was due to the intervention of the coaching and the medical team. These teams identified any potential risk factor for player's health before initiating the training session.

Warning signs and incidence of injuries by months

The results displayed in Table 2 show a direct relationship between training volume and the number of warning signs. However, the number and incidence of injuries were not higher as compared to the other months. This situation was especially evident in August and September, the two months with the highest training volume. This period coincided with the preseason, where the training volume was higher, since the cumulative effect of all the previous sessions over the past weeks increased the risk of injury. This conclusion is in agreement with that of Anderson *et al.* (2003)[21], who studied a III-division female basketball team of the NCAA and observed that the risk of injury was higher during the first weeks of the season and gradually decreased as the season progressed as a result of players' adaptation. Milanez *et al.* (2014)[24] followed-up a professional female football team for five weeks. They reduced the training volume by 45% during the season with respect to the preseason and concluded that this was one of the main causes of the higher number of injuries sustained in the first weeks of training. Piggott (2008)[23] stated that 40% of injuries could be due to the increase in the training load. Gabbett and Domrow (2007)[25] observed that in contact sports, there was a correlation between the likelihood of sustaining an injury and the training load, especially during the preseason. In his study on semi-professional rugby players, Gabbett (2004)[26] found that the incidence of injuries in training sessions was strongly correlated (r=0.86) with the training load.

In August, although the risk of injury was much higher –as the significant number of warning signs show–, the number of injuries was low, which confirms that warning signs helped to reduce the number of injuries during the preseason. Conversely, it is to be noticed that April was the month with the lowest number of warning signs –only one– and with the highest number of injuries.

As regards the incidence of injuries, we highlight the difference between the value obtained for April (8.87inj./1,000h) despite the fact that only one warning sign was identified, and the value for August (3.76inj./1,000h) where 55 warning signs were detected. These results demonstrate that it is crucial that players learn to identify warning signs correctly in order to inform the coaching team about their state and prevent injuries.

Correlation between pre-training RPE and the incidence of injuries

The correlation value obtained between previous RPE and the number of injuries (r=0.09) demonstrates that a higher pre-training RPE does not involve a higher number of injuries (Table 3). These results are logical, since according to Anderson *et al.* (2003)[21], as soon as a warning sign is identified, training loads are adjusted to prevent injuries. This demonstrates the efficacy of the methodology employed in our study. The results obtained indicate that the threshold value of "6" established as a warning sign is a valid indicator of risk of injury.

The measures adopted were based on two key factors for team performance: the player's state of mind and their physical condition. According to the literature available, most injury prevention plans are aimed at correcting specific aspects such as postural changes, low force work levels or imbalance between time of exposure and rest periods[27,28]. However, these authors do not take into account that these factors do not operate individually but rather in complex interaction[17]. On the other hand, according to this author, prevention plans should not be evaluated only through experimental randomized, control-group studies, but also using more rigorous, semi-experimental studies including a more representative sample such as a team of professional athletes, and be performed in more realistic environments. Using pre-training RPE and warning signs allowed us to evaluate all these factors comprehensively and prevent injuries.

Despite the results obtained, we cannot ensure that they would have been different if we had not used RPE, since it would have been necessary to compare them with a control group[28]. However, this was not feasible for a professional team, where results are of paramount importance. Nevertheless, we could compare our results with those obtained by our research team in the 2004/2005 season[16], where the RPE scale was not used but the data collection methodology, the characteristics of the study population, the sports level and training methods used were the same, which is essential for both studies to be comparable[18]. In the 2013/2014 season there was a very significant reduction in the incidence of injuries with respect to the 2004/2005 season, which was 5.27inj./1,000h and 19.72inj./1,000h, respectively. This indicated that pre-training RPE can be used as an injury prevention measure.

Conclusions

- The use of a daily log requires some training of coaching teams and players.
- Pre-training RPE allows:
  - The use of warning signs indicating alterations in the state of the player before initiating any activity.
  - The modification of the training load and subsequent reduction in the risk of injuries.
- The methodology based on the identification of warning signs obtained using pre-training RPE for the prevention of injuries allowed us to reduce the incidence of injuries with respect to the previous season, which had similar characteristics.
- This study opens new lines of research and proposes viable injury prevention measures that can be included in the planning of training loads in team sports and will improve collective and individual performance.

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Original article

Comparison of body composition and physical performance between college and professional basketball players

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Summary

Introduction: The body composition, anthropometrics and physical performance of basketball players are fundamental for their practice of the sport. The purpose of this study was to evaluate and compare body composition and physical performance among college and professional basketball players.
Method: The sample consisted of 2 groups of male basketball players (n=17) (age: 23.61±3.45): Group 1 college players (n=9; age: 22.48±3.79 years), and Group 2 professional players (n=8; age: 24.88±2.69 years). Weight, height, fat mass and muscle mass were measured. Physical performance was measured in throwing the ball (m/s), gripping strength (Kg), speed over 20 metres, jumping and reactive strength. The mean height of the college basketball players was 179.44±7.97 cm, weight 83.61±14.64 kg, body mass index (BMI) 25.94±3.95 kg/m^2, % body fat mass 16.64±7.07, % Muscle mass 47.59±4.01; the mean height of the professional players was 181.50±8.42 cm, weight 89.73±25.56 kg, BMI 26.94±5.87 kg/m^2, % body fat mass 19.26±8.20, % Muscle mass 46.26±4.55.
Results: There were no significant differences in the % of body fat and muscle mass, similar results to those found in handgrip strength, ball-throwing speed and speed in 20 meters with and without the ball (p >0.05). For physical performance only the Q index and the floor contact time (DJ-t) in drop jumps presented statistical differences (p <0.05), with better results achieved by the college players.
Conclusion: College and professional basketball players do not show significant differences in body composition and variables associated with physical performance, especially the vertical jump capacity that is lower in both groups compared to what is reported in the scientific literature.

Key words:
Basketball. Body composition. Physical condition.

Comparación de la composición corporal y rendimiento físico entre jugadores de baloncesto universitario y profesional

Resumen

Introducción: La composición corporal, antropométrica y el rendimiento físico de los jugadores de baloncesto, son fundamentales en el desempeño deportivo. El propósito de este estudio fue evaluar y comparar la composición corporal y el rendimiento físico entre jugadores de baloncesto universitario y profesional.
Método: La muestra estuvo constituida por 2 grupos de jugadores de baloncesto varones (n=17) (edad: 23,61±3,45), el grupo 1 por universitarios (n=9; Edad: 22,48± 3,79 años) y el grupo 2 por profesionales (n=8; Edad: 24,88± 2,69 años). Se evaluó peso, talla, masa grasa y muscular. El rendimiento físico se midió a través del lanzamiento de balón (m/s), fuerza prensil (Kg), velocidad en 20 metros, capacidad de salto y fuerza reactiva. El grupo de jugadores de baloncesto universitario presentó una talla promedio de 179,44 ± 7,97 cm, peso corporal 83,61 ± 14,64 kg, índice de masa corporal (IMC) 25,94 ± 3,95 Kg/m^2, % masa grasa 16,64±7,07, % masa muscular 47,59±4,01, en cambio los profesionales presentaron una talla de 181,50 ± 8,42 cm, peso corporal 89,73 ± 25,56 kg, IMC 26,94 ± 5,87 Kg/m^2, % masa grasa 19,26 ± 8,20 y % masa muscular 46,26 ± 4,55.
Resultados: En la comparación del % de grasa corporal y masa muscular no existieron diferencias significativas, resultados similares a los encontrados en fuerza prensil, velocidad de lanzamiento del balón y velocidad en 20 metros con y sin el balón (p >0,05). En relación al rendimiento físico solo el índice Q y el tiempo de contacto en Drop Jump (DJ-t) presentaron diferencias estadísticas (p <0,05), con mejores resultados en el grupo universitario.
Conclusiones: Los jugadores de baloncesto universitario y profesional no muestran diferencias significativas en la composición corporal y las variables asociadas con el rendimiento físico sobretodo la capacidad de salto vertical que es menor en ambos grupos comparado con lo reportado en la literatura científica.

Palabras clave:
Baloncesto. Composición corporal. Condición física.

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Introduction

Basketball is one of the most popular sports in the world[1]. It has been described as intermittent[2], requiring players to alternate high intensity activities like jumping and sprinting with low intensity movements like walking[3]. It has been established that anaerobic fitness is important for tactical movements (i.e. transitions between defensive/ offensive play) and technical actions like shooting, however the 40 minutes that a match lasts require an important contribution by the aerobic metabolism[4], while every position in the game makes different physical demands[5].

Speed and agility are the essential aspects of almost all defensive and offensive movements carried out by basketball players in training sessions and matches[6]. The ability to repeat high intensity sprints interspersed with short recovery periods is considered to be a critical performance factor[7], and testing the athlete's power and aerobic capacity is fundamental in the modern sport[8]. Therefore to be successful, basketball players must develop a high level of physical aptitude and muscular strength appropriate to their role in the team, and also possess optimum body composition.

Study of the anthropometric characteristics and body composition of basketball players plays an important role in the selection process, since they may have a significant impact on performance[9]. Moreover basketball is influenced by body components, which offer a good biomarker for physical capacities[10]. Recent studies have found an association between anthropometric measurements and handgrip strength in basketball players, which would facilitate certain tasks such as gripping and throwing the ball[11-13].

However little data exists comparing the physical capacity and the anthropometrics of players with different skills.

Considering the above, the aim of this study was to evaluate the body composition and physical performance of basketball players as well as determining the influence of athletic performance level (college *vs.* professional).

Material and method

The investigation was a comparative descriptive study with transversal design and a quantitative approach. The sample was intentional and non probabilistic.

Participants

The sample consisted of 2 groups of male Chilean basketball players (n=17) (age: 23.61±3.45). Group 1 (G1) (n=9; age: 22.48±3.79) consisted of college players. Group 2 (G2) (n=8; age: 24.88±2.69) consisted of professional players of the second Chilean division. Both groups participated in basketball competitions in Chile.

The inclusion criteria were: (i) players should have at least 2 years experience in college and/or professional leagues (as appropriate); (ii) aged over 18; (iii) Chilean and; (iv) with no injury of any kind at the time of the physical assessment. Athletes who had not attended training sessions during the previous week were excluded.

The research respected the conditions of the 2013 Helsinki Declaration, and each player signed an informed consent to participation in the investigation.

Procedures

The assessments were carried out in March 2016, in a wooden-floored gymnasium with the regulatory dimensions for professional basketball. The anthropometric evaluations were carried out in one session at 09.00 h, after fasting ≥8 h. They were preceded by a general warm-up lasting 12 minutes, with cardio-respiratory activation, joint mobility, skipping, and changes of rhythm, direction and speed. Body composition was determined using a Biospace Inbody 120 segmental multi-frequency system (Biospace Inc, Japan®). The players stood upright on the machine and the information was obtained through 8 electrodes placed on the feet (metatarsus-calcaneus) and hands (metacarpals of the 2nd-5th finger and phalanx of the thumb). The results produced impedance measurements in 2 different frequencies (20kHz and 100kHz), recording mean values for body water content (L), proteins (Kg), minerals (Kg), body fat mass (Kg), muscle mass (Kg), BMI , % body fat mass, % fat-free mass, % muscle mass.

To assess ball-throwing, a ball weighing 8 pounds was used, brand Assess2Perform (Ballistic Ball™), fitted internally with a wireless accelerometer. The subjects had to lean their backs against a wall with legs at a 90° angle, thus eliminating any function of the trunk. From this position they were asked to throw the ball as far and as fast as possible. The best performance in metres per second (m/s) achieved in the 3 attempts allowed was recorded.

The handgrip strength was determined using a Baseline® dynamometer (±1 kg) (Enterprises Inc. USA). The test protocol consisted of three maximum isometric contractions for 5 s, standing with the arm bent at 90°, with rest periods of 60 s; the best result was recorded. All the athletes used their dominant hand.

Speed over 20 metres was timed using an electronic timing system (Brower Timing System, Salt Lake City, UT). The participants sought maximum possible acceleration. To record the intermediate times, wireless photosensitive cut-out switches were placed every 5 metres. The speed over 20 m with a basketball was also evaluated, using the same protocol and materials described above plus a ball. The players had to achieve maximum speed over 20 metres while dribbling the ball. In each of these tests the players did a familiarisation test and then had three attempts, with the best time being recorded.

To measure jumping capacity and reactive strength a jumping platform was used (AXON JUMP 4.0, Bioengineering Sports, Argentina)[14] for the following standardised tests: Squat Jump (SJ), Countermovement Jump (CMJ), Abalakov, Drop Jump (DJ) from a height of 50 cm. In all the tests the height achieved was recorded, and in the DJ the reactive strength (Q) and the floor contact time (DJ-t) were also measured. In all the jump tests the players did several familiarisation jumps, and were then allowed three attempts with the best result being recorded.

Statistical analysis

The data are presented as mean±standard deviation. The normality of the variables was assessed by the Shapiro-Wilk test. Student's t test was used for the comparison between the two groups. All these analyses were done with the SPSS programme, version 23.0. The confidence level was 95% ($p < 0.05$).

Results

The mean height of the two groups was similar ($p > 0.05$). The mean body fat of the college players was 16.64±7.07%, whereas the professional players was 19.26±8.20% ($p > 0.05$). The % muscle mass was higher in the college players at 47.59±4.01% v/s 46.26±4.55%, however the difference was not significant (Table 1).

Table 2 shows that there is no significant difference between the groups in ball-throwing speed ($p > 0.05$). In the strength of the dominant hand the college players presented higher values 47.86±12.38 kg v/s 43.68±8.43 kg but the difference was not significant. No significant difference was found in any of the jump tests, expect that in the drop jump test the floor contact time (DJ-t) was shorter and the Q index was higher in the college group, both differences being significant ($p<0.05$).

Table 3 shows that in the assessment with photoelectric switches for speed while dribbling the ball (CB) the college players reached 5 m in 1.14±0.05 s and covered the 20 m in 3.23±0.10 s, while the professional players reached 5 m in 1.12±0.08 s and covered the 20 m in 3.23±0.15 s. In speed without the ball (SB) the college players reached 5 m in 1.14±0.05 s and covered the 20 m in 3.42±0.14 s, while the professional players reached 5 m in 1.15±0.11 s and covered the 20 m in 3.41±0.20 s. No significant differences between groups were found over any distance, with or without the ball.

Table 1. Results (mean±standard deviation) of anthropometric characteristics and body composition.

Variables	College Players	Professional Players	p-value
Age (years)	22.48±3.79	24.88±2.69	0.159
Height (cm)	179.44±7.97	181.50±8.42	0.613
Weight (Kg)	83.61±14.64	89.73±25.56	0.548
BMI (Kg/m^2)	25.94±3.95	26.94±5.87	0.686
% body fat mass	16.64±7.07	19.26±8.20	0.491
% Muscle mass	47.59±4.01	46.26±4.55	0.531

BMI: Body Mass Index

Table 2. Results (mean±standard deviation) in ball-throwing speed, gripping strength and jumps.

Variables	College players	Professional players	p-value
Ball-throwing (m/s)	5.30±0.83	5.31±1.07	0.982
Hand gripping strength (Kg)	47.86±12.38	43.68±8.43	0.442
CMJ (cm)	36.21±5.45	33.64±5.44	0.971
Abalakov (cm)	41.78±4.38	42.15±5.09	0.873
DJ (cm)	43.10±4.97	43.56±5.59	0.859
DJ-t (ms)	272.05±34.17	43.56±5.59	0.05
Q Index	2.35±0.22	1.43±0.16	0.04

BMI: Body Mass Index SJ: Squat Jump. CMJ: Countermovement Jump. DJ: Drop Jump. DJ-t: Floor Contact Time in Drop Jump.

Table 3. Results (mean±standard deviation) of speed with and without the ball.

Variables	College players	Professional players	p-value
CB 5m (s)	1.14±0.05	1.12±0.08	0.513
CB 10m (s)	1.83±0.17	1.89±0.09	0.444
CB 15m (s)	2.58±0.08	2.58±0.12	0.964
CB 20m (s)	3.23±0.10	3.23±0.15	0.955
SB 5m (s)	1.17±0.09	1.15±0.11	0.513
SB 10m (s)	1.98±0.09	1.95±0.08	0.450
SB 15m (s)	2.71±0.09	2.71±0.14	0.937
SB 20m (s)	3.42±0.14	3.41±0.20	0.849

SB; without ball. CB; with ball.

Discussion

The aim of this study was to evaluate the body composition and physical performance of basketball players as well as determining the influence of athletic performance level (college *vs* professional). No between-groups differences ($p > 0.05$) were found in weight, % fat mass, % muscle mass, ball-throwing speed, handgrip strength, speed and jumping ability. In the vertical jumps associated with the explosive and reactive strength of the lower limbs, the college team presented better results in DJ-t and the Q index.

In the present study, there were no significant differences in height and weight between the college group (179.44 cm and 83.61 Kg) and the professional players (181.50 cm and 89.73 Kg). Nikolaidis *et al*. described anthropometric characteristics and physical condition in different players grouped by age[15]. The elite group, which consisted of 31 players drawn from 3 first division teams, had a mean height of 195.7 cm weight of 95.3 Kg; they were compared with 35 players who had classified in the under-15 group (178.2 cm and 72.4 Kg) and 35 participants aged under 18 (186.1 cm and 79.3 Kg). The under-15 group presented the lowest values when compared to the professionals and the under-18s.

No significant differences were found in % fat mass and % muscle mass. The mean fat mass was 16.64% in the college group and 19.26% in the professional players. The results found are higher than those reported by Zhao *et al.* in elite Chinese players with national and international experience, who presented 14.40% fat mass[16]. U-18 players of the Greek academy presented a mean fat mass of 13.7 kg[17]; in the present study both groups presented a higher fat mass in Kg. Similarly, a study of a sample of Australian players reported 13% and 17.4% fat mass respectively in players occupying backcourt and frontcourt positions[18].

The % muscle mass was higher in the college players (47.59%) than in the professionals (46.26%), although it should be noted that both groups present lower values than those reported in premier league players in Serbia (51.26%)[19].

As for the sprint performance there are no significant differences in any of the measurements of the two groups. These results can be compared to those of previous studies, since they were obtained using very reliable equipment, validated for this type of assessment[20,21]. In particular there is a previous study which compared the speed over 20 metres of team-sport players, showing that there were no significant differences in the test: 16 basketball players presented a performance of 3.14 s while a group of 20 handball players recorded 3.13 s over the same distance; this absence of a significant difference agrees with the results of the present study[22].

However, Köklü reported significant differences in maximum acceleration races when comparing basketball players of different competitive levels; there was even an inverse relation between the speed performance and the competitive level, i.e. players at a lower competitive level may be significantly faster than those competing at the highest level[5]. Other studies by contrast have shown evidence that elite basketball players in different categories, when compared with lower level players, present an association between their high performance and their physiological, morphological and maturity profiles[23,24].Thus studies exist with both positive and negative associations for the correlation between speed performance and competitive level in basketball players.

No significant differences were found in the variables SJ and CMJ when the two groups in the present research were compared. The mean result in the SJ was 33.54 cm in the college group and 34.64 cm in the professionals. These performance results are inferior compared to a young team of 18 players who took part in the Italian national championships and had six years' experience of basketball training; their SJ result was 39.3 cm[25]. They are also lower than those reported by Callejas *et al* in elite Spanish (47 cm) and Japanese players (44.6 cm)[26].

The college group achieved a mean of 36.21 cm in the CMJ, and the professionals 36.36 cm (no statistical significance). These values are lower than reported in a study which compared an elite team with three years' experience at national or international level (56.6 cm) with a college second team (51.6 cm)[27]. Struzik *et al.* propose that the CMJ may be a good measure for determining the jumping capacity of basketball players in jump shots[28]. Although the results of the present investigation are markedly lower than those previously reported[27], the specific nature of training may in the long term alter players' performance in assessments of their strength and power-producing capacity[29].

In the assessment of vertical jumps associated with the explosive and reactive strength of the lower limbs, significant differences were found in the DJ-t, which was longer in the professionals, and the Q index, which was higher in the college group. In both cases the college group obtained better results, while there were no differences in the DJ results. To date there have been few studies which focus on these variables, however earlier research has show that in team sports they have a significant correlation with speed and maximum acceleration capacity[30], as well as degree of neuromuscular fatigue[31] and fitness for sport[32]. Díaz *et al.* reported that in an analysis of the elastic component and the technical component, there were no significant differences between Spanish players classified as professionals and those classified as college. These results differ from the findings of the present study[33].

In the application of the test to assess the action of the arms when executing a vertical jump with countermovement (Abalakov), the groups presented no statistically significant differences; the same finding is reported in the study by Massuca & Fragoso, who separated a group of team sports athletes into 2 sub-groups (successful v/s less successful) and found no significant differences[34]. The vertical jump is prevalent in various technical actions in basketball, such as shooting at the basket and defensive or offensive bouncing; it should therefore be considered an important aspect to develop in the two groups in this study.

Proprioception and motor control influence the mechanics and efficiency of the shot[35], in our study there were no significant differences in the velocity of the Ball-throwing, this may be associated with the level of performance of the players.

The limitations of the study were not to present a larger number of players by their tactical position in the game, so as to complement the study with comparisons by specific position, considering the functions of each team member. In future research it is proposed increase the sample size, incorporating a wider age range and generating a broader profile of the Chilean basketball player for use by trainers, physiologists and physical trainers of the professional and university leagues.

In conclusion, the results of this study indicate that the college and professional players don´t show significative differences in the body composition and variables associated with physical performance. Consequently, it can be inferred that the difference of competitive level between these players is determined by technical variables, as well as by tactical aspects associated to the understanding of game. The results from present investigation can be useful for basketball coach like so professionals and researchers associate to sport sciences and related fields. As a prospective, it seems important to carry out more studies that consider these variables in new contexts, as well as to develop research of this type that also incorporates the analysis of tactical aspects of the players.

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Breathing at extreme altitudes. Scientific projects "EVEREST" (Second part)

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Summary

Climbing to the highest height on Earth, the Mt. Everest (8.848 m), without supplementary oxygen equipment involves a physiological demand that is close to the maximum human tolerance. Exposures at extreme altitudes drastically conditions lung function, stores of oxygen and physical performance. This review brings interesting aspects about respiration, blood gases and aerobic exercise reported by those scientific projects that have carried out physiological measurements between 8,000 m and 8,848 m above sea level, under real or simulated altitude: the Operations "Everest I" (1946), "Everest II" (1985), "Everest III-COMEX" (1997), and the Expeditions "AMREE" (1981), "British 40th Anniversary Everest" (1993), and "Caudwell Xtrem Everest" (2007). These fascinating scientific research events, along with other outstanding biomedical expeditions performed above 5,500 m, like especially the "Silver Hut" (1960-61), "Italiana all'Everest" (1973), and "British Everest Medical" (1994), including those pioneer scientific reports made on the XIX century until the most recent research projects performed, have laid the foundations and knowledge on the human tolerance to such extreme levels of hypobaric hypoxia, where the lung, breathing and respiratory chain takes on a major role requiring fine physiological adjustments to ensure cellular oxygenation. Geophysical aspects, climatic factors and other environmental conditions that limit the biological viability and can affect the respiratory health of climbers on the upper troposphere zone at the subtropical latitude where that mountain is located are likewise reviewed and analyzed. Every year, hundreds of climbers try to reach the top of Mt. Everest, but only a few of them achieved their goal without inhaling supplemental oxygen, including some exceptionally gifted Sherpa natives, protagonist on unsuspected exploits in the highest mountain on terrestrial surface, whose summit touch the physiological limit of survival for the human being.

Key words:
Altitude. Oxygen uptake. Hypoxia. Mountaineering. Atmospheric pressure. Respiration.

Respirar en altitudes extremas. Proyectos científicos "EVEREST" (Segunda parte)

Resumen

Escalar el punto más alto de la Tierra, el Mt. Everest (8.848 m), sin equipos de oxígeno conlleva una demanda fisiológica que está próxima a la máxima capacidad de tolerancia humana. Exponerse a altitudes extremas condiciona drásticamente la función pulmonar, el nivel de oxígeno y el rendimiento físico. Esta revisión reúne interesantes aspectos respiratorios, de gases sanguíneos y ejercicio aeróbico aportados por aquellos proyectos científicos que han llevado a cabo mediciones fisiológicas entre 8.000 m y 8.848 m, en altitud real o simulada, como las Operaciones "Everest I" (1946), "Everest II" (1985) y "Everest III-COMEX" (1997), y las Expediciones "AMREE" (1981), "British 40th Anniversary Everest" (1993) y "Caudwell Xtrem Everest" (2007). Estos fascinantes eventos de investigación, junto a otros destacados proyectos biomédicos realizados a más de 5.500 m, muy especialmente las Expediciones "Silver Hut" (1960-61), "Italiana all'Everest" (1973) y "British Everest Medical" (1994), incluyendo aquellas pioneras observaciones científicas llevadas a cabo en el s.XIX hasta los más recientes proyectos de investigación realizados, han sentado las bases del conocimiento sobre la tolerancia humana ante niveles de hipoxia hipobárica extrema, donde el pulmón y la cadena respiratoria adquieren un trascendente protagonismo requiriéndose de finos ajustes fisiológicos que garanticen la oxigenación celular. Asimismo, se exponen ciertos aspectos geofísicos, factores climáticos y otros condicionantes ambientales que limitan la viabilidad biológica y pueden afectar la salud respiratoria de los alpinistas situados en las cotas superiores de la troposfera a la latitud subtropical donde se encuentra ubicada dicha montaña. Actualmente cientos de alpinistas intentan alcanzar la cumbre del Mt. Everest todos los años, pero solo algunos consiguen su objetivo sin inhalar oxígeno suplementario, entre ellos algunos excepcionalmente dotados nativos Sherpa, protagonistas de insospechadas hazañas en la montaña más elevada de la superficie terrestre, cuya cima roza el límite fisiológico de supervivencia para el ser humano.

Palabras clave:
Altitud. Consumo de oxígeno. Hipoxia. Montañismo. Presión atmosférica. Respiración.

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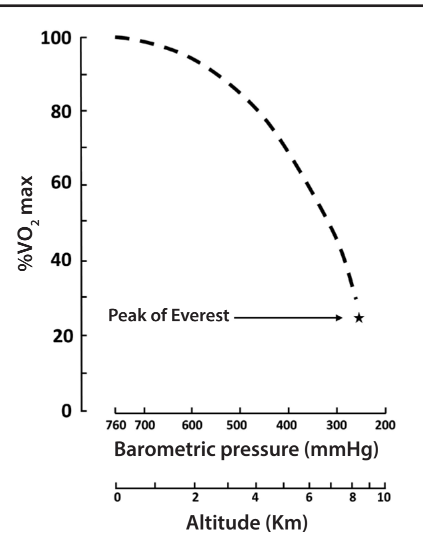
Maximum oxygen transportation and uptake faced with extreme levels of hypoxia

Physiological adaptations that occur when faced with a very low oxygen blood transportation must ensure tissue homeostasis, even during physical exercise, as this situation drastically reduces SaO_2 at extreme altitudes[31,48,63-65]. Blood samples taken at 8,400 m, descending from the peak of Everest, reveal average SaO_2 values of ~55%, with a figure below 35% detected in one of the subjects studied[36]. During maximum physical exercises, SaO_2 measurement below 50% were obtained at an altitude of 5,800 m[66], as well as at 6,300 m with FiO_2 of 14% (PiO_2 ~43 mmHg), simulating the 8,848 m of Mount Everest[48], even SaO_2 below 40% with identical PiO_2 but in a hypobaric chamber[39].

Hypoxia causes an exponential reduction in maximum aerobic power. At 7,000 m the maximum oxygen uptake (VO_2max) is reduced by ~60%[14] and at 8,848 m by 70-80% compared to sea level[39,48,67] (Figure 2). Mountaineers at an altitude that simulates the peak of this mountain have proven to develop a VO_2max ~1—1,2 L•min^{-1} in diverse studies[31,39,48,68], and some subject display slightly lower values[39,69]. Despite this, cell function must be guaranteed, via a capillary PO_2 that must be kept above 15 mmHg[35]. The physiological explanation behind this major reduction of the organic availability of oxygen is complex[67], and it seems to radiate mainly in the diffusive limitations of gases both in terms of the lungs (alveolar/capillary) and the muscles (capillary/mitochondrial), as the ventilatory and cardiac systolic functions have a lesser influence[70-72]. Two factors are key in terms of the lungs: on the one hand the effect of an interstitial oedema (initially sub-clinical) induced by the hypoxic vasoconstriction and the subsequent PHT, as mentioned before, and on the other hand the reduction of the blood transit time through the alveolar capillary bed, which is due to the increase of the cardiac output (parallel to that of the systemic circuit) and the hypoxic vasoconstriction itself, which both cause a marked increase in the flow speed of the pulmonary micro-circulation. As a result of this insufficient transit time, the haemoglobin does not reach its optimum balance when faced with very low levels of PAO_2 and the blood leaves the lung with a more reduced SaO_2 than expected. However, it is estimated that mountaineers usually ascend over 6,000 m of altitude at a physical exertion intensity of 50-75% of their VO_2max[73,74], though during the climb of the last stretch of the final pyramid of Everest, it is feasible that this ascends to 85-90% of the VO_2max[75,76].

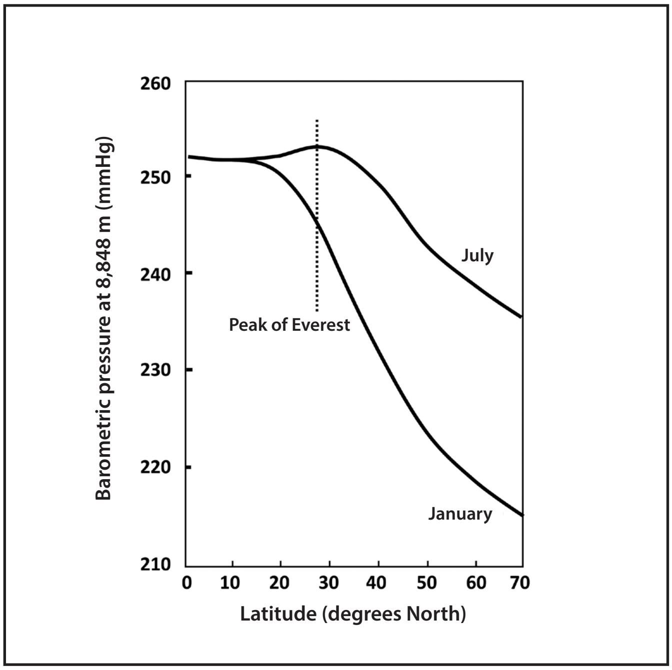
Figure 2. Percentage of maximum oxygen uptake depending on altitude and barometric altitude. Average value obtained by different authors (Based on Richalet and Herry[45]).



Assuming that 1 MET (basal metabolism) is equivalent to 3.5 mL•min^{-1}•kg^{-1}, it has been calculated that remaining at the peak of Mt. Everest without performing physical exercise requires a minimum metabolic requirement of 1.4 METs (~5 mL•min^{-1}•kg^{-1} or ~350 mL•min^{-1})[32,77]. The most important example of this being possible was the mountaineer Babu Chiri Sherpa, who in May 1999 remained on the peak of Everest for 21 hours straight without breathing supplemental oxygen. However, in order to climb to that point in summer without oxygen masks, reaching an average VO_2max ~15.3 mL•min^{-1}•kg^{-1} with a PiO_2 equivalent to 8,848 m[31,39,48] as well as the linear relationship PiO_2-VO_2max[18,32,48] and the extremely slow ascent pace performed by Messner over the last 100 m (2 m•min^{-1}; body weight plus equipment ~150 kgm•min^{-1}), it is estimated that a minimum functional reserve of 3.5 METs (VO_2max ~12.3 mL•min^{-1}•kg^{-1}) is required, at an exertion intensity of 85% of VO_2max, i.e. 3 METs (VO_2 ~10.5 mL•min^{-1}•kg^{-1})[76]. Therefore, taking into account the percentage of aerobic reduction at the peak of Everest, a VO_2max between 49 and 61 mL•min^{-1}•kg^{-1} at sea level[39,48] was proposed as a minimum metabolic requirement, climbing to ~90% of the VO_2max[75], with these values being below those previously estimated[68,78]. In fact, the first people to climb Mt. Everest that did not use supplemental oxygen revealed a VO_2max of 49—66 mL•min^{-1}•kg^{-1} at low altitude[79]. In general, Caucasian mountaineers that ascend between 4,500—8,848 m reveal average VO_2max values of ~51—61 mL•min^{-1}•kg^{-1}, (range ~43—67 mL•min^{-1}•kg^{-1}) at low altitude[75,80,81]. Above 6,000 m there is a direct correlation between the maximum altitude reached and VO_2max, as the higher the value, the more guarantees there are that the climb to extreme altitudes will be successful, and not just this; it is also shown to be a physiological safety parameter for climbing heights over 7,500 m[75]. Various genetic polymorphisms are associated with a higher aerobic performance at great altitude, among which it is worth highlighting the angiotensin-converting enzyme (allele ACE-I)[82], though scientific data is still very scarce to claim sufficient proof in this respect.

The average PB on the peak of this mountain in the times of year when it is usually climbed (May and October) is 251—253 mmHg[33]. However, the PB undergoes minor daytime variations and marked seasonal oscillations[62,83], with these variations having a significant physiological transcendence on physical performance, especially on

Figure 3. Barometric pressure variation at the altitude of Mount Everest (8,848 m) in relation to different times of year and geographical latitudes of the Northern hemisphere (Based on West *et al.*[34,62]).



VO_2max[33,34,77]. Therefore, the success rate for climbing Everest without supplementary oxygen will depend largely on the PB prevailing on the day the peak is climbed. As Figure 3 shows, the PB is at its highest during the months of July and August (~255 mmHg), therefore, the VO_2max will be higher and the mountain ascent will be more physiologically accessible; the PB is at its lowest in January and February (~243 mmHg) and this is when the greatest reductions of VO_2max occur, approximately 10—12% compared to summer levels[33,62,84]. The current record of the number of ascents to Mount Everest is held by three Sherpas: Appa, Phurba Tashi and Kami Rita, with 21 ascents each. However, the Sherpa Ang Rita — known as the "snow leopard" because of his exceptional physical performance at extreme altitudes — is the only human being to ever scale this mountain on 10 separate occasions without inhaling supplemental oxygen, and is the only person ever to have performed this ascent in this way in winter. A few years after achieving this feat, performed on 22nd December 1987, we detected that his VO_2max was 66.7 $mL•min^{-1}•kg^{-1}$ at sea level[85], a value that is significantly higher than that measured in other internationally renowned Sherpas[86]. It is known that a higher VO_2max at sea level entails a greater VO_2max at great altitude[39], a fact that enabled Ang Rita to scale the peak of Everest in winter without oxygen equipment, despite the drastic reduction of ~80% which his VO_2max presumably experienced at the altitude of 8,848 m. A VO_2max ~13.3 $mL•min^{-1}•kg^{-1}$ would align with the minimum value needed of 3.9 METs, which has been estimated to complete the ascent under these conditions[76]. On the day that Ang Rita achieved this winter climb, the PB was 247 mmHg at the same altitude and latitude of Mount Everest, i.e. 9 mmHg less than during the ascent performed by Messner on 20th August 1980, but 4 mmHg higher than the levels registered on the peak mid winter, a fact that was decisive in the success of the sporting achievement accomplished by this sherpa[84].

Despite minimum PB changes entailing very significant variations of VO_2max at such extreme altitudes, it is worth highlighting that other advantageous factors associated with the chronic hypoxic adaptation shown in people from Tibetan lineage also influence their best physical performance at such high altitudes. These natives present a lower reduction of maximum heart rate compared to levels observed in Caucasians; a larger surface of pulmonary gas exchange and alveolar-capillary diffusion capacity; minimal PHT and right ventricular overload; greater HVR (hypoxic ventilatory response) and a lack of, or lower rate of central apnoea and Cheyne-Stokes-type respiratory rhythms during sleep; less erythrocytosis; and blood viscosity with higher SaO_2 values[87-96]. These adaptive mechanisms found in Tibetans, from whom phylogenetically the Sherpas descended[97], are the best anthropologic example of adaptation to altitude, because their permanent exposure to environmental hypoxia originates from the Upper Palaeolithic Era, surpassing the Andean highlander natives by thousands of years[89,96]. More than ten genes have been described that are involved in their extraordinary adaptive response to great altitude, though a few of them specially related to blood-oxygen transportation, seem to play a particularly important role: allotype EPAS1 (HIF-2α) and allotype PHD2/EGLN1, its negative regulator[98-100]. A higher frequency of allele ACE-I has also been reported in Sherpas[101].

In general, aside from climatic aspects, three physiological mechanisms are particularly decisive in being able to climb to 8,848 m of altitude without oxygen canisters: the extreme hyperventilation generated reduces the $PACO_2$ stabilising the PAO_2, acute hypocapnic alkalosis facilitates the oxyhaemoglobin saturation in the lungs, and the PB increase, despite being minimal, significantly raises the VO_2max[48]. Despite these extraordinary adaptations, which enable survival at such high altitudes, human beings have limiting physiological factors in terms of physical performance in this environment, despite this being a contested aspect. Within this limitation, PB has a relevant role to play, as well as the energy expenditure of hyperventilation, the gas diffusion capacity through the alveolar-capillary membrane, the peripheral PO_2 and the transfer of oxygen to active muscles[67,71,102,103]. Moreover, the on-going sympathetic stimulus caused by chronic hypoxia inhibits the maximum chronotropic response, a fact that has been fundamentally linked to a progressive desensitisation of the beta-adrenergic myocardial receptors[104]. Recently, particular emphasis has been placed on the sporting success of the two mountaineering pioneers – Messner and Habeler - who reached the peak of Mount Everest in 1978 without supplemental oxygen. Whilst they did not have exceptional VO_2max at low altitude[76], they would have undergone transcendental physiological changes in the increase of their muscular capillary density, and therefore, an optimum periphery diffusion of oxygen during that historical ascent[105]. While VO_2max is drastically reduced at extreme altitudes, the anaerobic capacity of the skeletal-muscle paradoxically reveals a similar reduction when faced with chronic exposure to hypoxia[106]. This is due to the major depletion of plasma bicarbonate upon compensating the respiratory alkalosis, with minimal increments of lactate being detected when faced with intense physical exertions in altitudes over 7,500 m[31,102].

Geophysical aspects, extreme climatic and environmental factors on aerobic performance and the health of mountaineers

If we take the standard atmosphere model of PB as a reference, at the peak of Everest there would be a value of 236 mmHg, some 17 mmHg less compared to real atmospheric measurements taken in springtime, and this would cause a reduction of the PiO_2 from 43 mmHg to 39.5 mmHg[33]. Because the $logPiO_2$—VO_2max relationship is very strong (~63 mL•min^{-1}•mmHg^{-1}), this loss of just 3.5 mmHg would produce a reduction of the VO_2max of ~222 mL•min^{-1}, i.e. adding ~21% more reduction to the altitude of 8,848 m, with a viable ascent to the peak of this mountain under these conditions seeming unlikely, as calculated by West *et al.*[33]. A PB of 236 mmHg in a real atmosphere is equivalent to an approximate altitude of some ~9,350 m at the latitude where this mountain is located, in other words, half a kilometre higher than Mount Everest. Due to the contraction shown by the troposphere towards the Earth's polar regions, this PB at this time of year would correspond to Everest being geographically located on a more northerly latitude. Therefore, despite the extreme hypobaric hypoxia that prevails at the peak of Everest, given its subtropical position (28ºN), mountaineers are favoured by the equatorial dilation of the troposphere[34,62]. As shown in Figure 3, the PB at around 8,850 m during mid-winter in the Artic polar circle (66ºN) reaches ~214 mmHg[34], i.e. this mountain would have a simulated elevation of a thousand metres higher.

Bailey calculated that where Mount Everest was located, and according to the PB that prevailed there at different times of year, an altitude of ~9,970 m could be the theoretical limit where the PiO_2 would ensure a VO_2max of 3.5 METs in summer, as well as ~9,660 m of 3.9 METs in winter, and at the altitude of ~11,900 m it would equal 1 MET[76]. *A priori*, the first and second figures would exceed already proposed estimations, and the third would exceed the experiment by Angelo Mosso, who simulated an altitude of 11,650 m in a hypobaric chamber. He reached a PB of 192 mmHg, equivalent to ~10,800 m of standard altitude[107], though taking into account the slightly oxygen-enriched mixture that he inhaled during this trial (FiO_2: 29.2%)[25], it would have given him a PiO2 corresponding to ~8,850 m, similar to the peak of Everest. It is worth mentioning that during the final phase of Operation Everest I, a simulated altitude of ~15,400 m was achieved whilst acclimatised subjects breathed 100% oxygen, with these subjects revealing a certain degree of momentary tolerance to this atmosphere. Non-acclimatised flight pilots do not usually tolerate altitudes of 13,000 m, despite inhaling supplemental oxygen[38]. In fact, being exposed to altitudes over ~12,000 m, even while resting and breathing maximum concentration oxygen, the SaO_2 drops drastically, and does not guarantee the cell viability of the organism, therefore pressurised equipment is needed[108].

The Himalayan mountain range rises slowly but constantly, due to the compression between the tectonic plates of India and Eurasia. Due to this fact, it is intriguing to consider speculations regarding the amount of time that it will be possible to ascend Mount Everest without using oxygen equipment. Considering that the annual elevation rate is ~3 cm, Bailey calculated that it will not be until approximately the year 29,000 and 39,000 - in winter and summer respectively - when Everest will reach an altitude at which the PiO_2 will ensure the physiological limit that is compatible with developing a VO_2max ~3.5 METs at its peak, and the year ~104,000 when it would equal ~1 MET[76]. However, it is clear that the precise future behaviour of continental platforms is unknown, as well as the exact characteristics of the troposphere in thousands of year's time, due to the effect of global warming or climate changes that modify the composition of atmospheric gases. While there are numerous speculations regarding the effect of global warming on the Himalayan region, certain measurements reveal that since 1948 the troposphere has been increasing its pressure on average by ~1,8 mmHg each decade, therefore, in keeping with this trend it could be possible that progressively developing a greater VO_2max on the peak of Mount Everest may be feasible, and climbing it may become more accessible aerobically in the future. However, within this environmental context, mountaineers will have to face new technical difficulties caused by the thermal increase, such as the risk of potential avalanches and rock-climbing sections caused by the disappearance of the ice on a large part of the routes[109].

The wind that strikes the edges and walls of this great geological formation could reduce the PB due to the Venturi effect, though calculations using the Bernoulli equation reveal that local PB changes would be minimal, below 1 mmHg, for which it would have a very minor physiological impact on the VO_2max[34]. This is particularly true before and after monsoons, when there is greater meteorological stability, and winds do not usually exceed 55 km•h^{-1}, a time that is chosen to attempt the ascent with the highest possible chance of success and safety[83]. However, during these brief anti-cyclonical windows of opportunity occurring in May and October, sudden gales occur, and the high layer of the troposphere can even be fed with great masses of subtropical water vapour which interact with the stratospheric jet stream current, causing convective vortexes with strong hurricane-force storms over Everest[110]. During winter, this jet-stream current moves towards southern Central Asia, completely covering Himalayan mountain range, and cyclonic winds with speeds reaching around 300 km•h^{-1} can hit the peaks of over 8,000 m of altitude. Days with strong gales are avoided by mountaineers given the high risk of accidents that these conditions entail, as well as the chill factor associated with low temperatures that the wind has on the body. On the peak of Mount Everest, temperature of minus 60ºC can be reached when masses of Siberian polar air penetrate the Tibetan plateau, though during the climatic windows in May and October, the average wind temperature is usually around ~ minus 26˚C[111], though the chill factor can give values below minus 50˚C[83], with warmer temperatures more usual throughout the day during these periods[34]. The atmospheric factors largely explain why only 25% of mountaineers that attempt to climb Everest in spring or summer actually manage to reach the peak, and why this success rate drops to just 4% of those that attempt it in winter[112], though the first percentage seems to have risen in recent years.

Breathing low-temperature air increases the risk of suffering from a pulmonary pathology, and at extreme altitudes upper respiratory tract problems are frequent, which improve upon descent[113,114]. The low relative humidity in the high layers of the troposphere cause a large loss of water vapour through the lungs, exceeding 60—80 mL•h^{-1} during moderate-intensity physical exercise[62]. The nasal inspiratory flow increases with altitude, but at 8,000 m it is lower than expected in accordance with the prevailing low air density[115]. Episodes of bronchospasm are infrequent at high altitude, especially among well-controlled asthma suffers[116], as atmospheric pollution reduces in high mountain regions and this environment has been shown to be clearly beneficial to these pacients[117]; even exposures at extreme altitude appear to be safe in cases of moderate-intensity asthma sufferers[118]. With the exception of bronchospasm episodes that are not directly related to pollen allergens (breathing cold, dry air, vigorous physical activity), it has not been proven that bronchial spasm episodes are favoured, specifically, by the hypoxia and hypocapnia of altitude, given that the oxygen supplement does not seem to increase ventilatory flows at extreme altitudes[59]. The stratospheric ozone, whose existence has been detected at a level very close to Mount Everest, represents a potential added health-risk for mountaineers, as it causes the inflammation of the mucous membranes of the airways, bronchoconstriction, coughing and/or dyspnoea, deteriorating pulmonary function[62,119,120]. The upper respiratory tracts re-heat low-temperature air inhaled quickly, for which it reaches optimum thermal graduation at the level of the alveolar, clearly not constituting a reason to support high-altitude pulmonary oedema HAPE, secondary non-cardiogenic oedema to PHT, already relatively frequent at heights below 5,000 m[72,121-125]. It is difficult to estimate the HAPE rate at altitudes over 8,000 m, but the presence of interstitial plasma cause by subclinical HAPE would be frequent, which would alter the gas exchange[69,126], as observed in athletes at much lower altitude[127-131]. Even with no oedema, the low gradient alveolar-capillary oxygen pressure (because of the reduction of PB and excessive pulmonary blood transit) increases the degree of hypoxemia especially during physical activity[131], an exercise capacity that is already limited by the PHT[132].

Aside from the hypoxia caused by a HAPE, just breathing in an oxygen-deprived atmosphere causes deterioration to the central nervous system and the development of cerebral oedemas that could cause an accident on the mountain, or cause the mountaineer to lose consciousness[10]. Neuropsychological alterations are frequent[133,134], some are transitory and are associated with a focal reduction of cerebral blood flow caused by extreme hypocapnia[135]. Paradoxically, subjects with a higher HVR - therefore a greater PaO_2 and better adaption to hypoxia – suffer from more residual cognitive dysfunctions, possibly due to the reduction of blood flow caused by very low levels of $PaCO_2$[136]. The possibility of permanent encephalic hypoxic damage was suggested in Himalayan mountaineers[137] and although the exact mechanism of brain damage in this demographic has not been well clarified[138-141], the fact that Sherpas appear to have certain neurological protection against extreme altitudes is very suggestive[142].

Conclusions

Since the pioneering scientific experiments from the 19th century, which used hypobaric chambers to attempt to establish if a "hypoxia"[26] or "acapnia"[25] respiratory situation was the result of certain limitations and pathological disorders that appear in high mountain areas, numerous later studies, especially the fascinating projects carried out in the Mount Everest region or simulating its altitude have established the basic concepts on the respiratory response mechanisms in humans exposed to this extreme environmental setting. Pulmonary adaptation to severe levels of hypoxia, especially during physical exercise, is astonishing, yet the diffusion capacity of gas exchange at an alveolar-capillary level seems to play a determining role as a limiting factor of oxygen availability in the body. However, evidence reveals that pulmonary respiration can guarantee cell respiration, even faced with such levels of reduced partial pressure of oxygen, and on the peak of Mount Everest these levels are very close to the limit of human physiological tolerance. The subtropical geographic latitude upon which the Himalayas are found enable an ascent up to the highest point without inhaling supplemental oxygen, due to the geoid effect or equatorial convexity of the troposphere, a fact labelled "cosmic coincidence"[143] by the prestigious physiologist John West. Even so, three physiological mechanisms will be particularly decisive in managing to reach the 8,848 m of altitude in this way: extreme hyperventilation, acute respiratory alkalosis, and favourable atmospheric pressure; factors that facilitate and increase the saturation, transportation and oxygen uptake during the day that the climb is attempted.

Some respiratory pathologies, such as asthma episodes or HAPE, must always be contemplated when at great altitudes, as they may evolve seriously, and they may be difficult to control in such a hostile environment, potentially endangering lives. Breathing hypoxic air frequently entails the appearance of neuropsychological disorders, with these being one of the main causes behind the accident rate at extreme altitudes. Despite the risks involved in attempting to scale the colossal pyramid of rock and ice that forms Mount Everest, its inhospitable peak is a coveted goal year after year for hundreds of mountaineers from all over the world. Achieving such a major challenge without any supplementary breathing equipment is only possible for very few people, even for natives with thousands of years of adaptation to great altitudes, such as those from the Tibetan lineage, with their exceptional genetics and physiological capacity to endure environmental hypoxia.

The testimony of the mythical mountaineer Reinhold Messner is highly compelling, as he describes the sensations he experienced when climbing the final metres of Mount Everest, becoming, alongside Peter Habeler, the first to climb it without using oxygen equipment: "*…I have the feeling I am about to burst apart. As we get higher, it becomes necessary to lie down to recover our breath… Breathing becomes such a strenuous business that we scarcely have strength felt to keep moving forward… I am nothing more than a single, narrow, gasping lung, floating over the mists and the summits*"[144]. Undoubtedly, overcoming the onslaught of the forces of nature inherent to the highest point on Earth represents a first-degree physiological challenge, a dangerous sporting feat that takes place on a colossal backdrop far from the public applause.

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Frostbite: management update

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Summary

The popularity of winter sports is leading to an increase in the number of subjects exposed to environmental pathologies such as frostbite. This is the reason why the patient's profile is changing from the classical descriptions of adults with pre-existing conditions, basically those with cognitive impairment that prevented them from a proper protection against cold or as an occupational illness in working routines related with low temperature exposures. Nowadays these disorders occur mainly in healthy athletic young patients who expose themselves voluntarily to the cold environment, both for professional or amateur aims. Frostbite can occur as a single pathology or can take part in a more complex clinical picture that includes more serious conditions, as hypothermia or trauma. In addition to this fact, it is not uncommon that frostbite appears in exhausted and dehydrated subjects. The likelihood of such injuries taking place in remote areas further complicates its management, primarily because of the delay in diagnosis and definitive treatment. Sequelae after frostbite are common and potentially limiting for the posterior sports career. In recent years, efforts have been made to establish algorithms intended for rescue and expedition doctors to manage mountain medical care based on scientific evidence. Current recommendations include prompt identification and immediate medical care, followed by early hospital treatment if necessary and specific long-term rehabilitation programmes. This review attempts to describe current knowledge of the physiopathology and the clinical aspects of frostbite, in addition to new management perspectives, from in-situ emergency care through to hospital treatment.

Key words:
Frostbite. Rewarming. Iloprost. Amputation.

Actualización en el manejo de las congelaciones

Resumen

La popularidad de los deportes de montaña conlleva que cada vez haya más individuos expuestos a patologías ambientales como son las congelaciones. De esta forma, el perfil de los pacientes está variando respecto a las descripciones clásicas, donde se consideraban lesiones propias del adulto con patología de base, principalmente alteraciones cognitivas que le impedían protegerse adecuadamente del frío, o bien como una enfermedad laboral en profesiones relacionadas con la exposición a las bajas temperaturas. Actualmente esta patología se presenta más frecuentemente en jóvenes sanos y deportistas que se exponen voluntariamente al ambiente frío para la práctica deportiva. Las congelaciones pueden presentarse como una patología aislada o formando parte de un cuadro clínico más complejo, que puede incluir la hipotermia o patología traumática. Añadido a este hecho, es frecuente que se presenten en individuos debilitados por la fatiga y la desnutrición. La posibilidad de que esta patología tenga lugar en entornos remotos añade complejidad a su manejo y empeora el pronóstico debido al retraso del tratamiento definitivo. Las secuelas tras las congelaciones son frecuentes y potencialmente limitantes para la práctica deportiva posterior. En los últimos años se han hecho esfuerzos para basar los algoritmos de actuación de las patologías de montaña en la evidencia científica, destinados tanto al público deportivo como al personal sanitario. En síntesis, estos versan en la identificación y tratamiento inicial tempranos seguidos de tratamientos hospitalarios administrados de forma precoz en caso de ser necesarios y programas de rehabilitación específicos y prolongados. La presente revisión trata de describir las recomendaciones actuales, desde la identificación y clasificación de las congelaciones hasta los nuevos avances en el manejo sobre el terreno, médico inicial y hospitalario de las mismas.

Palabras clave:
Congelación. Recalentamiento. Iloprost. Amputación.

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Definition and background

Frostbite is the most common local injury due to cold and normally occurs when part of the body is exposed to temperatures below the freezing point of intact skin[1], which is estimated to be around -0.55ºC[2], without proper protection and for a sufficiently long period of time[3]. Frostbite has typically been described as an occupational injury (e.g. due to military, mining or industrial tasks), and as characteristic of subjects with permanent or transitory cognitive impairment that prevents them from protecting themselves against the cold[4]. In the last years, the rate among young, healthy adults has increased due to the popularity of winter sports such as skiing, mountaineering, ice climbing and technical climbing/alpinism, at both professional and amateur levels. The incidence among winter mountaineers appears to be very high, as much as 37% in the only study published[5]. Moreover, given that the subjects most frequently affected are aged between 30 and 49 years[6] and usually are physically active, frostbite leads to a substantial interruption in their normal activity. It is worth considering that in most cases it leads to long-term sequelae, particularly if subject's daily activities require exposure to low temperatures to ensure they are carried out safely, or if their job involves constant environmental low temperatures (e.g. ski patrollers, mountain guides, avalanche forecasters and workers involved in cold-chain maintenance).

Frostbite is among the most common consultation causes at Mount Everest medical post (27.5% of traumatic injuries) and Denali medical post (18.1% of total injuries)[7], although these data probably underestimate the actual number of cases, since the number of mild injuries not requiring medical attention is unknown. It is also worth noting that it is the most frequent reason for evacuation from Everest Base Camp[8] and the leading cause of injury at altitude in the Karakoram mountain range[9]. Around 80 cases of frostbite are registered per year at Chamonix Hospital, two thirds of which are diagnosed as superficial.

Risk factors

Since human beings have limited physiological strategies to protect themselves against the cold, any situation that compromises the body's protection capacity in the general population (alcohol abuse, mental illness, very young and very old age, etc.) is considered a risk factor for frostbite. Other intrinsic characteristics of individuals, such as pathologies that affect the vascular bed, neuropathies, Raynaud's syndrome, smoking, genetic predisposition (DD genotype for the angiotensin-converting enzyme) and previous history of frostbite, are predisposing factors that are widely described in the literature[1,4,10]. Other factors include preventable actions such as wearing external body piercing jewellery and constrictive elements (rings, snowboard bindings, elastic clamps, etc.)[11].

It has been suggested that a lack of appropriate clothing and equipment among those who practice sports in cold and high-altitude conditions and the absence of a competent guide can lead to this kind of injury[5], but more investigation is required to confirm this assumption. Any adverse event involving immobilization in a cold environment, including spinal injuries and fractures of large bones, increases the risk of frostbite due to the increased exposure time and the possibility of vascular impairment. With respect to environmental factors, the absolute temperature reached and exposure time are relevant, especially the latter, given that the severity of frostbite is related to the length of time the tissue has been frozen[1,6]. Incidence increases at altitude, particularly from 5100 meters above sea level, due to local factors such as haemoconcentration, a rise in vascular permeability and dehydration, and potential cognitive impairment secondary to hypoxia that can delay or limit self-protection reflexes[9].

Location of injury

Distal areas are the most unprotected from the cold and the most exposed; in addition, the high surface-area-to-volume ratio of fingers makes retaining body heat very difficult, so hands and toes account for up to 90% of frostbite injuries. With respect to alpine climbing, when the terrain verticality is such that crampons and ice axes are required, heat loss by conduction from the distal parts of the extremities is accelerated by contact with snow and ice, which is associated with repetitive trauma during the ascent. Nose, ears and lips cool down more slowly than the extremities[12], but may be affected if the area is not properly protected; other areas may be affected due to exposure in specific situations (e.g. the perineum in subjects sitting on metal surfaces, the penis in Nordic skiers and the knees in prolonged resuscitation manoeuvers)[5,10].

Physiopathology of frostbite

The pathogenesis of frostbite is based on local ischemia, cellular injury and destruction caused by ice crystal formation, and damage resulting from reperfusion after rewarming.

The skin's initial vasomotor response to cold is vasoconstriction, which preserves the core temperature against cutaneous heat loss. The intensity of this phenomenon depends on the severity of the cold and the individual's intrinsic vasomotor response. Secondary ischemia resulting from this process and neuronal cooling cause the initial clinical alterations in sensitivity. If exposure continues, secondary vasodilatation takes place due to the reduction in smooth muscle sensitivity to adrenergic stimuli in five to 10 minute cycles[13]. This process occurs to provide a certain amount of local protection against cold stress. The extent of this phenomenon varies between individuals and increases with exposure, and it has therefore been suggested that there is some grade of acclimatisation to cold[11]. In the context of extremely low temperatures, freeze/thaw cycles result in a thrombotic stage, which causes a progressive local ischemia. This involves cellular death and endothelial destruction, which, in turn, activate a pro-inflammatory response that nourishes the oedema, platelet aggregation and thrombosis cycle[1,4]. If the extremity continues to cool down, arteriovenous shunts may open and generate a non-irrigated distal area that protects the central compartment from further temperature loss, thus sacrificing peripheral zones that are not essential for survival[14].

On the other hand, if the skin continues to cool down, extracellular crystals cause extracellular oncotic pressure to increase, which can lead to dehydration, altered electrolytic balance, lysis and cellular death; if the reduction in temperature occurs rapidly, intracellular crystals may appear. These may expand and generate mechanical cell destruction by disrupting the organisation of the cellular membrane and intracellular organelles[1].

During the rewarming process, inflammatory changes start taking place, with the appearance of oedema, vasodilation and vascular stasis preceding platelet aggregation and thrombosis, whose clinical manifestations are blisters and severe pain. Prostaglandins and thromboxane appear to play an important role in this process, and these molecules are emerging pharmacological targets for frostbite. After this process, and depending on the grade of the secondary microvascular impairment this sequence can result in two different situations: recovery with blood clot dissolution, resulting in viable tissue, or vascular collapse that results in cellular necrosis and the appearance of dry gangrene[15]. At this point, tissue damage is irreversible.

The consequences of refreezing a previously rewarmed area are devastating because of the massive cellular destruction caused by the formation of crystals in previously damaged tissue[14,16].

Clinical manifestations

In the early stages, alarm symptoms are frequent and often feel unpleasant: a cold sensation and hyperaesthesia or hypoaesthesia are common, though not always present. The affected area becomes numb until frostbite is established, at which point there is a total loss of sensitivity and anaesthesia.

Clinical examination at this point shows a waxy tissue that is yellowish-white or spotted, and differentiating mild from severe injuries is complex.

The rewarming process is painful in most cases and may even require the use of opioid analgesics to control the pain. The tissue at this point is hyperaemic and, depending on the severity, blisters will appear within six to 24 hours; distally located, serum-filled blisters suggest a superficial injury, while proximal, haematic blisters may indicate a deeper injury[1,14]. Blisters can persist for seven to 10 days if not drained. The appearance of any sensation (e.g. paraesthesia, pain or a stinging sensation), oedema and the capacity of skin to warp under local pressure are associated with a better outcome[11], although they do not change its clinical management. Severe frostbite can lead to local infection and systemic involvement. Black eschars are a sign of gangrene in deep tissue.

Classification

There are various proposals for classifying frostbite based on different criteria, including depth of injury, topography and clinical outcome. Given the wide spectrum of injury severity, from reversible changes after rewarming to cellular destruction, it is possible to establish a simple retrospective classification in superficial or deep frostbite based on the preservation or loss of damaged tissue after recovery[16], normally between three weeks and two months after injury. The Wilderness Medical Society guidelines suggest this same classification, but prospectively, after rewarming, based on the probability of tissue loss (Table 1).

Cauchy *et al*. (2001) proposed a predictive scale based on three aspects: topographic extension after first rewarming and, after 48 hours, the presence and aspect of blisters and radiotracer uptake in a bone scan (Table 2).

Table 1. Criteria for Classification of superficial or deep frostbite.

Superficial frostbite	No or minimal anticipated tissue loss, corresponding to 1st- and 2nd-degree injury
Deep frostbite	Deeper injury and anticipated tissue loss, corresponding to 3rd- and 4th-degree injury

According to McIntosh SE, *et al.*, Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Frostbite, *Wilderness Environ Medicine.* 2014;25:4.

Table 2. Grading score for severity of frostbite injury.

	Grade I	Grade II	Grade III	Grade IV
Extent of initial lesion at day 0 after rapid rewarming	Absence of initial lesion	Initial lesion on distal phalanx	Initial lesion on intermediary (and) proximal phalanx	Initial lesion on carpal/tarsal
Bone scanning at day 2	Useless	Hypofixation of radiotracer uptake area	Absence of radiotracer	Absence of radiotracer uptake area on the carpal/tarsal
Blisters at day 2	Absence of blisters	Clear blisters	Haemorrhagic blisters on the digit	Haemorrhagic blisters over carpal/tarsal
Prognosis at day 2	No amputation	Tissue amputation	Bone amputation of the digit	Bone amputation of the limb +/- systemic involvement+/- sepsis
Sequelae	No sequelae	Fingernail sequelae	Functional sequelae	Functional sequelae

According to Cauchy E, *et al.*, Retrospective study of 70 cases of severe frostbite lesions. A proposed new classification scheme, *Wilderness Environ Med.* 2001;12:248.

This classification, which makes early prognosis possible, was designed in the context of injuries in the French Alps, where there is an effective rescue system that facilitates access to hospitals with the capacity to carry out complex radiological examinations within a short period. In more remote environments, immediate specialised medical attention is not possible within 48 hours of injury, so the outcome is estimated based on clinical examination alone, and the amount of tissue loss is highly unpredictable.

In-situ treatment of frostbite

Emergency treatment should be initiated as soon as frostbite is suspected. General recommendations from the International Commission for Alpine Rescue (CISA-IKAR) and the Medical Commission of the International Climbing and Mountaineering Federation (UIAA MedCom) for immediate treatment must be adapted to each particular situation[17]:

- Move out of the wind.
- Consider turning back.
- Drink fluids (warm if possible).
- Remove boots, but consider that there may be problems replacing them if swelling occurs.
- If wet, replace socks and gloves with dry ones.
- Warm by placing foot/hand in companion's armpit/groin for 10 minutes only.
- Replace boots.
- Give one aspirin or ibuprofen to improve circulation (if available and not contraindicated)
- Do not rub the affected part, since this may cause tissue damage.
- Do not apply direct heat.

If sensation in the affected area returns, it is worth acting on the assumption that previous prevention strategies failed and that continuing to expose the affected body parts under the same conditions is dangerous. If this does not happen, medical treatment may be needed and rewarming in a warm shelter or protected area is recommended.

Treatment of frostbite in base camp, hut or protected area

In the event that transferring the patient to a healthcare centre is difficult or will take too long (over two hours[18]), rewarming must be started in situ, as long as there is no possibility of refreezing and the environment allows for this procedure to be carried out safely[19]. Although walking with established frostbite in the foot is not recommended, self-evacuation in remote areas may be necessary, and the priority is to reach a safe location protected from the cold, rather than remaining immobile in a hostile environment. If an assisted rescue is possible, the extremity should be protected and immobilised with a non-compression bandage. The objective is to reach a safe place, where rapid rewarming can be initiated, considering that the use of heat sources during the transfer should be avoided. Incidentally, frostbite can rewarm spontaneously during attempts to keep the victim warm during transport; in this case, it is not recommended that slow rewarming is actively avoided, but it is imperative to ensure that refreezing does not occur, since this would reduce the possibility of viable tissue[1].

In general, frostbite, as a local injury, must be treated after life-threatening conditions and systemic disorders such as hypothermia and trauma.

Frostbite rewarming

Rewarming must be started as soon as possible and carried out in a water bath (ideally with a diluted antibacterial agent) at a generally accepted temperature of 37ºC-39ºC[20]. Considering that the benefits of faster rewarming are not clear, higher temperatures should be avoided, since they cause more pain and may produce associated burn wounds[19]. Conversely, slow rewarming with lower water temperatures can induce ice crystal fusion, and thus create larger structures that are more damaging to tissue.

Reperfusion criteria are recovery of sensation, normal or red/purple coloration at the distal part of the extremity and pliability of the affected tissue, which occur after 30 minutes to 1 hour of hydrotherapy[11,17]. Active movements inside the heating vessel are beneficial during rewarming[2]. Patients must be informed of the possibility of pain intensification and macroscopic changes of the injury during this process. Early treatment is essential for bone reperfusion and posterior viability[21] and the absence of recovery of sensation after rapid rewarming is a predictive factor for poor prognosis[2].

Water baths should be continued twice a day. The affected area should be kept clean and dry, and the extremity should be elevated above heart level to prevent oedema and venous stasis[4]. Massage and rubbing are not recommended, as mechanical stress on the injured area can cause further damage.

Injuries may present different grades of severity in the same limb, so keeping graphic records can be useful for the clinical monitoring of the evolution of injuries. It can be assumed that if there is loss of tissue, it will be more distal than the damage initially observed[22].

Basic treatment *in situ*

The use of NSAIDs is justified in order to reduce the oedema that can compromise blood flow and local circulation[23]. Acetylsalicylic acid irreversibly inhibits thromboxane-A2 synthesis in platelets, so many authors recommend its use[15,17], although others prefer the administration of ibuprofen[1]. There are no studies that demonstrate the superiority of one treatment over the other.

Oral vasodilators have been recommended on a theoretical basis and because of the low risk associated with their use. The capacity of pentoxifylline to increase erythrocyte deformability may improve blood flow in the damaged area if prescribed as an adjunctive therapy two to six weeks after injury[24]. Buflomedil is an alpha-adrenergic receptor inhibitor with good results in isolated cases that have not been reproducible in subsequent studies[25]. There is currently no scientific evidence to recommend the use of either medication[14,15].

Antibiotic coverage should be reserved in cases of associated cellulitis or potentially contaminated injuries, or where there are septic or traumatic concomitant pathologies that require it, since frostbite itself is not an infectious disease and antibiotic prophylaxis does not prevent secondary infections.

In-situ treatment with heparin has not demonstrated efficacy in modifying the clinical course of frostbite, but it might be recommended to prevent deep vein thrombosis if prolonged immobilization of the patient is needed in the case of frostbite in the lower limbs.

There is consensus in favour of using needles to drain clear blisters if movement is restricted and for conservative management of haemorrhagic blisters, since there is assumed to be deep structural damage underlying them[1,2]. In any case, blisters drain spontaneously within a few days. After treatment of the wound, the area should be cleaned, dried, covered with a topical aloe vera gel[26] and protected with a non-compression bandage that allows oedema to form without restricting blood flow. Dressings should be changed at least every six hours[11], although this depends on the availability of supplies and the specific conditions prior to evacuation.

Frostbite usually occurs in patients who are debilitated by fatigue, dehydration and undernourishment, all of which limit the body's capacity to produce heat[12]. During treatment, it is important to maintain acceptable levels of blood volume, orally if the patient is alert and intravenously if not, especially if clinical signs of dehydration are present, in which case small saline boluses are recommended[14]. In the presence of hypothermia, secondary to the suppression of vasopressin, larger volumes may be necessary, ideally warmed before infusion[2]. Rest and nutrition are essential for recovery, especially for patients in remote locations who face long return journeys.

The use of hyperbaric chambers at high altitude (>3500 m) has been proposed to prevent secondary intense vasoconstriction due to hypoxia and improve the benefits of in-situ treatment and rewarming[27].

Supplementary oxygen is recommended above altitudes of 4500 m[11] or if arterial oxygen saturation is lower than 90%, since tissue recovery depends to a great extent on sufficient tissue oxygenation[14].

Low-molecular weight dextran reduces blood viscosity and prevents microthrombi formation and could be a good therapeutic tool in the future considering their low anaphylactic risk and for those patients who are not good candidates for iloprost or thrombolytic therapy.

Advanced medical treatment in the field

Recent publications of isolated cases suggest that emerging therapies reserved for hospital treatment, such as iloprost and rt-PA (human recombinant tissue plasminogen activator), could be used in the field in the future for severe frostbite through resource-limited treatment strategies[22], although there are no randomised trials that justify this procedure at present. It would be particularly useful to develop optimal in-situ medical care, particularly for patients with severe frostbite who are not close to a hospital, and since the therapeutic window of these drugs is the first 12-48 hours.

Need for evacuation

If frostbite is considered the only reason to assess the possibility of evacuation, mild frostbite (grade I) does not justify ending the activity, but prevention strategies should be improved and the potential risk for refreezing assessed. Grade II frostbite does not require urgent evacuation, but the need for medical care on the field requires the activity to be discontinued for treatment and the regular application of dressings. Severe frostbite (grades III/IV) is a medical emergency in which a delay in treatment worsens prognosis, increases the risk of amputation and risks further systemic involvement.

Hospital management

The anamnesis of a patient admitted to hospital with frostbite should include the time the injury occurred (although this can be difficult to define), the moment in which first rewarming took place, and the type and frequency of any medical treatment received.

Complementary examinations are not required as routine in mild frostbite. For severe frostbite with a risk of tissue loss, angiography can show residual vascular occlusions after rewarming, thus allowing local thrombolytic treatment to be carried out and its effectiveness monitored[28]. The tendency to use Doppler ultrasounds to evaluate blood flow is becoming more popular these days, with angiography being reserved for when vascular interventions are required.

Scintigraphy with Tc99 can predict surgical indication and the extent of tissue loss after 48 hours of injury in 84% of cases[29]. While the application of this technique makes it possible for the patient to find out the extent of their injury and their prognosis at an early stage, waiting for the natural demarcation of necrosis is still recommended before surgery is carried out.

Nuclear magnetic resonance makes it possible to view soft tissues, vessels and ischemic areas clearly and noninvasively[29], although there is little experience of its use in frostbite.

Patients with severe frostbite who are attended within the first 12-24 hours in a hospital with intensive-monitoring capacity are candidates for thrombolytic treatment with rt-PA, either intravenous or intra-arterial with catheter guidance in the absence of contraindications. The aim is to restore arterial flow by eliminating thrombotic residues when distal tissues are still viable, and thus significantly reduce the number of amputations[30,31]. Although there are published dosage recommendations[2], no comparative studies have been made to strongly support an specific infusion titration. In addition to the possibility of bleeding, the most relevant secondary effect is the appearance of post-reperfusion oedema that can lead to compartment syndrome by raising interstitial pressure[10].

Infusion of vasodilators prior to rt-PA reverts the vasospasm associated with frostbite without any aditional adverse effects[32,33]. An open-label study showed that coadministration of heparin and rt-PA, both in intravenous or intra-artery delivery, appears to be a safe and effective practice for reducing vascular microthrombi formation[34]. Treatment with rt-PA should end when blood flow is restored in the distal vessels (observed with angiography) or after 48 or 72 hours in the absence of recovery[31,33]. Those patients at risk of tissue loss with a complete angiographic response have a very good prognosis[35].

Given the good results of this intervention in several case reports and published studies, it seems that patients with severe frostbite should be rapidly evacuated to hospital in order to take advantage of the therapeutic window, although there is a shortage of randomised trials to support these measures[28,34,36].

Table 3. Comparison between different thrombolytic management regimes.

Reference	Cases (n)	Grade of injury	Initial treatment	Type of administration	Drug	Dosage	Study type	Amputation rate
Wexler *et al.* 2017[41]	6	No data	Rapid rewarming	Intra-venous	tPA+/-aspirin+/-warfarin+/-heparin	initial bolus dose followed by a 6-hour infusion of tPA	Retrospective case review	24.6%
Jones *et al.* 2017[42]	7	No data	No data	Intra-venous	tPA + heparin +/- coumarin +/- antiplatelet	tPA at 0.15mg/kg IV bolus+ tPA. IV infusion (0.15 mg/kg) over 6h up to a total dose of 100mg. After: heparin+/-coumarin+/-antiplatelet agent	Retrospective case review	27.5%
Tavri *et al.* 2016[35]	13	At risk of tissue loss	?	Intra-arterial	t-PA	27,5 mg (12-48 mg) during 34h (12-72h)	Retrospective review	20,5%
Cauchy *et al.* 2016[43]	20	Severe	Rapid rewarming+ 250 mg aspirin +buflomedil 400 mg for 1 hour.	Intra-venous	Aspirin + tPA + ilosprost	tPA 100 mg, single dose + iloprost 2 ng/6 h+ Aspirin 250 mg	Retrospective case review	27.3% for grade 3, 44.4% for grade 4
	41	Severe	Rapid rewarming+ 250 mg aspirin +buflomedil 400 mg for 1 hour.		Aspirin + buflomedil	After, daily treatment of aspirin and buflomedil		62.5% for grade 3, 100% for grade 4
	58	Severe	Rapid rewarming+ 250 mg aspirin +buflomedil 400 mg for 1 hour.		Aspirin + iloprost	Aspirin and IV iloprost 2 ng/6 h		4.9% for grade 3, 66.7% for grade 4
Ibrahim *et al.* 2015[28]	3	Severe	Rapid rewarming+fluid replacement	Intra-arterial	tPA + heparin	tPA 4 mg bolus+infusion 1mg/hr+ heparin until PTT 50-70 s for maximum 48 h	Retrospective case review	0%
Handford *et al.* 2014[2]	-	Severe	-	Intra- arterial	tPA + heparin	tPA 3 mg over 15 min followed by constant infusion of 1 mg/h. Maximum 48h of no improvement + 500 units/hr heparin for 4 hours	Review	No data
Cauchy *et al.* 2011[21]	16	Severe frostbite (grade3/4)	Rapid rewarming of the areas with frostbite plus 250 mg of aspirin and IV administration of buflomedil (400 mg)	Intra -venous	Aspirin + iloprost + tPA	250 mg of aspirin + iloprost (2 ng per kilogram per minute for 6 hours per day) for 8 + tPA (100 mg) for the first day	Prospective, randomized, open-label Controlled trial	19%
	15	Severe frostbite (grade3/4)	Rapid rewarming of the areas with frostbite plus 250 mg of aspirin and IV administration of buflomedil (400 mg)		Aspirin + buflomedil	250 mg of aspirin and buflomedil (400 mg for 1 hour per day) for 8 days		60%
	16	Severe frostbite (grade 3/4)	Rapid rewarming of the areas with frostbite plus 250 mg of aspirin and IV administration of buflomedil (400 mg)		Aspirin + iloprost	250 mg of aspirin plus a prostacyclin (0.5 - 2 ng of loprost per kilogram of body weight per minute for 6 hours per day)		0%
Johnson *et al.* 2011[36]	11	Severe	No data	Intra-venous	tPA + heparin	0.15mg/kg bolus + 0.15mg/kg/h6h to a maximum of 100mg. Followed with heparin to PTT 2X control for 3–5 days	Retrospective case review	59%
Bruen *et al.* 2007[31]	6	Patients with perfusion defects	Immediate rewarming and fluid resuscitation as appropriate	Intra-arterial	tPA + heparin	tPA initial rate of 0.5 to 1.0 mg/h + Heparin at 500 U/h until normal perfusion or maximum 48 h	Retrospective case review	10%
	26	Varying degrees of injury severity. Not treated with thrombolytic therapy	Immediate rewarming and fluid resuscitation as appropriate	-	-	-		41.5%
Twomey *et al.* 2005[34]	13	No data	Rapid rewarming	Intra- venous	tPA + heparin	0.15 mg/kg bolus + 0.15 mg/kg/h6h to a maximum of 100 mg. Followed by IV heparin to PTT 2 control for 3–5 days, then Coumadin 4 weeks	2 Groups Arterial Venous Prospective, open label, unblinded	19% (not reported by route of administration)
	6	No data	Rapid rewarming	Intra -arterial	tPA + heparin	0.075 mg/kg/h 6 h. Repeated additional 6 h if repeat scan abnormal		19% (not reported by route of administration)

On the other hand, iloprost is a prostacyclin analogue with vasodilator and antiplatelet properties that has been associated with reductions in digital amputations in severe frostbite, so many authors recommend its intravenous administration as a first-line treatment[22,29]. Dose titration in published clinical experiences is based on the appearance of adverse effects within the therapeutic range (starting at 0,5-2 ng/kg/min, with 0,5 ng/kg/min increases every 30 minutes until the maximal toleration rate is achieved and maintaining its infusion 6 hours/day for 5-8 days), with consideration for the fact that the patient must be maintained in the supine position to prevent orthostatic hypotension[21]. The effect of its association with rt-PA is not well known, although in accordance with a recent randomized trial it seems to be optimal in grade IV frostbite within the first 12 hours[21]. Contraindications of its use include unstable angina, recent cardio-vascular events and increased risk of bleeding. The advantages of iloprost over rt-PA are that it does not require interventionist procedures, the therapeutic window is larger, it can be administered in patients with trauma and intensive monitoring, other than blood pressure monitoring, is not required.

Tetanus vaccination is recommended, according to the usual schedule.

If amputation is required, surgical intervention must be delayed until viable tissue can be demarcated accurately, provided that an emergency justification for proceeding (e.g. gangrene, sepsis and compartment syndrome). This measure is justified by the possibility that tissue initially considered non-viable is restored[37] and the risk of surgical trauma interfering with the healing of proximal tissues[4,6]. This is not carried out in normal conditions until at least four to six weeks after injury, including in patients receiving thrombolytic therapy, which can imply the need for psychological support.

Sequelae

Sequelae after frostbite are common and occur independently of its severity. In a study of 30 patients with grade II frostbite, 63% were found to have sequelae after four to 11 years from injury (cold sensitivity 53%, digital numbness 40%, reduction in touch sensitivity 33%)[38]. It is estimated that sensitivity disturbances are present for at least four years in nearly all those who have suffered from frostbite[11].

Chronic pain secondary to frostbite is very common and is usually refractory to conventional analgesics. It sometimes responds to drugs designed for neuropathic pain (e.g. amitriptyline and gabapentin). Despite efforts to treat later symptoms (e.g. pain, paraesthesia and numbness) with chemical and surgical sympathectomies, there is no clear indication for their use.

Other common problems include hyperhidrosis, secondary to an abnormal response of the sympathetic system, trophic alterations in skin and fanerae, digital flexor retraction and high susceptibility to future cold-related injuries. Alterations of skin colouration, ranging from depigmentation to local cyanosis, are not uncommon.

Long-term sequelae include osteoporosis, and where the frostbite affected the joints, osteoarthrosis with damaged joint surfaces and a decline in joint mobility with tendinous retractions of the flexor musculature[39].

Digital amputations (partial or total) involve functional limitations to daily life and sporting activities, given the alteration in the normal biomechanics of the limb. Gait will be severely impaired if frostbite affects the metacarpophalangeal joint in the foot. Risk factors related to amputation include duration of exposure to cold, absence of proper equipment, exposure to cold in remote areas, presence of infection and delay in treatment.

There is a broad consensus on the need to prioritise an early multidisciplinary rehabilitation programme for patients who have undergone amputation, including prompt controlled mobilisation to prevent tendinous retraction and reach optimal levels of functional recovery[10,15], and long-term, non-aggressive treatment[40] (Table 3).

Conclusions

Frostbite is no longer primarily an occupational pathology or characteristic of subjects with cognitive impairment. It has become a common cause of morbidity among healthy young adults who voluntarily expose themselves to cold, usually while practising winter sports. Knowledge of activity planning, survival skills and cold protection is strongly recommended as basic prevention tools. Early recognition of frostbite is essential to ensure prompt diagnosis and early initial treatment, since a delay in first rewarming is associated with a worse prognosis. At present, in-field treatments are relatively basic and can be initiated by non-qualified subjects with the proper training. New perspectives are focusing on improving initial care by applying advanced treatments under medical supervision. For superficial frostbite, there is no need for further complementary tests beyond the clinical monitoring of the injury. For severe frostbite, scintigraphy with Tc99 is a good prognosis predictor after 48 hours of injury. Angiography is both an imaging and a therapeutic tool, but less invasive options such as MRI and ultrasound appear to be good alternatives when direct thrombolysis is not required. Emerging hospital treatments have a therapeutic window that needs to be known to take fast and optimal decisions regarding patient evacuation, considering the rescue time lapses and the hospital resources of each mountain area and country. Surgical interventions must be delayed until there is a clear demarcation of the necrotic area. Long-term sequelae are prevalent among subjects with frostbite, even in non-severe injuries. A multidisciplinary approach to caring for patients with frostbite is needed in the management of long-term functional sequelae.

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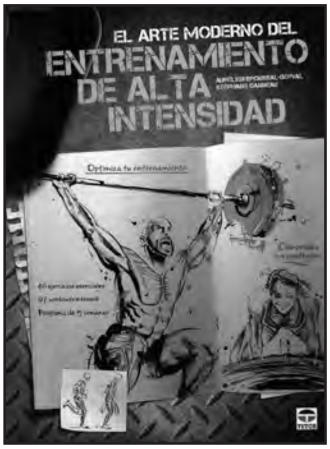

EL ARTE MODERNO DEL ENTRENAMIENTO DE ALTA INTENSIDAD

Por: Aurélien Broussal-Derval y Stéphane Ganneau
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Impresores 20. P.E. Prado del Espino. 28660 Boadilla del Monte. Madrid.
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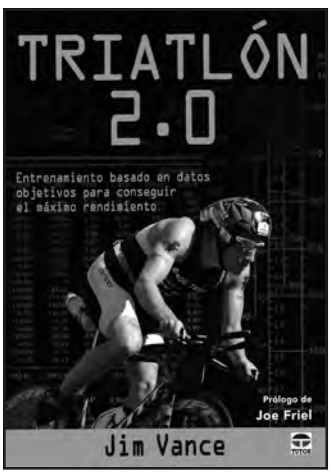
E-mail: info@edicionestutor.com Web: www.edicionestutor.com
Madrid 2017, 224 páginas, P.V.P: 25 euros

Esta obra singular es deporte y fuerza, movimiento y pasión. Es una guía distinta de cualquier otra. Desde los asombrosos dibujos hasta los 127 *workouts,* está diseñada para marcar diferencias. Tanto si se utiliza su contenido como complemento de un programa de entrenamiento ya existente, como si se quiere sustituir un programa de entrenamiento que se ha vuelto monótono y obsoleto, se ojeará y utilizará este libro una y otra vez.

Se verá el entrenamiento de alta intensidad con ojos completamente nuevos. En sus páginas se encontrarán 40 ejercicios, cada uno detallado y representado de manera artística, fotografías y variaciones; 127 *workouts* y circuitos para combinar cosas diferentes; recomendaciones para el calentamiento, la seguridad y la prevención de lesiones; y si se está dispuesto a asumir el reto: un programa original de 15 semanas de duración.



TRIATLÓN 2.0

Por: Jim Vance
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Madrid 2017, 272 páginas, P.V.P: 25 euros

Puede que los triatletas comprometidos sean de todos los deportistas, los mayores entendidos en tecnología. Tienen los dispositivos de vanguardia y saben que los datos para mejorar su rendimiento están a mano, pero unirlo todo puede resultar una tarea confusa de enormes proporciones. El triatleta, entrenador, investigador y autor Jim Vance sostiene que, a pesar del acceso a la información adecuada, la mayoría de los triatletas empiezan las pruebas infraentrenados o sobreentrenados. Por eso ha desarrollado Triatlón 2.0, el primer programa que se aprovecha de lo último en ciencia y tecnología.

El libro examina los aparatos más utilizados en este deporte, como medidores de potencia (o vatímetros) para ciclismo, rastreadores GPS y monitores de frecuencia cardíaca (o pulsómetros). Recoge las mediciones más precisas, descubre lo que significan e, igual de importante, lo que no. Y así después, el lector puede poner los números a su propio servicio: trasladando sus datos a un programa exhaustivo basado en sus necesidades de rendimiento y objetivos.

ENTRENAMIENTO DEL CORE PARA CORREDORES

Por: Christian Roberto López Rodríguez
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Madrid 2017, 200 páginas, P.V.P: 18 euros

Correr es algo más que ponerse las zapatillas, tener voluntad y tratar de sumar kilómetros. Los corredores deben completar su preparación con ejercicios de fuerza específicos y de la zona media, el core, para maximizar la eficacia de carrera y desarrollar una óptima longitud y frecuencia de zancada. Esta obra ofrece una metodología innovadora para el desarrollo de la musculatura de la zona media basada en el empleo progresivo de movimientos funcionales, sin necesidad de máquinas de musculación, y teniendo en cuenta toda la temporada a través de una periodización deportiva bien planificada.

Los ejercicios, con variantes para progresar, se describen de tal manera que cualquier deportista puede llevarlos a la práctica siguiendo las pautas metodológicas desde el comienzo del programa. Este libro no solo servirá a los *runners* de cualquier nivel, desde los de carreras populares hasta profesionales de élite, sino a todos los deportistas que en su disciplina, individual o de equipo, requieren de la carrera en los movimientos en el terreno de juego.

2018		
Congrès francophone de médecine de montagne	17-21 Enero Champéry, (Suiza)	web: www.grimm-vs.ch
II Jornadas Nacionales SETRADE	15-16 Marzo Vitoria	http://www.setrade.org/congresos/jornadasvitoria2018/
36 Congress International Society for Snowsports Medicine	15-17 Marzo Arosa (Suiza)	web: http://www.sitemsh.org/
15th International Scientific Conference and 14th annual Congress of the Montenegrin Sports Academy	12-15 Abril Bubva (Montenegro)	web: http://csakademija.me/conference/
World Congress on Osteoporosis, Osteoarthritis and Musculoskeletal Diseases	19-22 Abril Cracovia (Polonia)	web: www.wco-iof-esceo.org/
18th ESSKA Congress	9-12 Mayo Glasgow (Reino Unido)	web: http://esska-congress.org/
56 Congreso SERMEF	16-19 Mayo Gijón-Asturias	www.sermef.es
7thWorld Conference on Women and Sport	17-20 Mayo Gaborone (Bostwana)	web: www.icsspe.org/sites/default/files/e8_7TH%20 IWG%20Conference%20docx.pdf
XXII Curso Asoc. Española Med. del Futbol	25-26 Mayo Málaga	web: http://www.aemef.org/es/
XXVII Isokinetic Medical Group conference:"Football medicine outcomes. Are we winning?"	2-4 Junio Barcelona	E-mail: conference@isokinetik.com web: www.footballmedicinestrategies.com
5th International Congress of Exercise and Sport Sciences	5-10 Junio Netanya (Israel)	web: https://events.eventact.com/EventsList/ 5sportsceince2017/General-Information
European Congress of Adapted Physical Activity (EUCAPA)	3-5 Julio Worcester (Reino Unido)	Andrea Faull. E-mail: a.faull@worc.ac.uk Ken Black. E-mail: k.black@worc.ac.uk
23rd Annual Congress of the European College of Sport Science	4-7 Julio Dublín (Irlanda)	web: www.ecss-congress.eu/2018/
World Congress of Biomechanics	8-12 Julio Dublín (Irlanda)	web: http://wcb2018.com/
12th World Congress of the International Society of Physical and Rehabilitation Medicine (ISPRM)	8-12 Julio París (Francia)	web: http://isprm2018.com/
The Annual World Congress of Orthopaedics	25-27 Julio Milán (Italia)	web: http://www.bitcongress.com/wcort2018 / http://www.bitcongress.com/wcort2018/programlayout.asp
World Congress of the Association Internationale des Ecoles Supérieures d'Education Physique (AIESEP)	25-28 Julio Edimburgo (Reino Unido)	web: http://aiesep.org/

XXXV Congreso Mundial de Medicina del Deporte	12-15 Septiembre Rio de Janeiro (Brasil)	web: www.fims.org
5th International Scientific Tendinopathy Symposium (ISTS)	27-29 Septiembre Groningen (Países Bajos)	web: http://ists2018.com/
VII Congreso Asociación Hispanoamericana de Médicos del Fútbol	Octubre Lima (Perú)	web: http://hispamef.com/
28º Congress European Society for surgery of the shoulder and the elbow (SECEC-ESSSE)	Ginebra (Suiza)	web: www.secec.org
2019		
12th Biennial ISAKOS	12-16 Mayo Cancún (México)	web: www.isakos.com
24th Annual Congress of the European College of Sport Science	3-6 Julio Praga (Rep. Checa)	E-mail: office@sport-science.org
14th International Congress of shoulder and elbow surgery (ICSES)	17-20 Septiembre Buenos Aires (Argentina)	web: www.icses2019.org
XV Congreso Nacional de Psicología de la Act. Física y del Deporte	Zaragoza	web: www.psicologiadeporte.org
2020		
25th Annual Congress of the European College of Sport Science	1-4 Julio Sevilla	E-mail: office@sport-science.org
XXXVI Congreso Mundial de Medicina del Deporte	24-27 Septiembre Atenas (Grecia)	web: www.globalevents.gr
2021		
26th Annual Congress of the European College of Sport Science	7-10 Julio Glasgow (Reino Unido)	E-mail: office@sport-science.org

Cursos *on-line* SEMED-FEMEDE

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Curso dirigido a los titulados de las diferentes profesiones sanitarias y a los titulados en ciencias de la actividad física y el deporte, destinado al conocimiento de las prestaciones y rendimiento del deportista, para que cumpla con sus expectativas competitivas y de prolongación de su práctica deportiva, y para que la práctica deportiva minimice las consecuencias que puede tener para su salud, tanto desde el punto de vista médico como lesional.

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Curso "FISIOLOGÍA Y VALORACIÓN FUNCIONAL EN EL CICLISMO"

Curso dirigido a los titulados de las diferentes profesiones sanitarias y a los titulados en ciencias de la actividad física y el deporte, destinado al conocimiento profundo de los aspectos fisiológicos y de valoración funcional del ciclismo.

Curso "AYUDAS ERGOGÉNICAS"

Curso abierto a todos los interesados en el tema que quieren conocer las ayudas ergogénicas y su utilización en el deporte.

Curso "CARDIOLOGÍA DEL DEPORTE"

ACREDITADO POR LA COMISIÓN DE FORMACIÓN CONTINUADA (VÁLIDA DEL 15/10/2016 AL 15/10/2017) CON 8,78 CRÉDITOS

Fecha límite de inscripción: 15/06/2017

Curso dirigido a médicos destinado a proporcionar los conocimientos específicos para el estudio del sistema cardiocirculatorio desde el punto de vista de la actividad física y deportiva, para diagnosticar los problemas cardiovasculares que pueden afectar al deportista, conocer la aptitud cardiológica para la práctica deportiva, realizar la prescripción de ejercicio y conocer y diagnosticar las enfermedades cardiovasculares susceptibles de provocar la muerte súbita del deportista y prevenir su aparición.

Curso "ALIMENTACIÓN, NUTRICIÓN E HIDRATACIÓN EN EL DEPORTE"

Curso dirigido a médicos destinado a facilitar al médico relacionado con la actividad física y el deporte la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para prescribir una adecuada alimentación del deportista.

Curso "ALIMENTACIÓN Y NUTRICIÓN EN EL DEPORTE"

Curso dirigido a los titulados de las diferentes profesiones sanitarias (existe un curso específico para médicos) y para los titulados en ciencias de la actividad física y el deporte, dirigido a facilitar a los profesionales relacionados con la actividad física y el deporte la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para conocer la adecuada alimentación del deportista.

Curso "ALIMENTACIÓN Y NUTRICIÓN EN EL DEPORTE" Para Diplomados y Graduados en Enfermería

ACREDITADO POR LA COMISIÓN DE FORMACIÓN CONTINUADA (NO PRESENCIAL 15/12/2015 A 15/12/2016) CON 10,18 CRÉDITOS

Curso dirigido a facilitar a los Diplomados y Graduados en Enfermería la formación precisa para conocer los elementos necesarios para la obtención de los elementos energéticos necesarios para el esfuerzo físico y para conocer la adecuada alimentación del deportista.

Curso "CINEANTROPOMETRÍA PARA SANITARIOS"

Curso dirigido a sanitarios destinado a adquirir los conocimientos necesarios para conocer los fundamentos de la cineantropometría (puntos anatómicos de referencia, material antropométrico, protocolo de medición, error de medición, composición corporal, somatotipo, proporcionalidad) y la relación entre la antropometría y el rendimiento deportivo.

Curso "CINEANTROPOMETRÍA"

Curso dirigido a todas aquellas personas interesadas en este campo en las Ciencias del Deporte y alumnos de último año de grado, destinado a adquirir los conocimientos necesarios para conocer los fundamentos de la cineantropometría (puntos anatómicos de referencia, material antropométrico, protocolo de medición, error de medición, composición corporal, somatotipo, proporcionalidad) y la relación entre la antropometría y el rendimiento deportivo.

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