

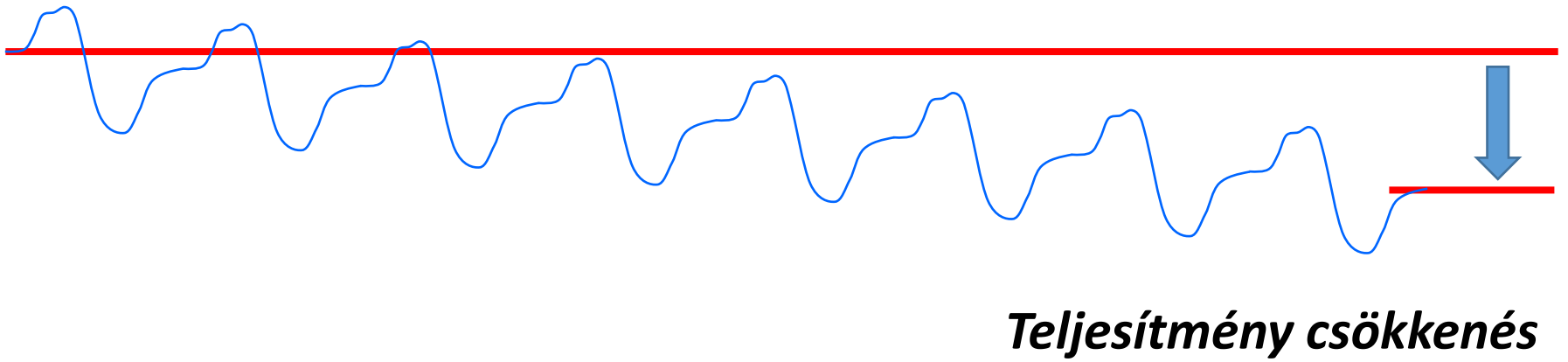
A silhouette of a person bending over, possibly in a state of exhaustion or contemplation, against a warm, orange-hued sunset background. The person is on the left side of the frame, and their shadow is cast on the ground. The background shows a horizon line over water or a flat landscape.

Túledzés élettana

Radák Zsolt

TF

Ábra 3.7

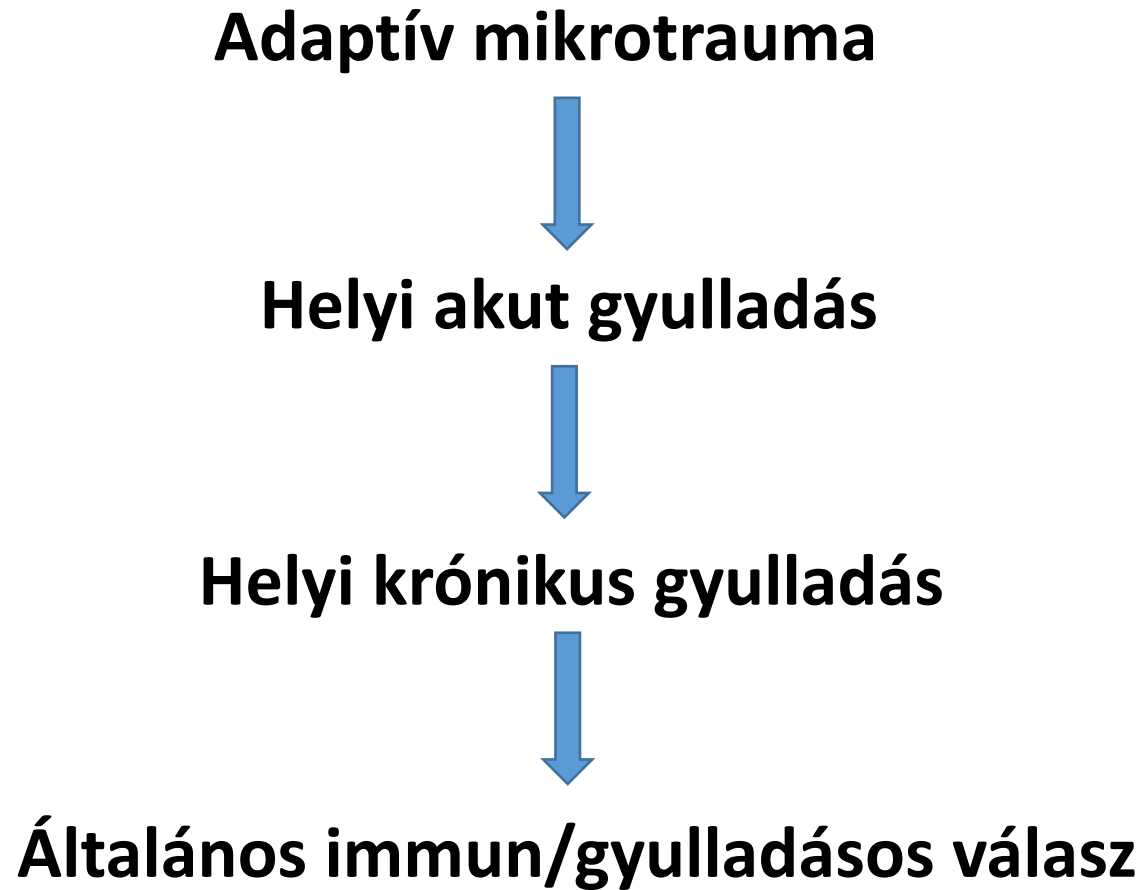


Mérni, mérni és mérni

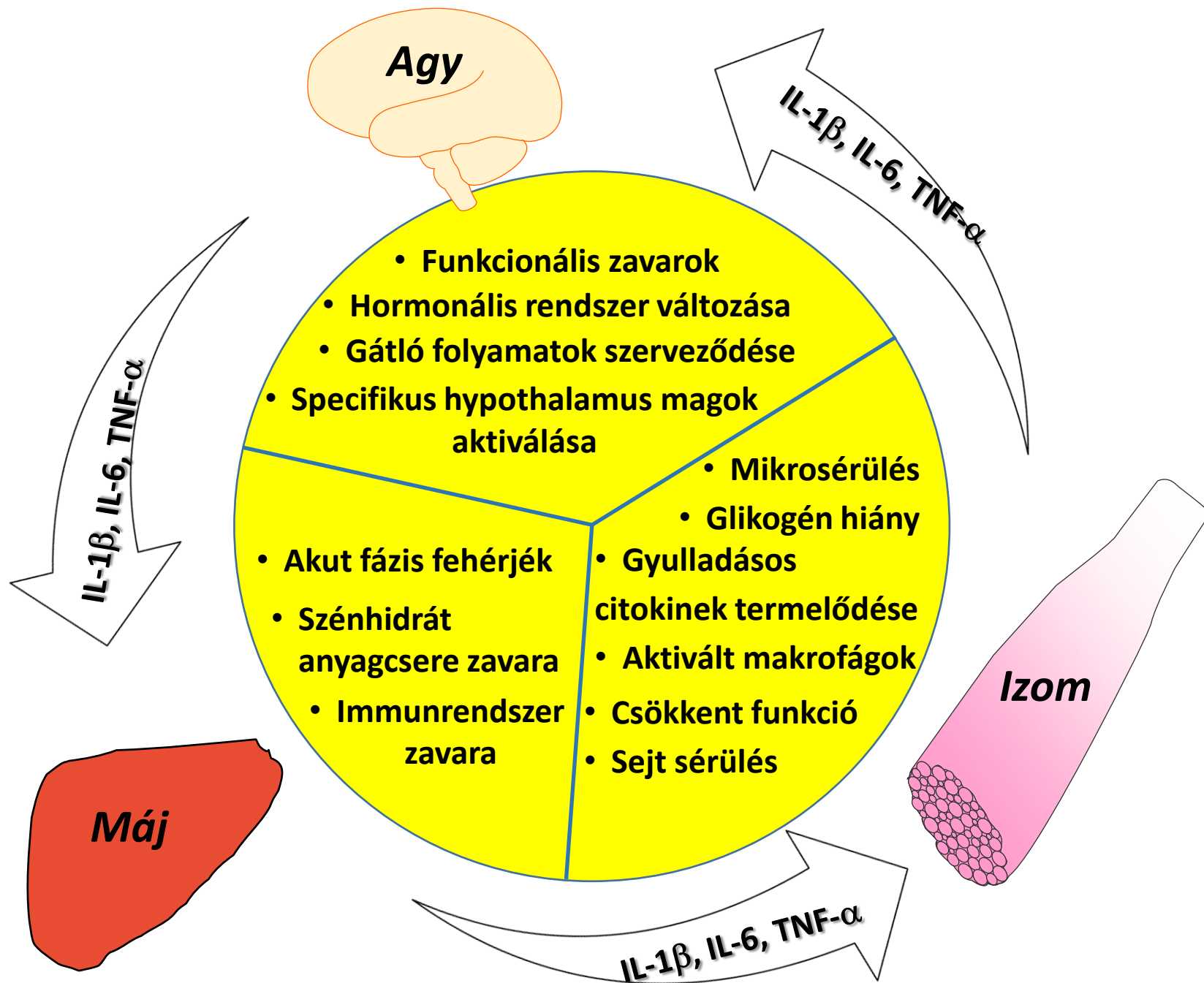
- Nyugalmi, maximális és megnyugvási pulzus
- Erő, gyorsaság, állóképességi tesztek, LT küszöb (sport specifikus legyen)
- „Biomarkerek”: CK, húgysav, cfDNS, kortizol/tesztoszteron, glutamin
-
- Alvás minőség: REM és NREM szakasz, alvás közbeni mozgás mennyiség

3.1. táblázat

Túledzettség jelei			
Élettani	Biokémiai	Immunológiai	Pszichés
Csökkent teljesítmény	Negatív fehérje egyensúly	Megnövekedett érzékenység	Depresszió
Romló koordináció	Romló cukor tolerancia	mindenfajta betegségre	Csökkenő önbizalom
Csökkent ferritin szint	Csökkenő izom glikogén	Felső légúti megbetegedések	Emocionális instabilitás
Csökkent ásványi anyag felszívás	Csökkenő csont ásványi anyag tartalom	Nyirokcsomó duzzadás	Koncentráció hiány
Abnormális T-hullám az EKG-n	Vashiány	Láz	Félelem
Csökkent terhelés tolerancia	Emelkedett kortizol szint	Bakteriális fertőzések	Kitartás hiánya
Krónikus fáradtság	Csökkent tesztoszteron szint	Herpesz	
Emelkedett nyugalmi, romló megnyugvási pulzus	Csökkenő glutamin szint	Csökkent limfocita szám	
Csökkent testsúly			
Emelkedett O ₂ felhasználás submax. terhelésnél			
Alvás zavar			
Étvágytalanság			
Ízület és izom fájdalmak			



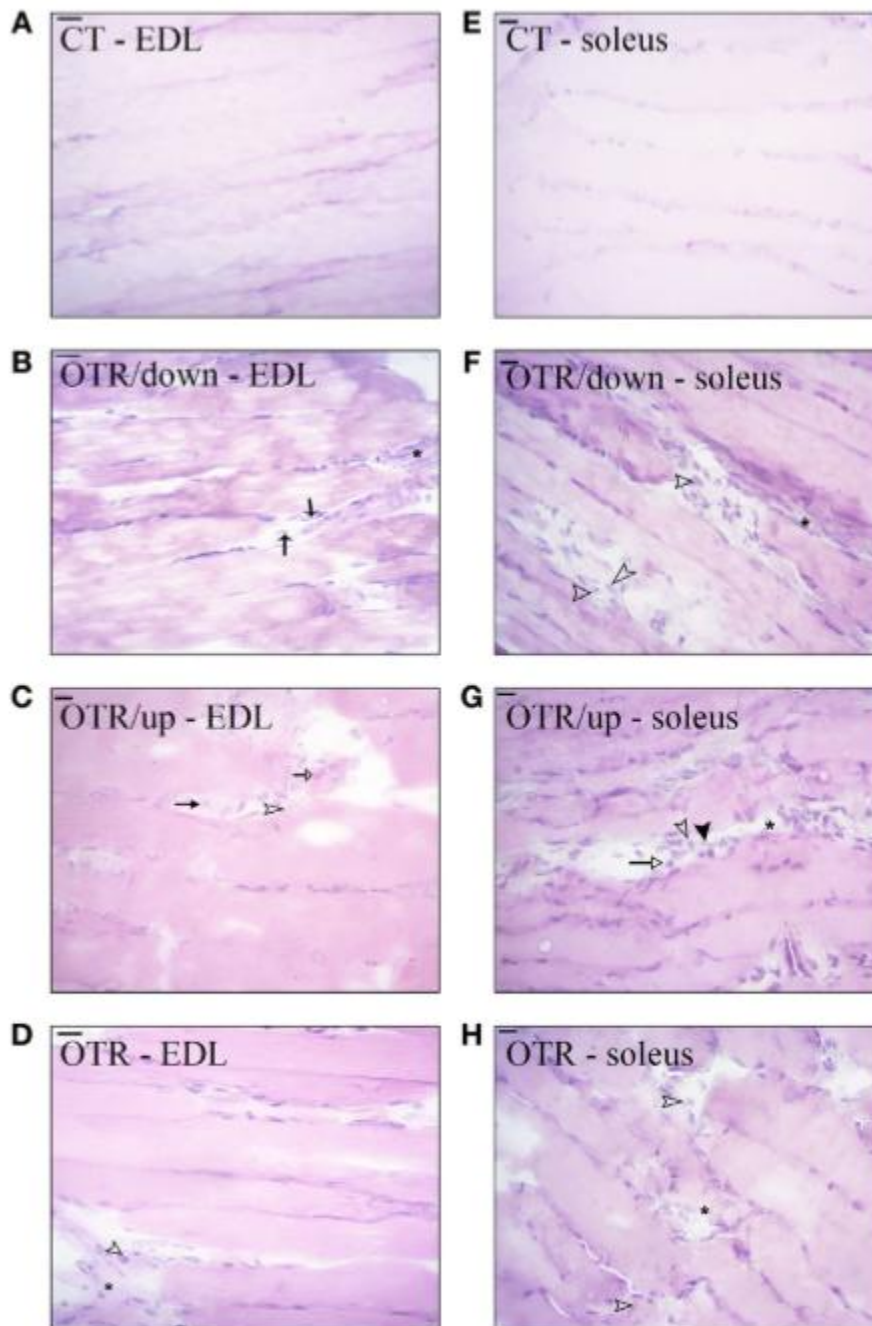
Ábra 3.9



Citokinek



Emelkedő és lejtőn futással okozott túledzés



Excessive training



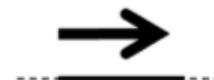
Nonfunctional overreaching



↑↑ proinflammatory cytokines



↑↑ proinflammatory cytokines



↑ proinflammatory cytokines

↑ IL-15

↑ Corticosterone

Serum

EDL

↑ proinflammatory cytokines

↓ p-AR

↓ GR

↑ IL-6

↓ Type IIa fiber

↓ p-AR

↑ proinflammatory cytokines

↑ Type IIb fiber

↓ p-AR

↓ GR

Muscle

Soleus

↑ proinflammatory cytokines

Micro-injuries

↑ Type IIb fiber

↓ p-AR

↓ GR

↑ IL-6

Micro-injuries

↓ Type IIa fiber

↓ AR

Micro-injuries

↓ GR

Cell Free DNA

Investigations:

- Cancer (*circulating tumor DNA – ctDNA*)
- Myocardial infarction
- Pro-inflammatory diseases (cirrhosis, hepatitis, systemic lupus erythematosus, rheumatoid arthritis)
- Chronic kidney disease
- Traumatic brain injury
- Exercise
- Prenatal screening "NIPT" (*cell free fetal DNA - cffDNA*)



cfDNA concentration in blood



17 fitt ffi vett rész a 12 hetes vizsgálatban, mely négy 3 hetes részre volt bontva (t1, t2, t3, t4).

Az első és negyedik periodusban az edzés terjedelem alacsony volt, míg a t2-ben nagy terjedelmű volt a terhelés.

A t3 maximális intenzitású edzéseket foglalt magában.

Table 1. Training volume and performance as well as CK, CRP, and UA concentration changes at rest (Baseline), and after low- (t1), high- (t2), very high- (t3), and low-volume (t4) resistance training

	Baseline	t1	t2	t3	t4
Mean training volume, tonnage/wk (SE) ^a	N/A	2.2 (0.2)	7.8 (0.6) ^d	14.8 (1.4) ^{d,c}	1.7 (0.3) ^f
Performance (maximal strength), kg (SE) ^b	96.6 (17.1)	99.2 (11.7)	109.6 (14.8) ^{c,d}	104.5 (14.9) ^c	107.2 (12.5) ^{c,d}
Cell-free plasma DNA, 8GenEq/mL (SE)	31.4 (13.8)	143.5 (22.9) ^c	289.9 (41.1) ^{c,d,e}	605.7 (116.4) ^{c,f}	74.8 (29.6) ^{c,d,f}
	(14.0–65.9)	(76.6–180.6)	(196.9–383.0)	(251.8–959.5)	(27.7–141.8)
CK activity, units/L (SE)	102.6 (19.1)	134.5 (20.1)	296.6 (49.6)	368.1 (44.0) ^{c,d}	161.7 (23.8) ^f
	(59.4–149.8)	(86.7–82.2)	(185.5–407.6)	(264.4–513.9)	(105.7–217.7)
CRP, mg/L (SE)	0.84 (0.17)	0.86 (0.19)	3.364 (1.07) ^{c,d}	4.8 (1.04) ^{c,d}	1.42 (0.30) ^f
	(0.45–1.24)	(0.42–1.31)	(1.00–5.77)	(1.89–6.64)	(0.73–2.11)
UA, mg/L (SE)	39.1 (4.0)	42.4 (4.0)	51.2 (3.7) ^c	66.6 (5.8) ^{c,d}	43.8 (4.3) ^f
	(30–48.2)	(33.3–51.5)	(42.8–59.6)	(53.4–79.5)	(34.1–53.5)

^a Mean number of tons lifted per week for all resistance exercises employed in the protocol. NA, not applicable.

^b Mean maximal strength in 1 representative exercise (squat).

^c Significant difference with baseline (T0).

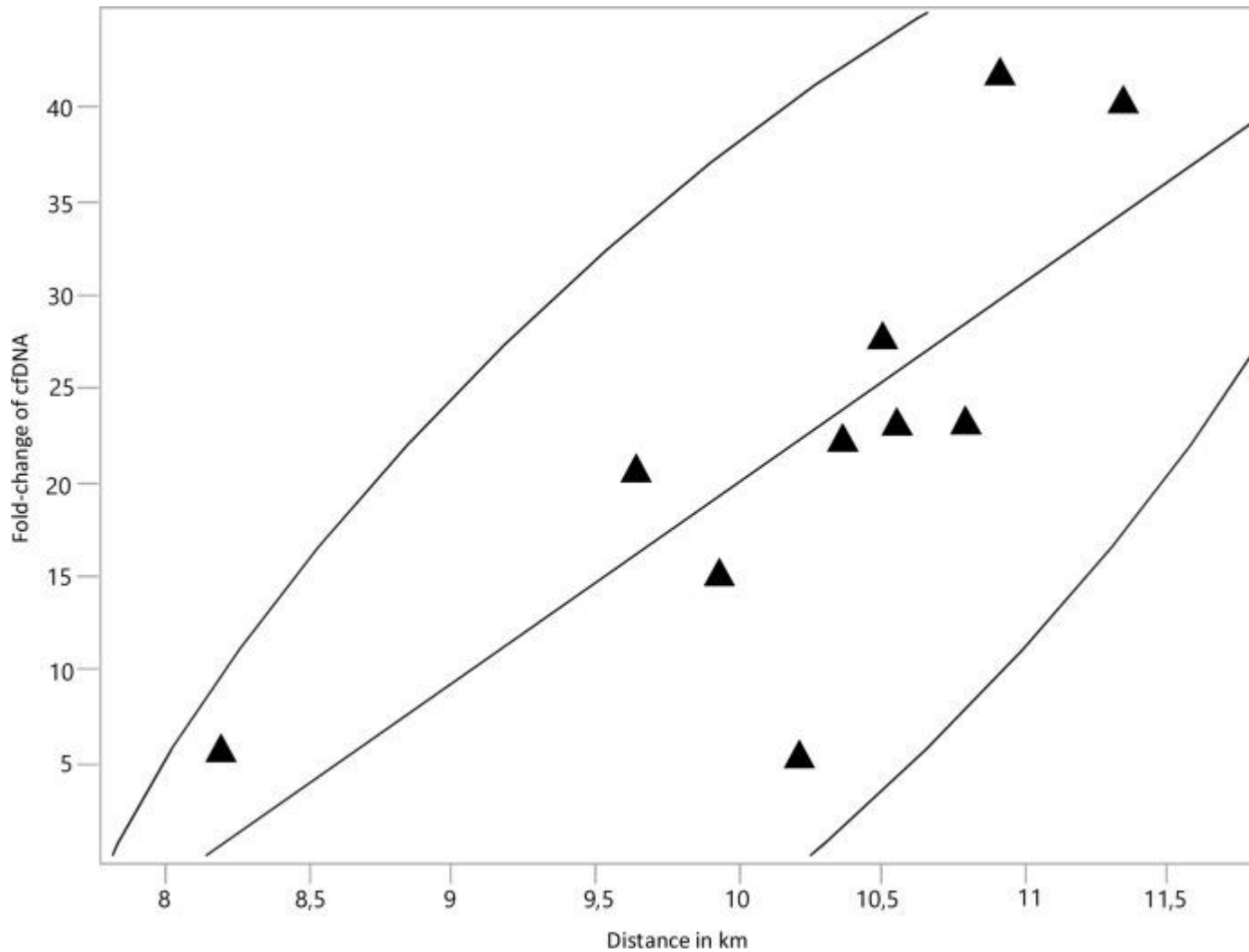
^d Significant difference with t1.

^e Difference between t3 and t4.

^f Significant difference between t4 and t3.

^g Significant difference with t4. Minimum and maximum values are shown in parentheses.

Correlation between total distance covered and fold-changes of cfDNA after the football game.



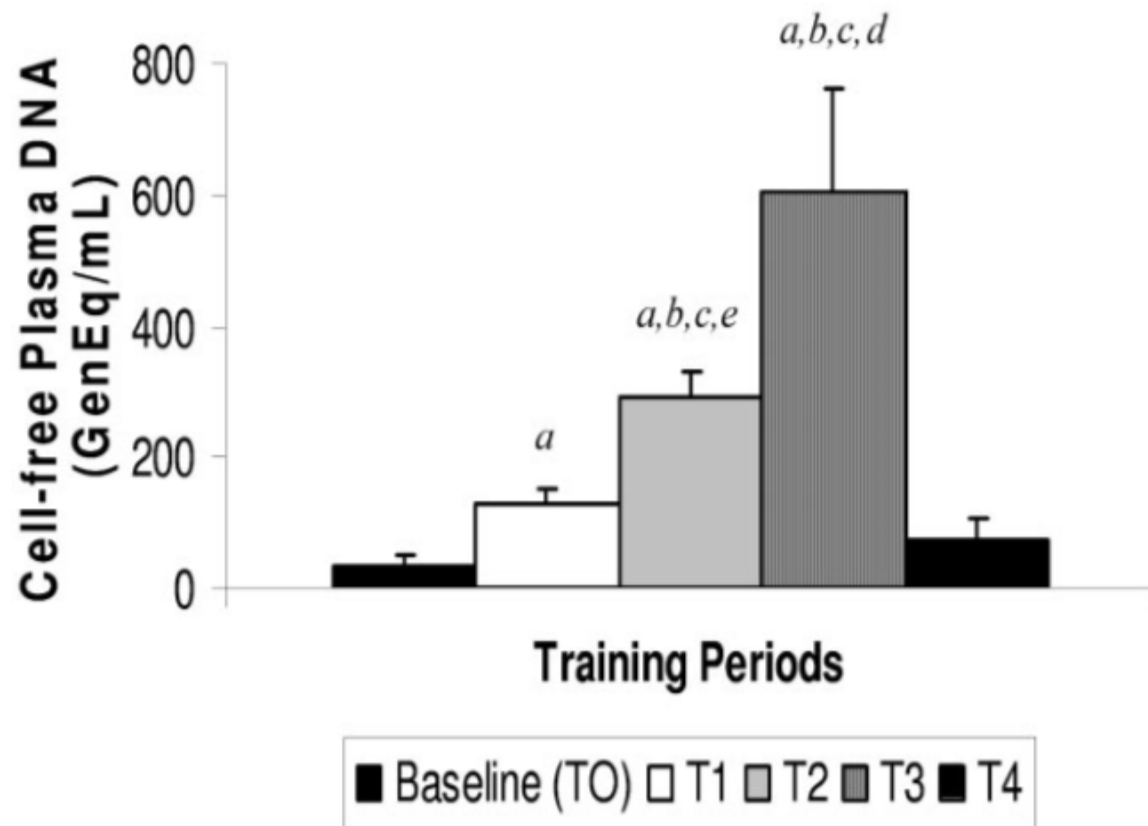


Fig. 1. Cell-free plasma DNA concentration in 17 study participants measured sequentially at rest (t0) and after low- (t1), high- (t2), very high- (t3), and low-volume (t4) resistance training.

^a Significant difference with baseline (t0)

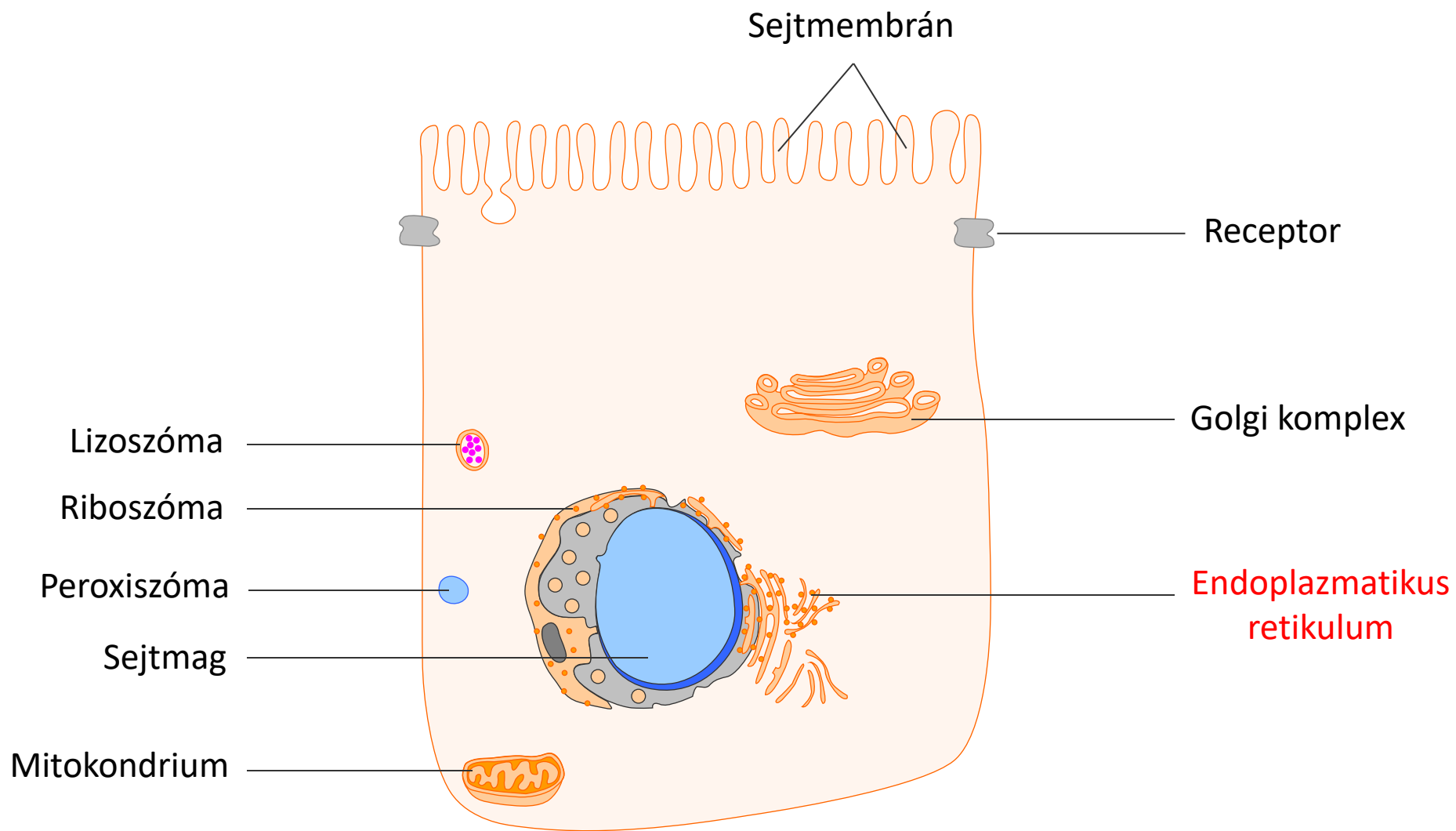
^b Significant difference with t1.

^c Significant difference between t3 and t2.

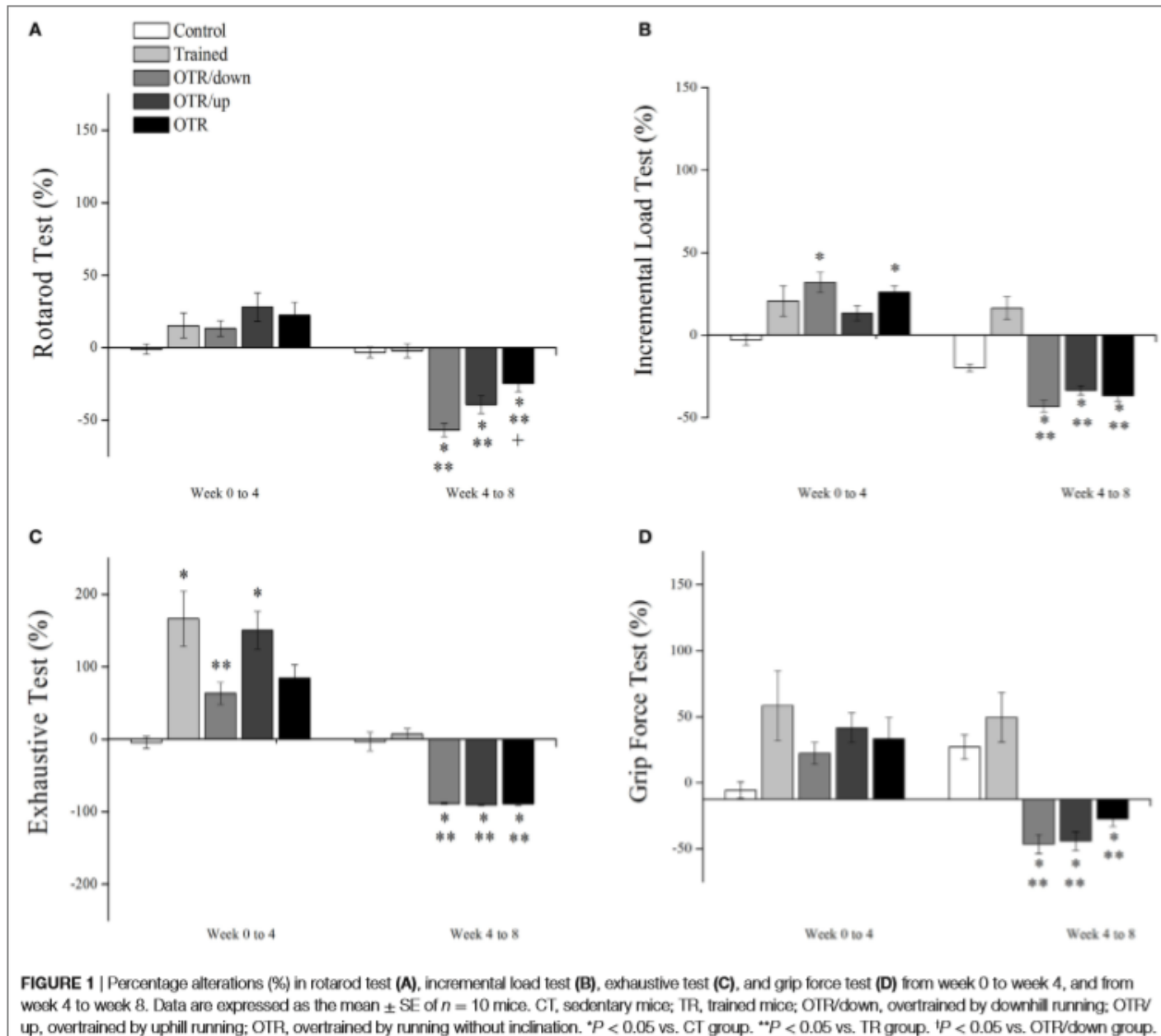
^d Significant difference between t4 and t3.

^e Significant difference with t4.

Ábra 1.1



Emelkedő és lejtőn futással okozott túledzés



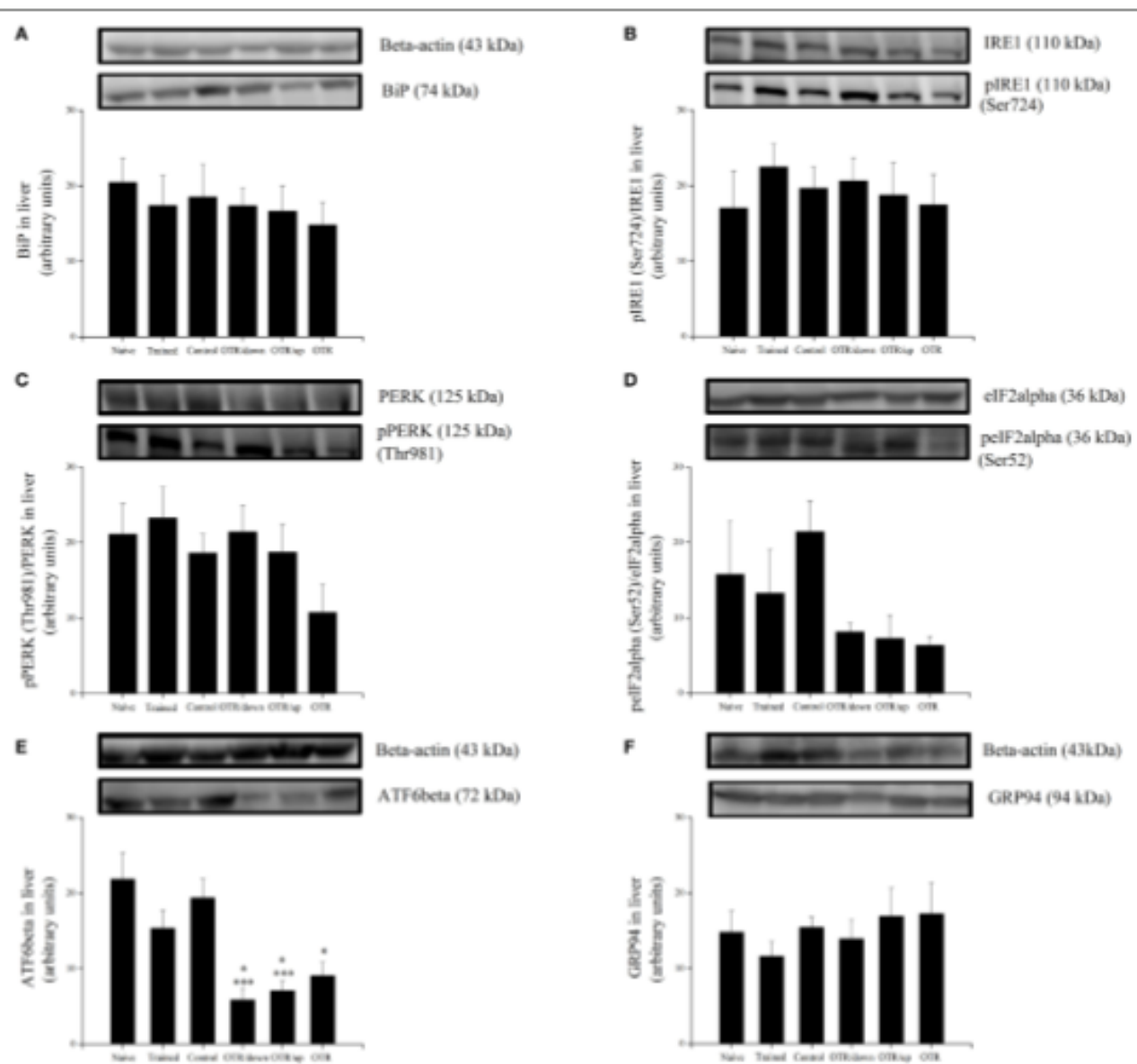


FIGURE 2 | Protein levels (arbitrary units) of BiP (A), pIRE1 (Ser724)/inositol-requiring enzyme 1 (IRE1) (B), pPERK (Thr981)/protein kinase RNA-like endoplasmic reticulum kinase (PERK) (C), pelf2alpha (Ser52)/eIF2alpha (D), ATF6beta (E), and glucose-regulated protein 94 (GRP94) (F) in the livers of experimental animals. Data are expressed as the mean $\pm$ SE of $n = 6$ mice. N, sedentary mice; TR, trained mice; CT, sedentary mice; OTR/down, overtrained by downhill running; OTR/up, overtrained by uphill running; OTR, overtrained by running without inclination. * $P < 0.05$ vs. CT group. *** $P < 0.05$ vs. N group. The original experiments are available in supplementary file 1 (i.e., BiP, pelf2alpha, eIF2alpha, and ATF6beta), file 2 (i.e., pIRE1, IRE1, pPERK, and PERK), and file 3 (i.e., GRP94).

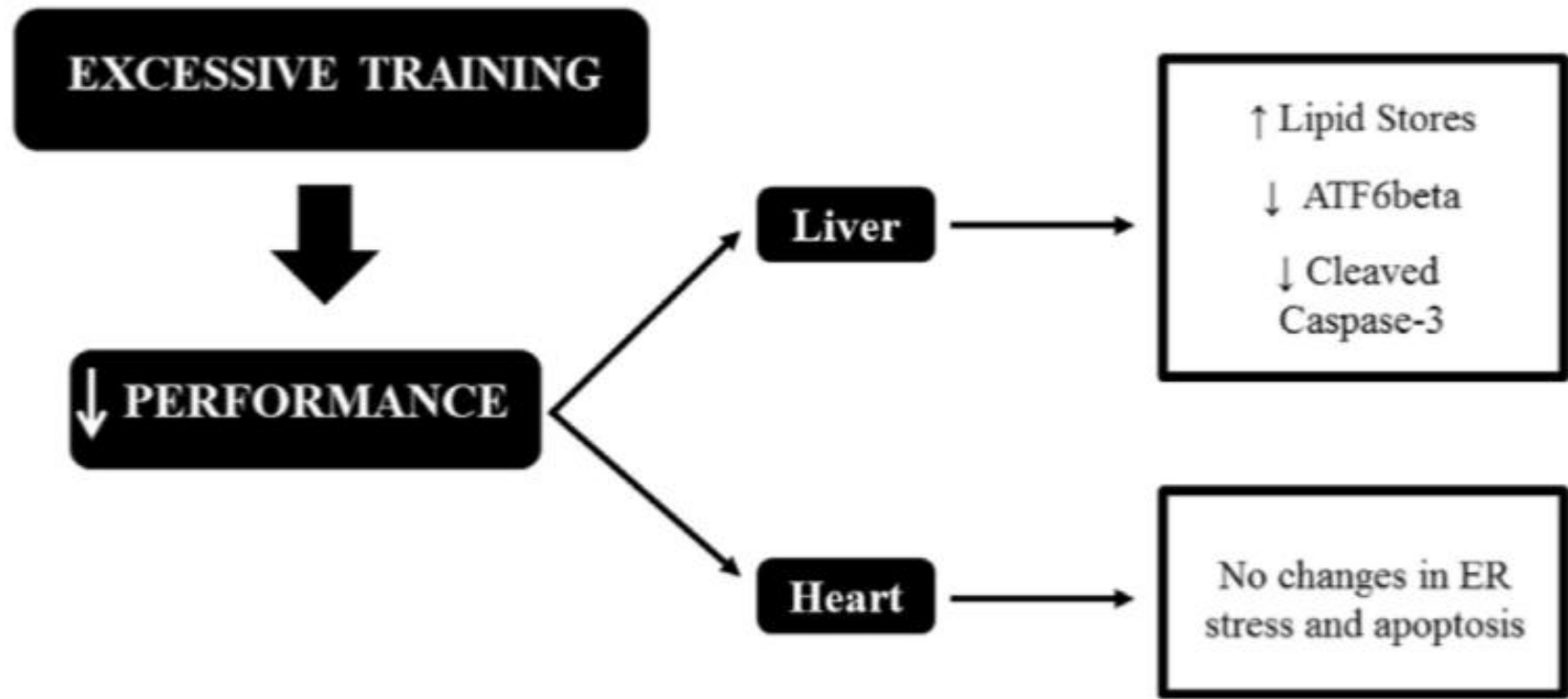
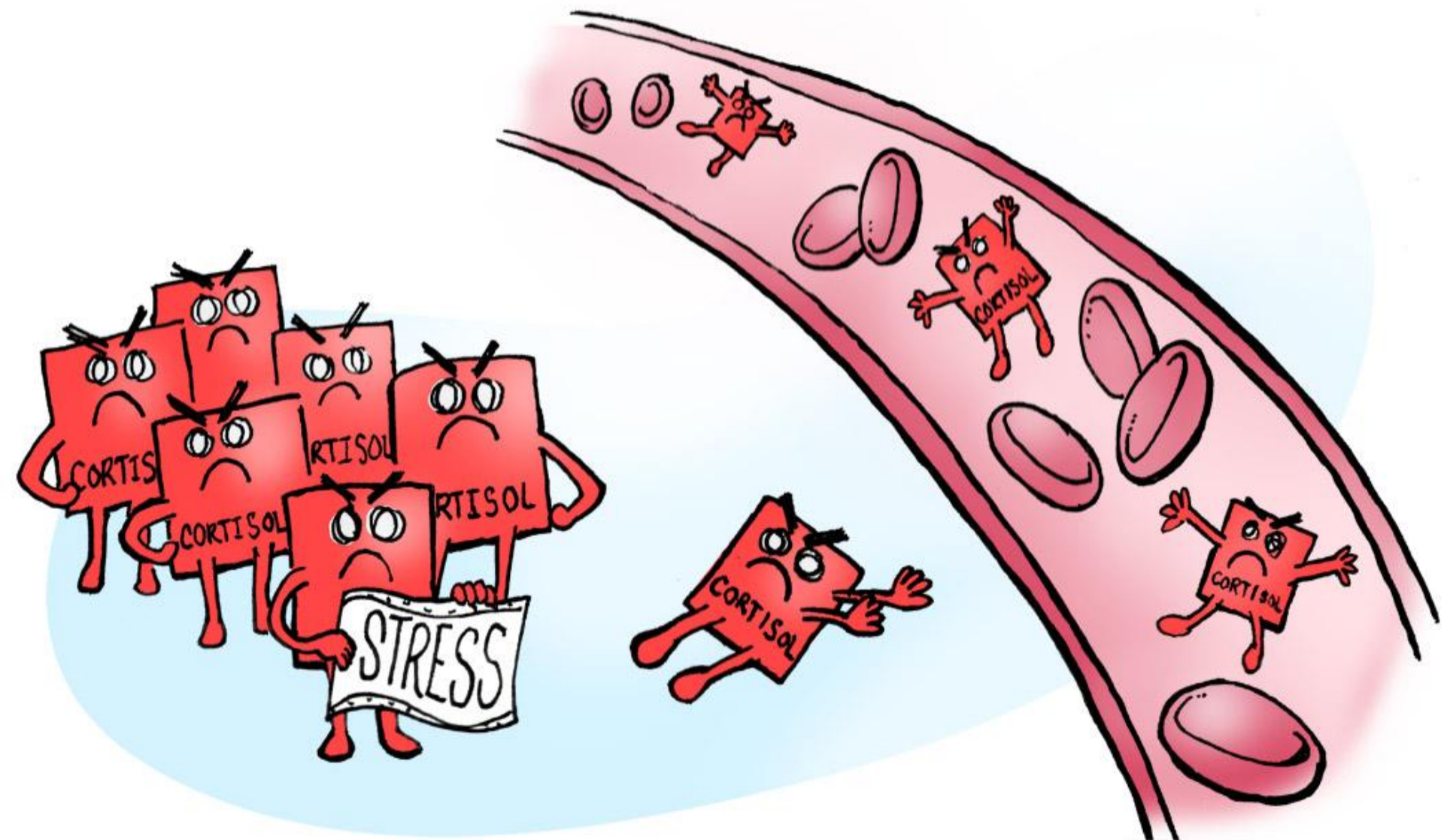


FIGURE 6 | Schematic model summarizing the relationships between endoplasmic reticulum (ER) stress, apoptosis, and excessive training in the liver and heart of C57BL/6 mice.



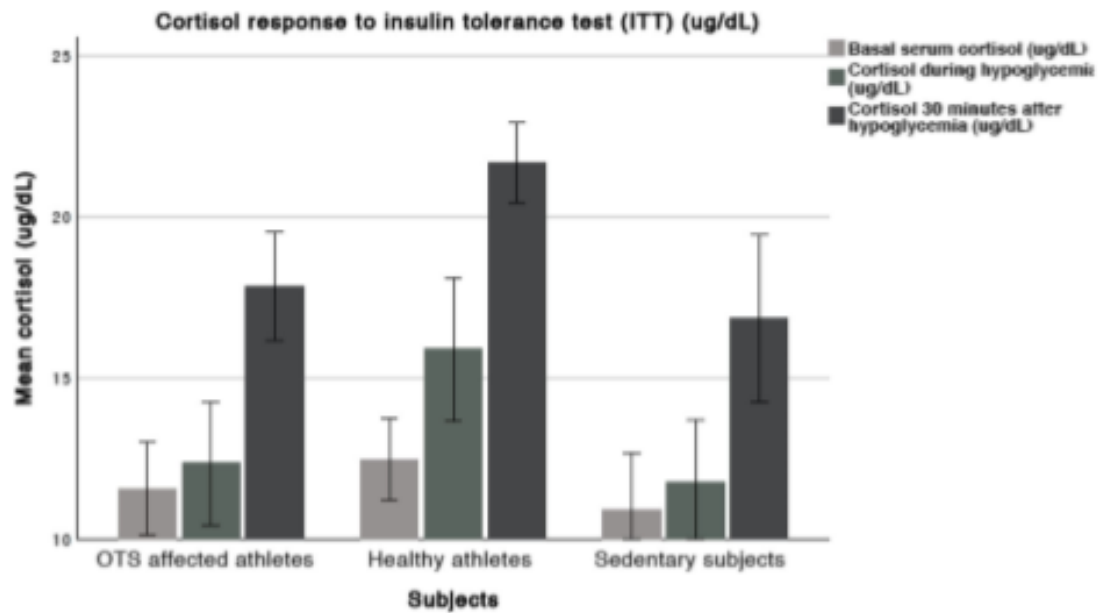


Fig. 1 Cortisol response to an ITT

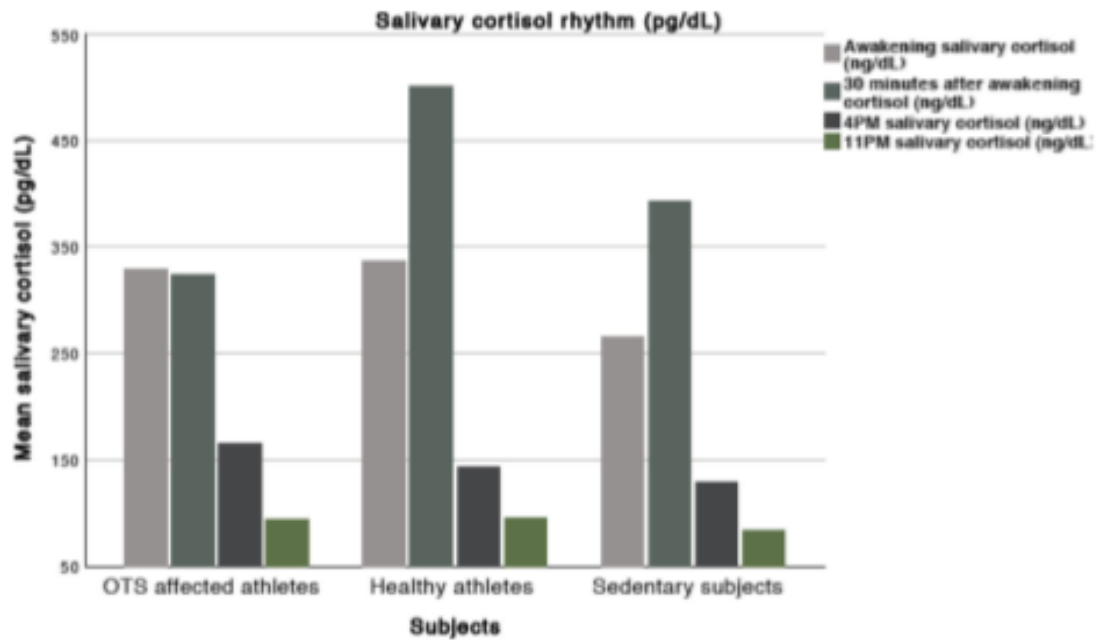
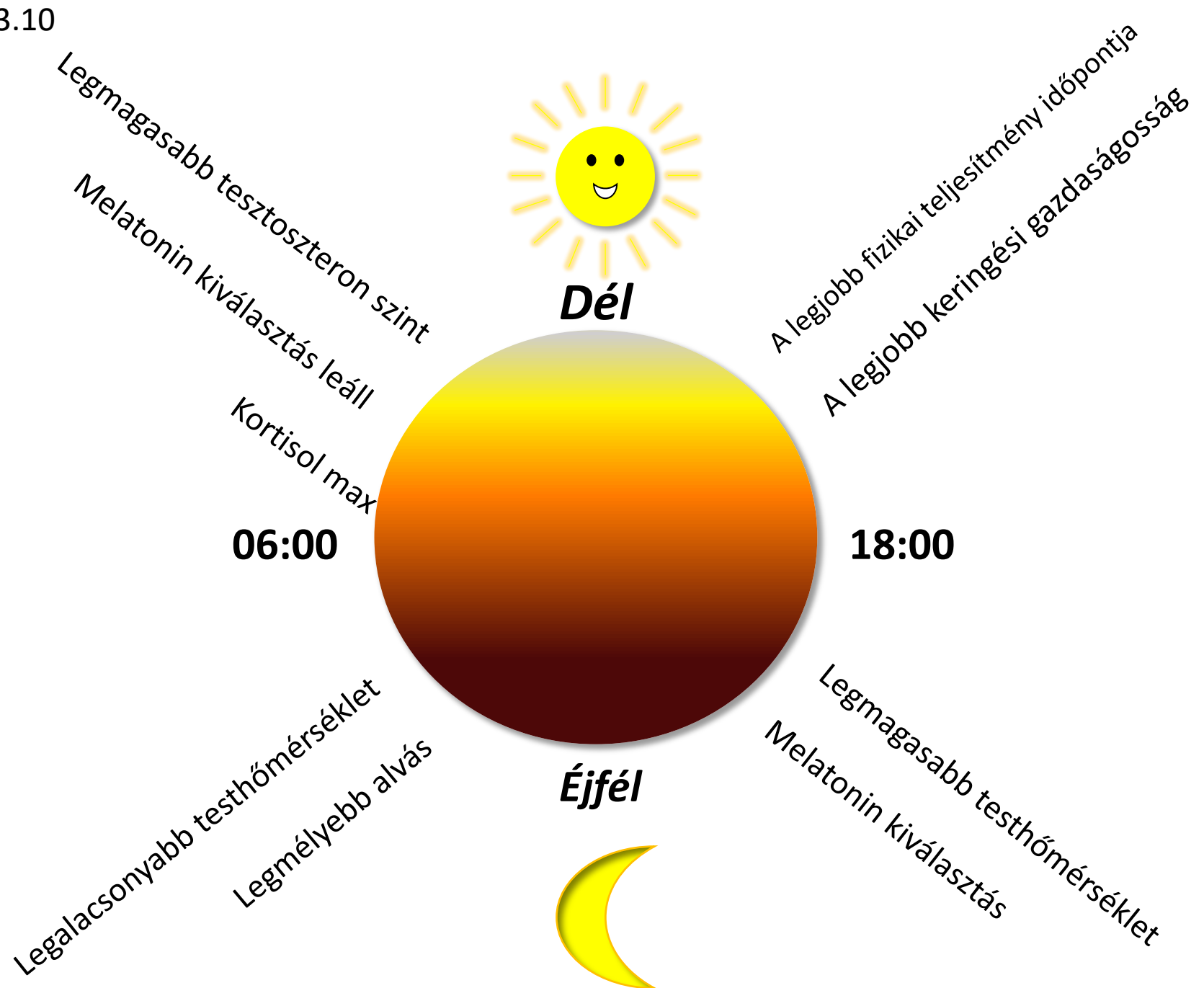
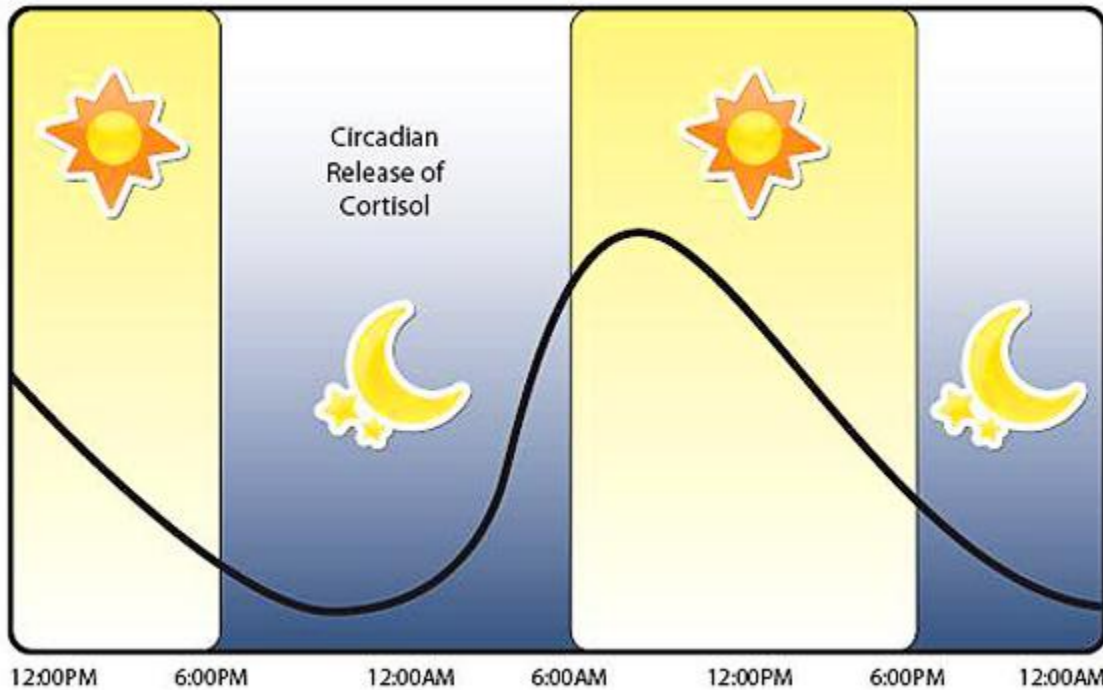


Fig. 4 Salivary cortisol rhythm

Ábra 3.10





Úszás

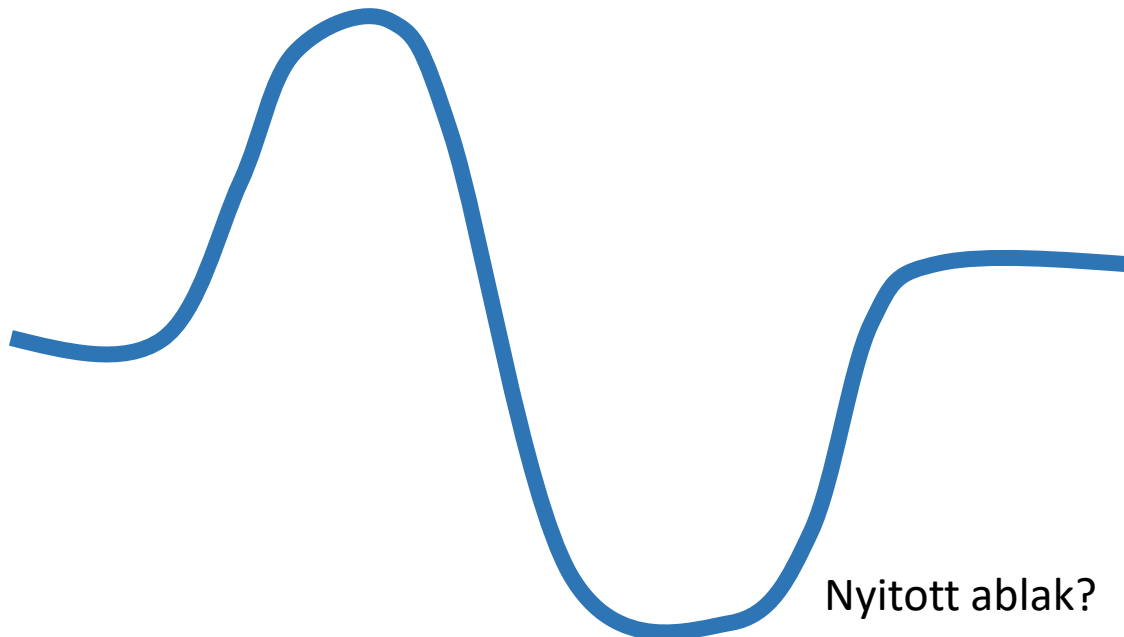
I: 90% VO₂max

D: napi 4 km-ről 9 km-re

Kortizol emelkedés 20 ug/dl-re

(Kirwan et al. 1988)

Kortizol válasz akut fizikai terhelésre



Test Time Line

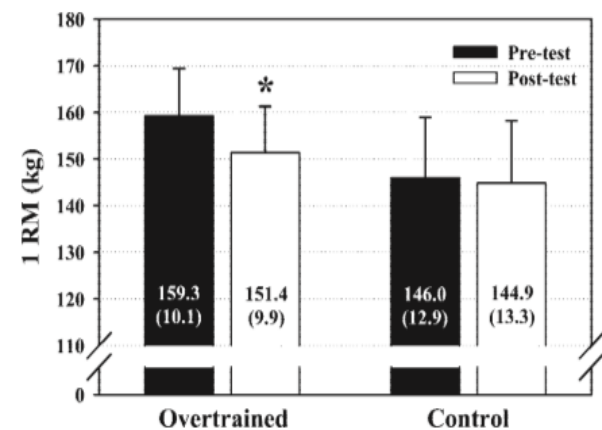
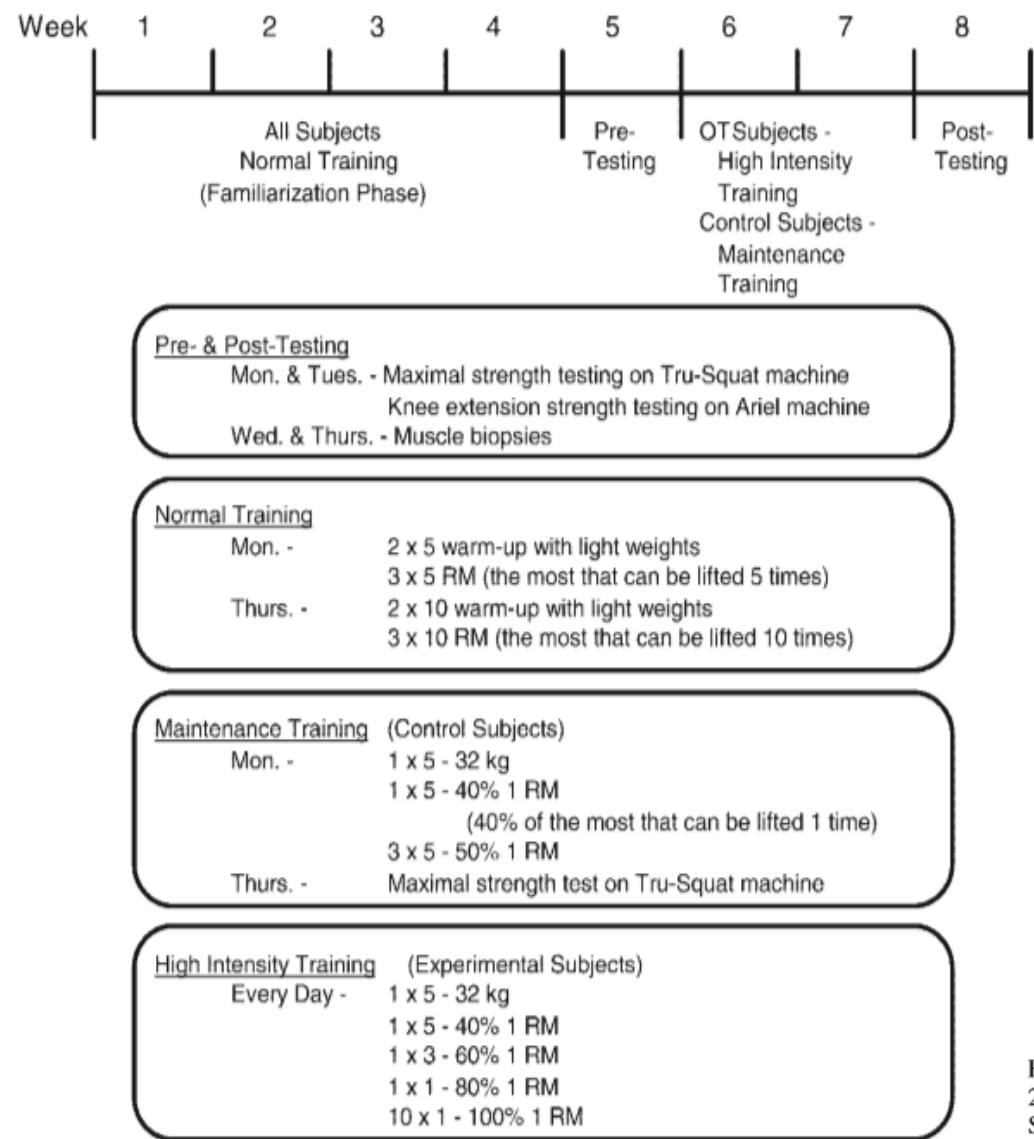


Fig. 3. One-repetition maximum (1 RM) strength responses due to 2 wk of high-intensity resistance exercise overtraining. Values are means $\pm$ SE. *Significantly different from pretest, $P < 0.05$.

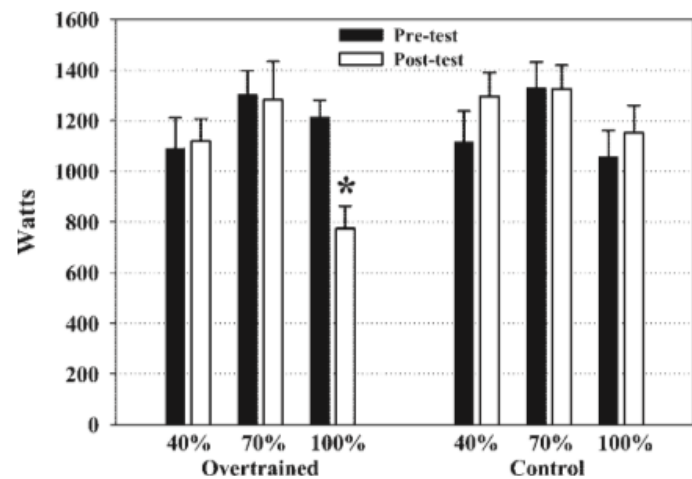


Fig. 4. Mean power responses at different relative intensities (%1 RM) due to 2 wk of high-intensity resistance exercise overtraining. Values are means $\pm$ SE. *Significantly different from pretest, $P < 0.05$.

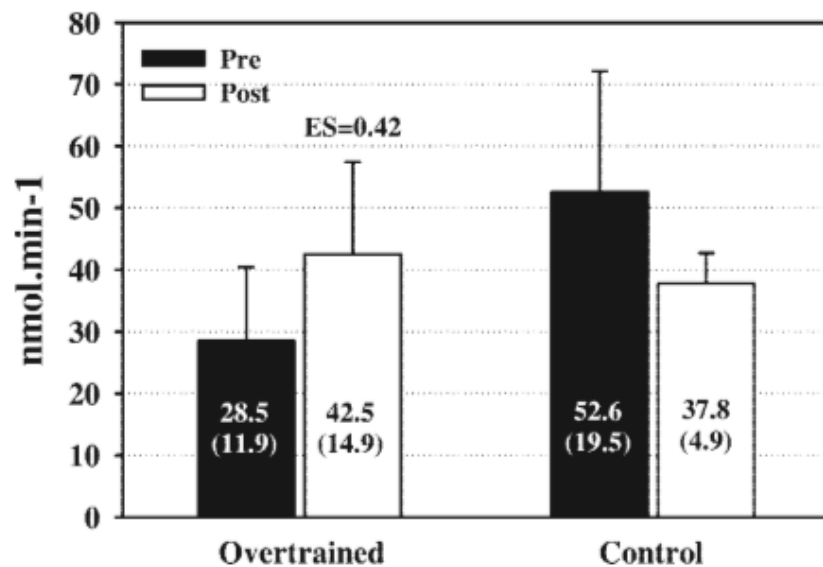


Fig. 6. Nocturnal urinary epinephrine responses indicated basal sympathetic activity. Values are means $\pm$ SE. Responses to 2 wk of high-intensity resistance exercise overtraining did not significantly change ($P > 0.05$), but the effect size (ES) for the OT group was moderate (Cohen's $D = 0.42$).

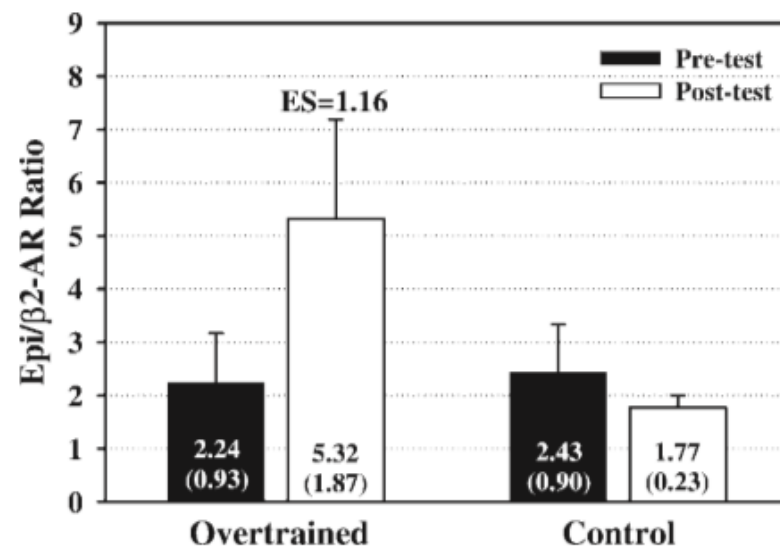


Fig. 7. Ratio of nocturnal urinary epinephrine (Epi) to β_2 -AR densities indicates receptor sensitivity. Values are means $\pm$ SE. Responses to 2 wk of high-intensity resistance exercise overtraining did not significantly change ($P > 0.05$), although effect size for the OT group was extremely large (Cohen's $D = 1.16$).

**Magas intenzitású erőedzés okozta túledzés
eredményeként csökkent a max. erő és teljesítmény.
A szimpatikus hatás nőtt, ellenben a B2-A receptorok
mennyisége és érzékenysége is csökkent, ami
részben magyarázhatja a teljesítmény csökkenést, ami
valószínűleg sejten belül a MAPK jelző rendszeren
keresztül valósult meg.**



Hormonal aspects of overtraining syndrome: a systematic review

Flavio A. Cadegiani and Claudio E. Kater*

Abstract

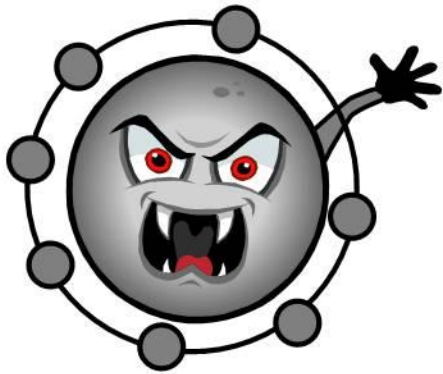
Background: Overtraining syndrome (OTS), functional (FOR) and non-functional overreaching (NFOR) are conditions diagnosed in athletes with decreased performance and fatigue, triggered by metabolic, immune, hormonal and other dysfunctions and resulted from an imbalance between training stress and proper recovery. Despite previous descriptions, there is a lack of a review that discloses all hormonal findings in OTS/FOR/NFOR. The aim of this systematic review is to evaluate whether and which roles hormones play in OTS/FOR/NFOR.

Methods: A systematic search up to June 15th, 2017 was performed in the PUBMED, MEDLINE and Cochrane databases following PRISMA protocol, with the expressions: (1)overtraining, (2)overreaching, (3)overtrained, (4)overreached, or (5)underperformance, and (plus) (a)hormone, (b)hormonal, (c)endocrine, (d)adrenal, (e)cortisol, (f)GH, (g)ACTH, (h)testosterone, (i)IGF-1, (j)TSH, (k)T4, (l)T3, (m)LH, (n)FSH, (o)prolactin, (p) IGFBP-3 and related articles.

Results: A total of 38 studies were selected. Basal levels of hormones were mostly normal in athletes with OTS/FOR/NFOR compared with healthy athletes. Distinctly, stimulation tests, mainly performed in maximal exercise conditions, showed blunted GH and ACTH responses in OTS/FOR/NFOR athletes, whereas cortisol and plasma catecholamines showed conflicting findings and the other hormones responded normally.

Conclusion: Basal hormone levels are not good predictor but blunted ACTH and GH responses to stimulation tests may be good predictors of OTS/FOR/NFOR.

Keywords: Overtraining, Overreaching, Adrenal, Cortisol, Hormone, Sports endocrinology



Free Radical

Defenseless and Damaged



<http://dchealthybytes.com>

Neurochem Int. 2005 Jun;46(8):635-40. Epub 2005 Apr 9.

The effects of moderate-, strenuous- and over-training on oxidative stress markers, DNA repair, and memory, in rat brain.

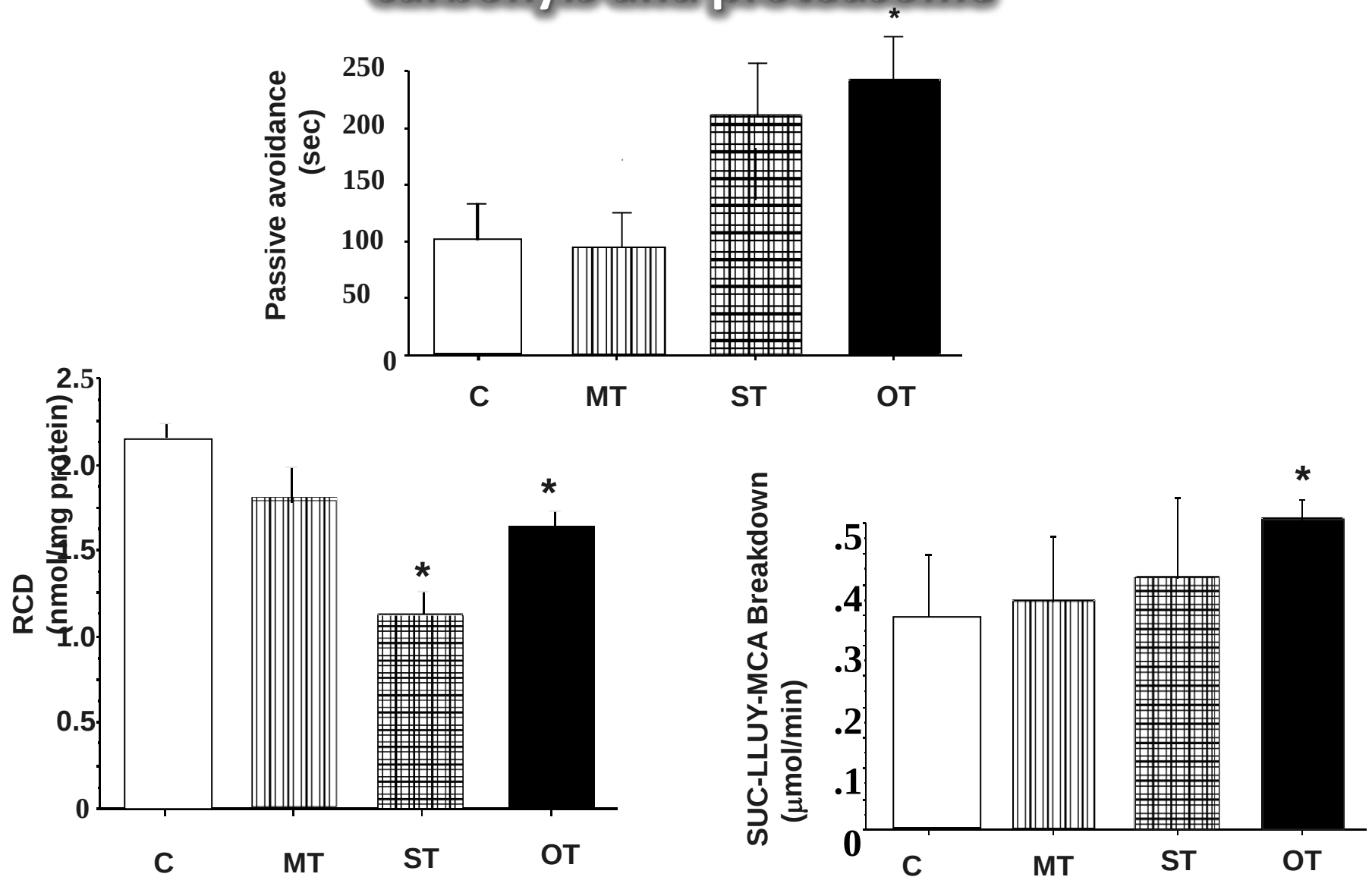
Ogonovszky H¹, Berkes I, Kumagai S, Kaneko T, Tahara S, Goto S, Radák Z.

Author information

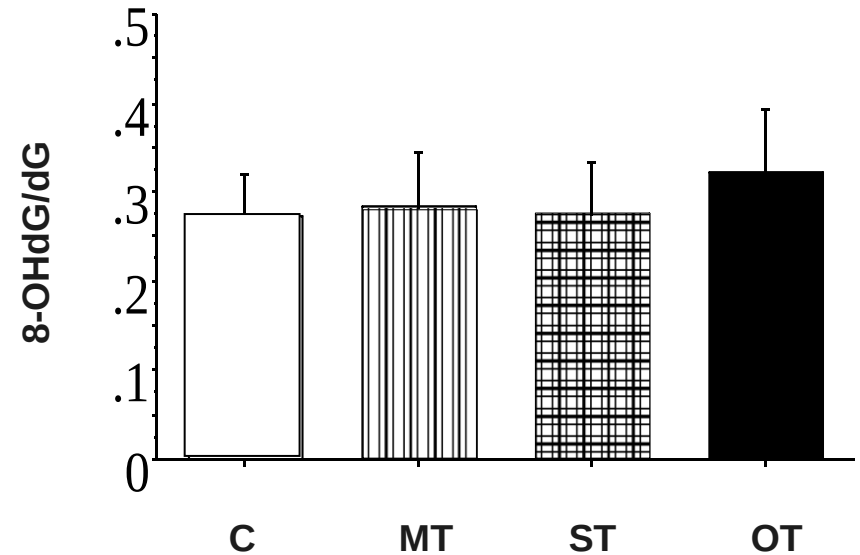
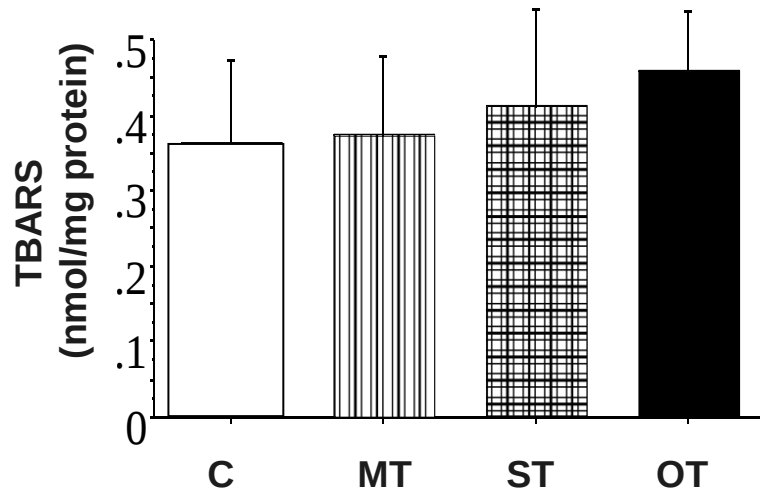
Abstract

We have tested the hypothesis that training with moderate- (MT), strenuous- (ST), or over- (OT) load can cause alterations in memory, lipid peroxidation, protein oxidation, DNA damage, activity of 8-oxoG-DNA glycosylase (OGG1) and brain-derived neurotrophic factor (BDNF), in rat brain. Rat memory was assessed by a passive avoidance test and the ST and OT group demonstrated improved memory. The content of BDNF was increased only in the OT group. The oxidative damage of lipids and DNA, as measured by thiobarbituric acid reactive substances (TBARS), and 8-hydroxydeoxyguanosine (8-OHdG), did not change significantly with exercise. Similarly, the activity of DNA repair enzyme, 8-oxoguanine DNA glycosylase (OGG1), was not altered with exercise training. On the other hand, the content of reactive carbonyl derivatives (RCDs) decreased in all groups and the decrease reached significance levels in the ST and OT groups. The activity of the proteasome complex increased in the brain of OT. The findings of this study imply that over-training does not induce oxidative stress in the brain and does not cause loss of memory. The improved memory was associated with enhanced BDNF content.

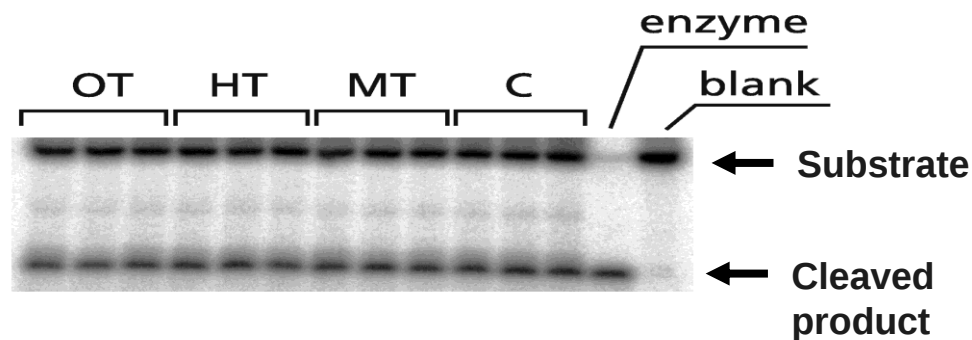
The effects of different training loads on brain function, carbonyls and proteasome



The effect of different training load on lipid and DNA damage and repair



OT brain fpg



C - control
MT - moderate training
HT - heavy training
OT - over training

The effects of moderate, strenuous, and overtraining on oxidative stress markers and DNA repair in rat liver.

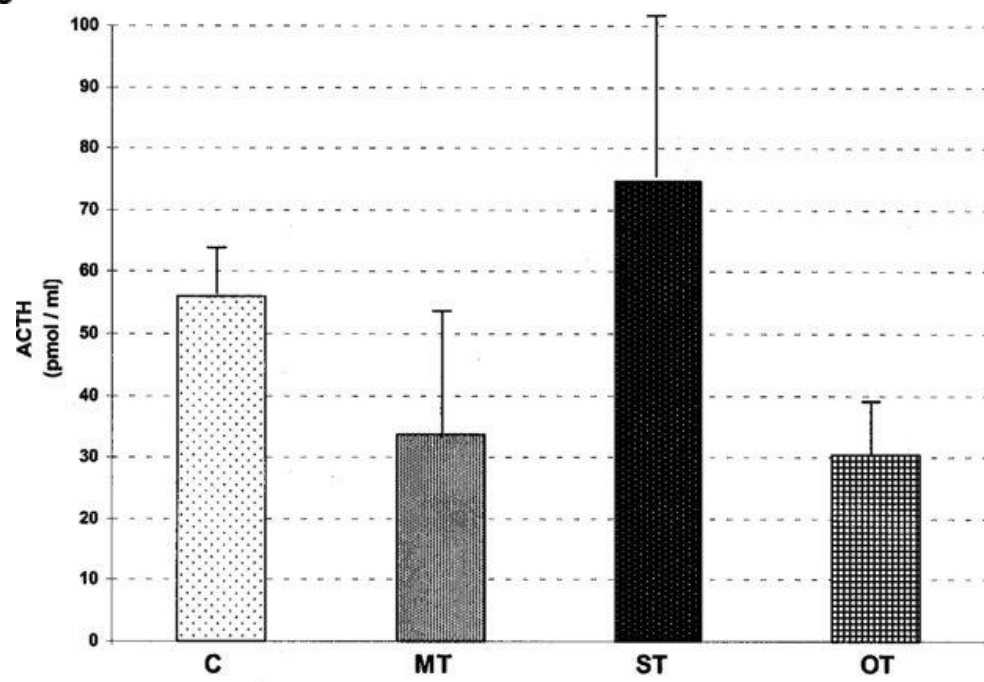
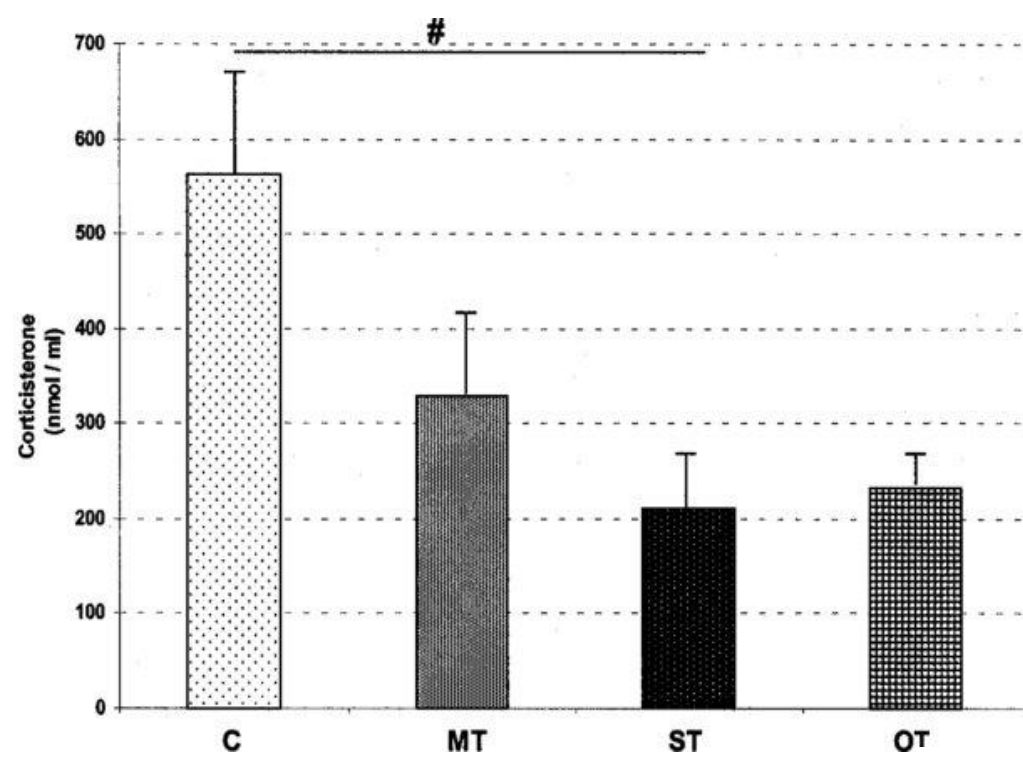
Ogonovszky H¹, Sasvári M, Dosek A, Berkes I, Kaneko T, Tahara S, Nakamoto H, Goto S, Radák Z.

Author information

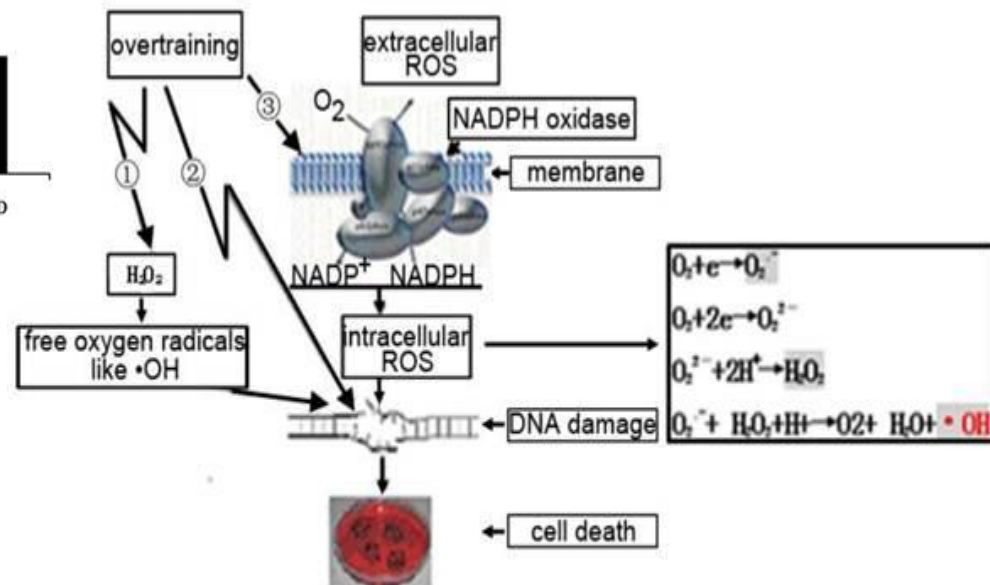
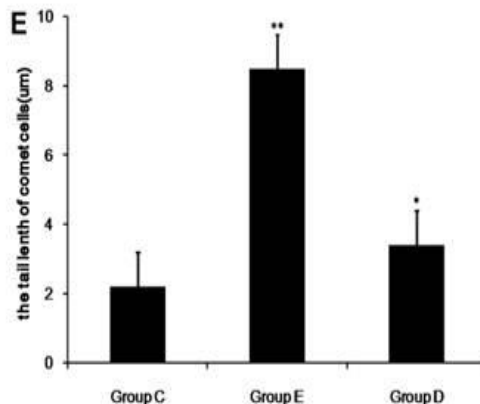
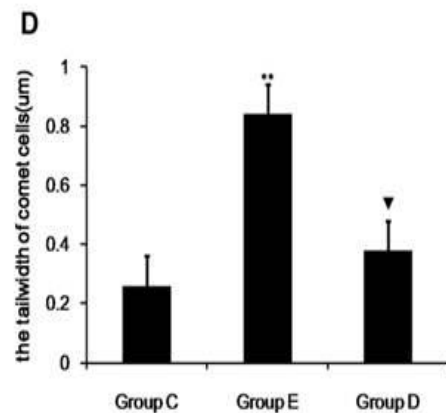
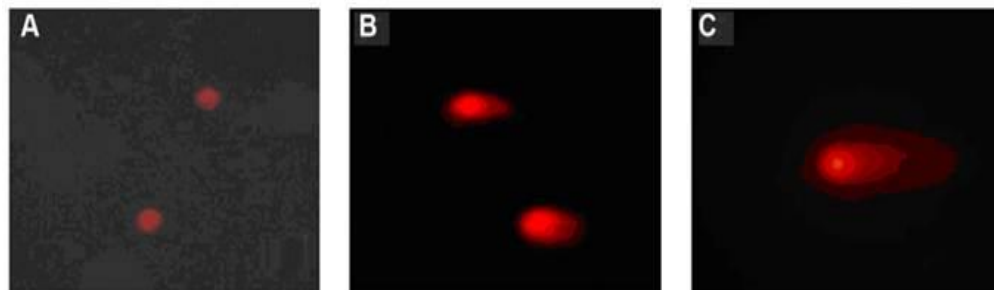
1 Institute of Sport Science, Faculty of Physical Education and Sport Science, Semmelweis Univ., 1123 Budapest, Alkotas u. 44, Hungary.

Abstract

Physical exercise above a certain load has been suggested as being a cause of oxidative stress. We have tested whether training with moderate (MT), strenuous (ST), or over (OT) load can cause alterations in the activities of antioxidant enzymes, lipid peroxidation, protein oxidation, DNA damage, or activity of 8-oxoG-DNA glycosylase (OGG1) in rat liver. The levels of corticosterone decreased in all exercising groups but the differences were not significant. Adrenocorticotrophin hormone (ACTH) levels decreased, not significantly, in MT and OT compared to C. Activity levels of antioxidant enzymes did not change significantly in the liver. The levels of reactive carbonyl derivative (RCD) content decreased in the liver of exercising animals, and the differences reached significance between control and moderately trained groups. The changes in the levels of lipid peroxidation (LIPOX) were not significant, but were lower in the exercised groups. The 8-hydroxydeoxyguanosine (8-OHdG) levels increased in the OT group, and the activity of OGG1 measured from crude cell extracts tended to increase in MT and ST. The findings of this study imply that overtraining induces oxidative damage to nuclear DNA, but not to liver lipids and proteins.



Túledzés és szabadgyökök





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Nagy nagy Terhelés

GH

Alvás minőség

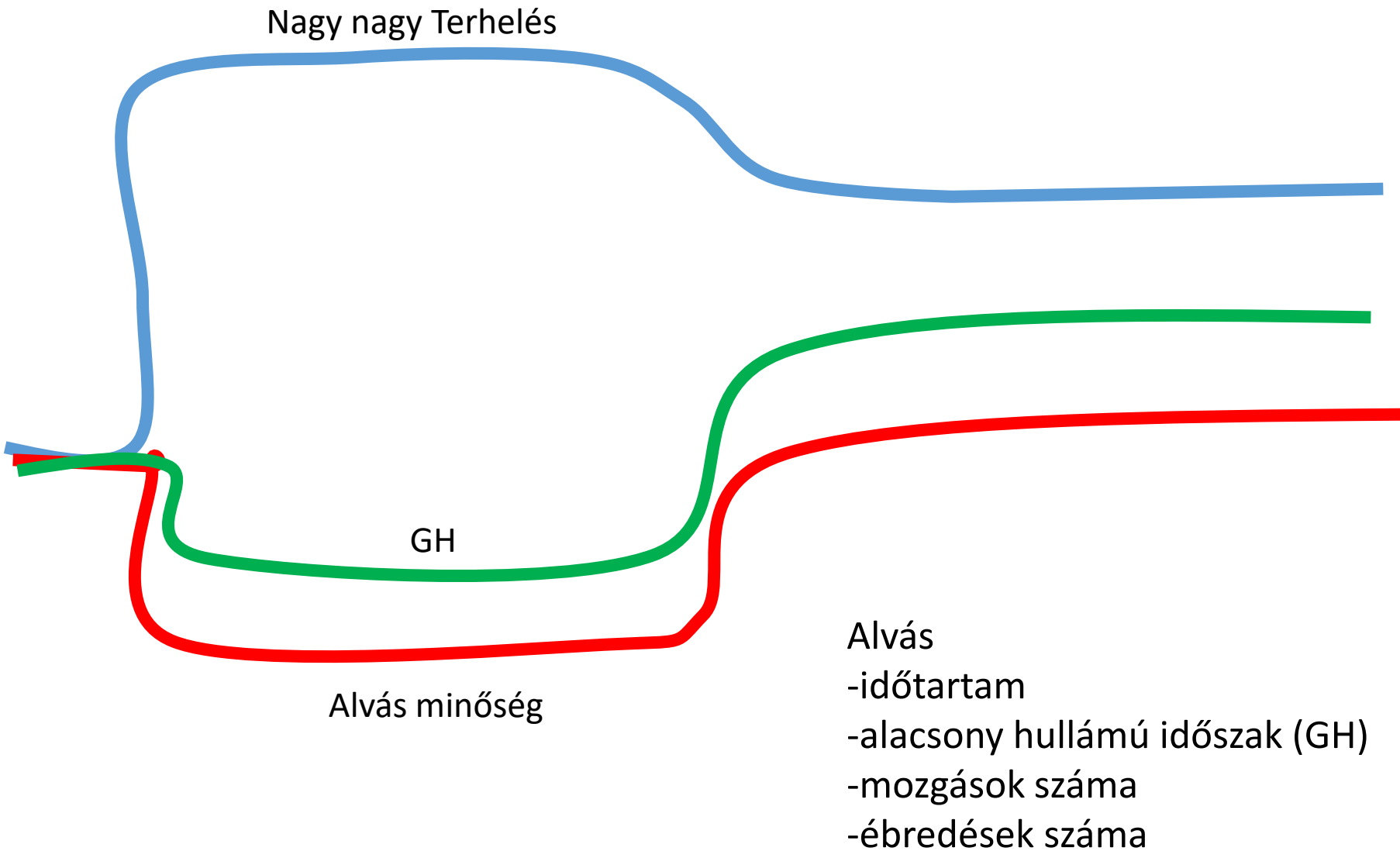
Alvás

-időtartam

-alacsony hullámú időszak (GH)

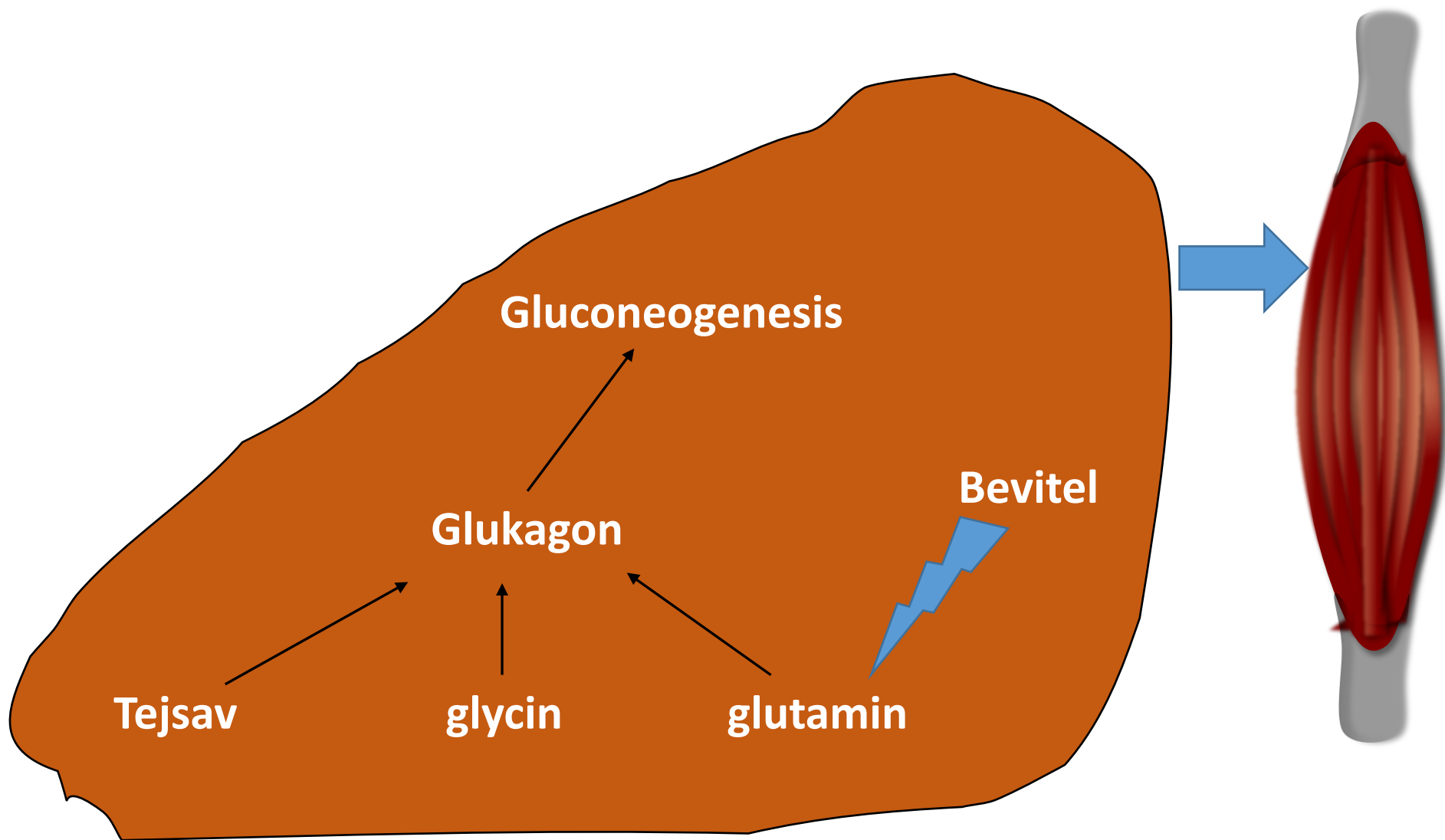
-mozgások száma

-ébredések száma



Megelőzés

- Folyamatos energia, fehérje és folyadék bevitellel el kell kerülni a negatív energia egyensúlyt és dehidratációt.
- 12 hetes kemény edzés után azok, akiknek a teljesítménye több mint 8%-kal csökkent, szinte kivétel nélkül negatív energia egyensúlyban voltak az edzés periodus alatt, míg azok akiknek a teljesítménye több mint 8%-kal nőtt, csak időlegesen max. 2 hétig voltak negatív energia egyensúlyban (Vanheest et al. 2014).
- Alacsony energia egyensúly serkenti a gyulladásos citokinek termelődését, ami az egyik oka lehet a túledzésnek.
- Dokumentáltan kedvező hatása van az alacsony szénhidrát (glikogén) szinttel végzett edzésnek (Bartlett et al 2013, Van Proeyen et al. 2011), de ez túledzést okozhat a negatív energia egyensúly miatt.



A túledzés regenerációja

- **Túledzés okának a megfejtése, ha lehet kijavítása**
- **Az edzés jellegének sarkalatos megváltoztatása**
- **Környezetváltozás**
- **Táplálkozási intervenció**
- **Masszázs, fürdő, regenerációs intervenciók**
- **Élményterápia**