

THE JUNIOR ELITE ATHLETE: Physiological Characteristics and Training Considerations



IAAF World Youth Coaches Conference

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United States Olympic Committee
Chair, ACSM Olympic/Paralympic Sports Medicine & Sport Science Committee

Thank you!

Kamsa hamnida (Korean)

Danke schön (German)

Ameseginalhu (Ethiopia)

Спасибо (Russian)

Arigato (Japanese)

Asante sana (Swahili/Kenyan)

Meitaki Ma'ata (Maori)

Paldies (Latvian)

Fa'afetai (Samoan)

Dziękuję (Polish)

Terima kasih (Malaysian + Indonesian)

Gracias (Spanish)

धन्यवाद (Hindi)

Dank u wel (Dutch)

Merci beaucoup (French)

شكراً جزيلاً (Arabic)

Kiitoksia (Finnish)

ευχαριστώ (Greek)

Благодаря (Bulgarian)

Grazie (Italian)

Obrigado (Portuguese/Brazilian)

Cheers (Australian / New Zealand)

Mahalo (Hawaiian)

Xie xie (Chinese)

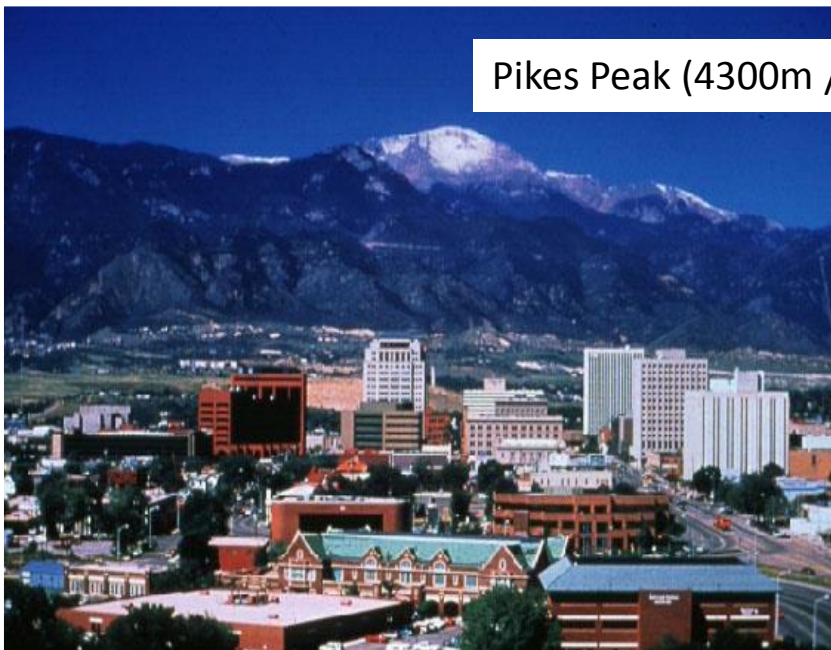




US Olympic Training Center

Colorado Springs

Pikes Peak (4300m / 14,115ft)



Colorado Springs (1885m / 6180ft)





Randall L. Wilber, PhD, FACSM

US Olympic Committee



TRACK/CC COACH (1976-1993)

- Pennsylvania
- Wisconsin
- Florida



SPORT PHYSIOLOGIST (1993-present)

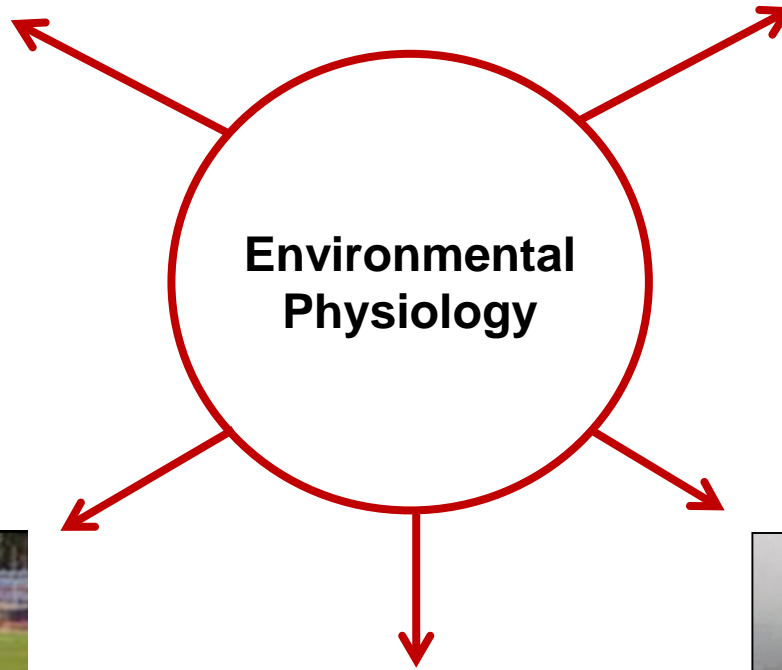
US Olympic Committee

- Salt Lake City 2002
- Athens 2004
- Torino 2006
- Beijing 2008
- Vancouver 2010
- London 2012
- Sochi 2014



US Olympic Training Center Colorado Springs





**Environmental
Physiology**

THE JUNIOR ELITE ATHLETE:

Physiological Characteristics and Training Considerations

- Introduction / Physiology 101
- From Childhood to Adolescence to Adulthood
- Increased Risk of Injury
- Training Considerations
- Overtraining
- Summary and Q/A



THE JUNIOR ELITE ATHLETE:

Physiological Characteristics and Training Considerations

➔ ■ Introduction / Physiology 101 →

Bone Structure & Metabolism
Endocrine/Hormone Response
Skeletal Muscle Fiber Types
Iron Metabolism

■ From Childhood to Adolescence to Adulthood

■ Increased Risk of Injury

■ Training Considerations

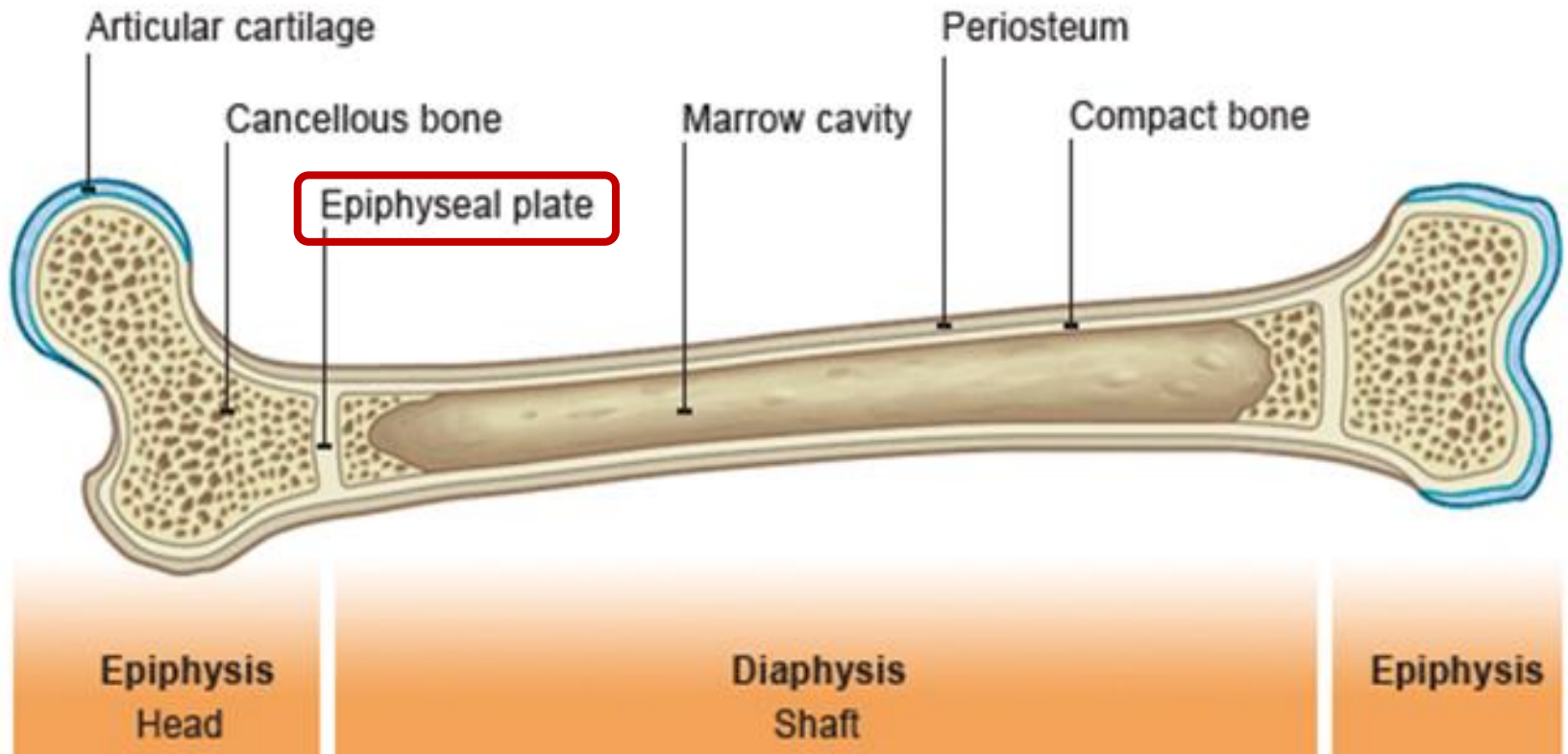
■ Overtraining

■ Summary and Q/A



PHYSIOLOGY 101

Bone Structure and Metabolism



PHYSIOLOGY 101

Endocrine/Hormone Response



PHYSIOLOGY 101

Endocrine/Hormone Response



REPRODUCTION

Follicle-stimulating h. (FSH)
Luteinizing h. (LH)
Prolactin

Pituitary gland

Pineal gland

GROWTH / DEVELOPMENT

Growth h. (GH)

Thyroid gland

Parathyroid glands

BONE DEVELOPMENT

Parathyroid h. (PTH)

GROWTH / DEVELOPMENT

ENERGY METABOLISM

Triiodothyronine (T3)
Thyroxine (T4)

BONE DEVELOPMENT

Calcitonin

Adrenal gland

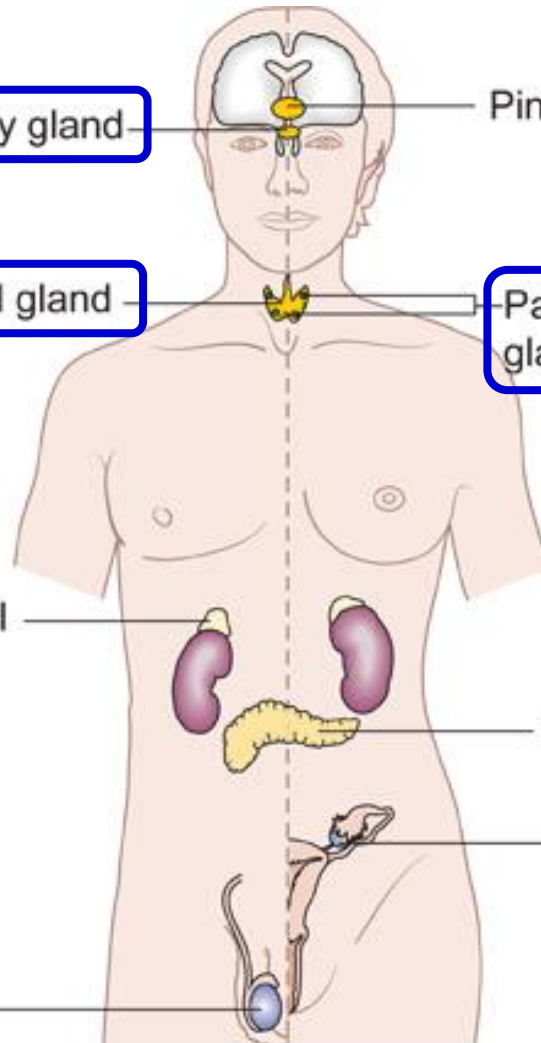
Pancreas

Ovary

REPRODUCTION

Estrogen
Progesterone

Testis



PHYSIOLOGY 101

Endocrine/Hormone Response



REPRODUCTION

Follicle-stimulating h. (FSH)
Luteinizing h. (LH)

Pituitary gland

Pineal gland

GROWTH / DEVELOPMENT

Growth h. (GH)

Thyroid gland

Parathyroid glands

BONE DEVELOPMENT

Parathyroid h. (PTH)

GROWTH / DEVELOPMENT

ENERGY METABOLISM

Triiodothyronine (T3)
Thyroxine (T4)

BONE DEVELOPMENT

Calcitonin

Adrenal gland

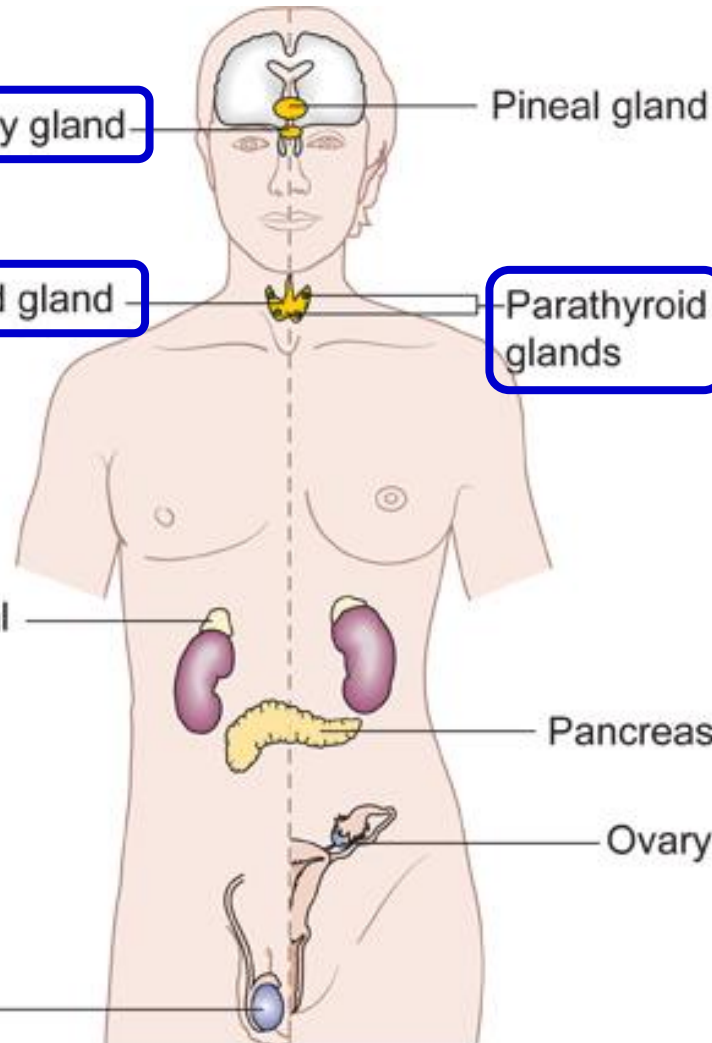
Pancreas

Ovary

REPRODUCTION

Testosterone

Testis



PHYSIOLOGY 101

Skeletal Muscle Fiber Types



Type I

“Slow” twitch
Bright red in color
Fatigue slowly

Oxidative Phosphorylation

3000 m SC
5000 m
10,000 m
Marathon

PHYSIOLOGY 101

Skeletal Muscle Fiber Types



Type IIa

“Fast/slow” twitch hybrid
Pale red in color
Fatigue slowly

Glycolysis (lactate)
Oxidative Phosphorylation

400 m H
800 m
1000 m
1500 m

PHYSIOLOGY 101

Skeletal Muscle Fiber Types



Type IIX

“Fast” twitch
White in color
Fatigue quickly

ATP / CP
Glycolysis (lactate)

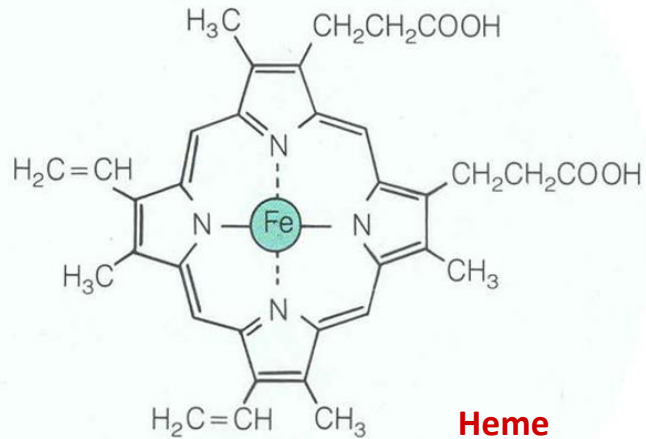
60 m
100 m
100 m H / 110 m H
200 m
400 m
LJ, TJ, PV
SP, D, J, HT

PHYSIOLOGY 101

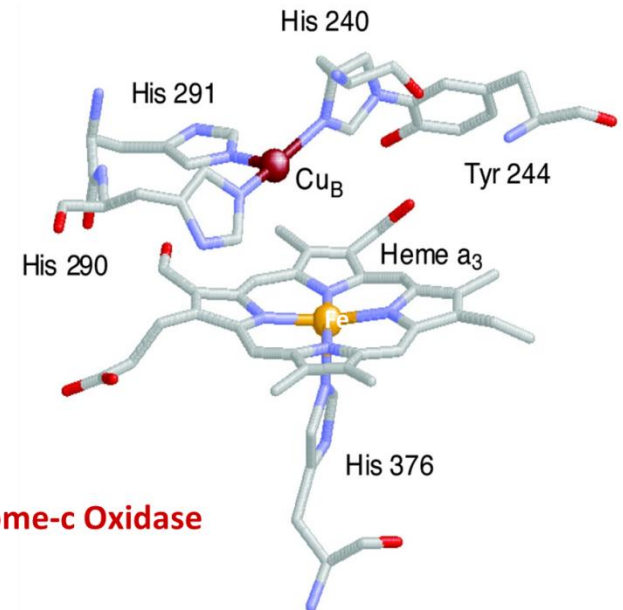
Iron Metabolism

Fe atom structure

The Atomium
Brussels



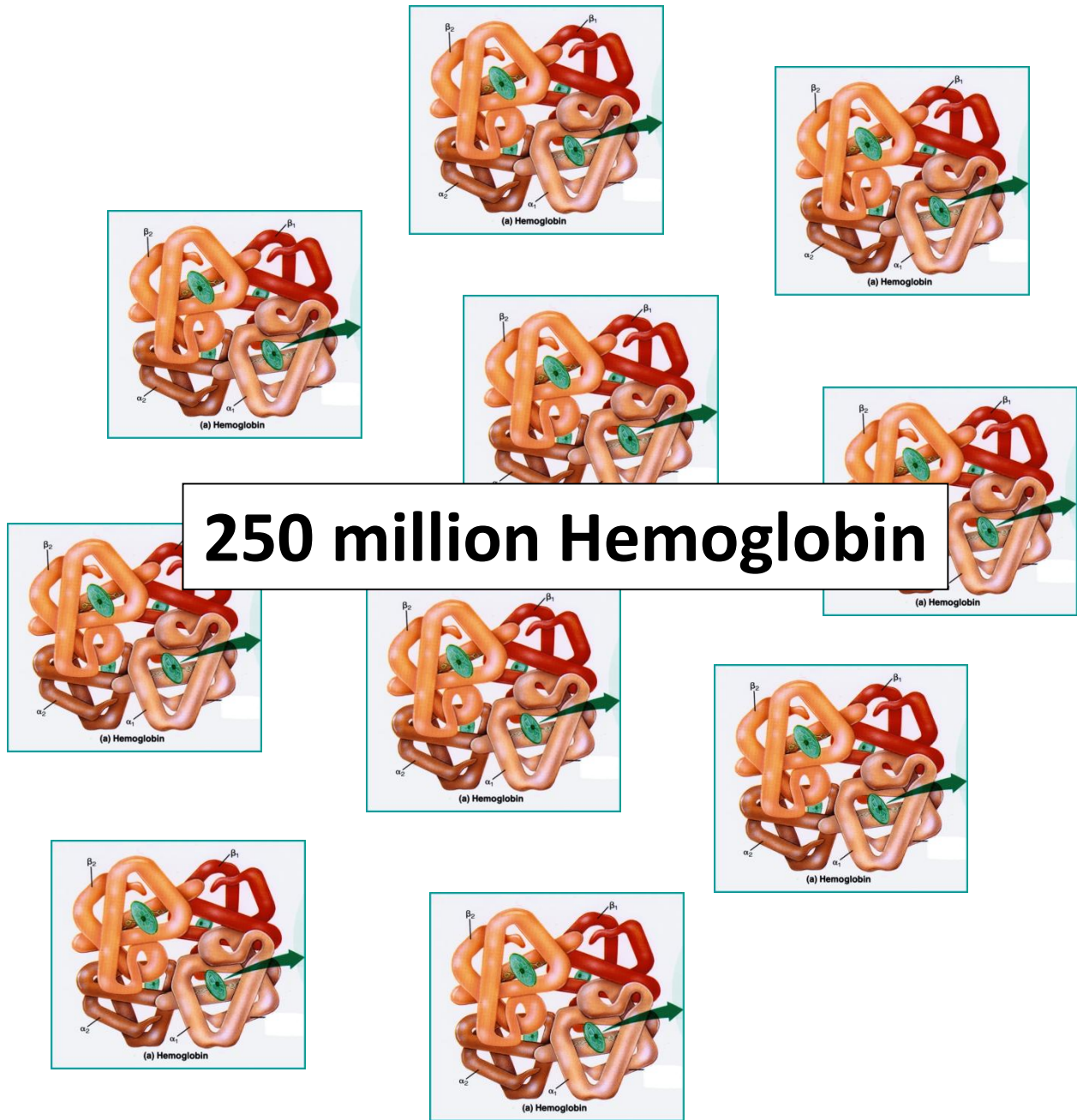
Heme

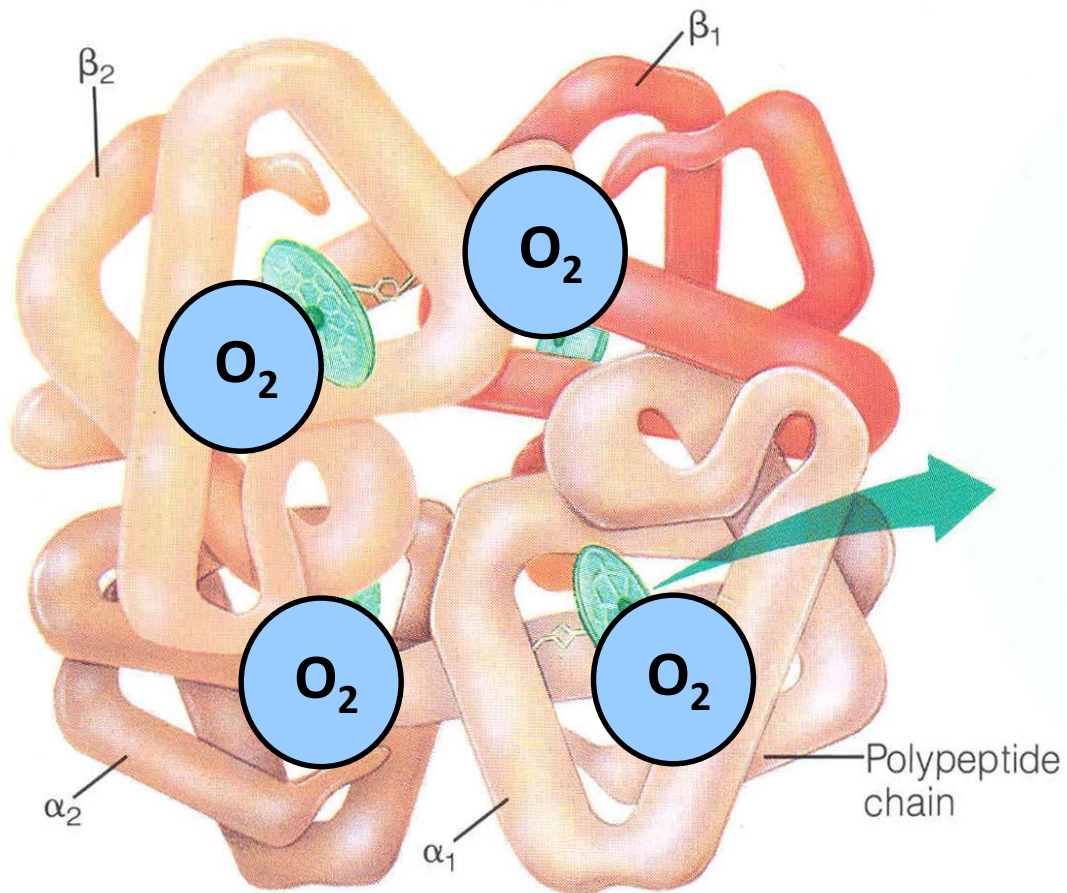


Cytochrome-c Oxidase

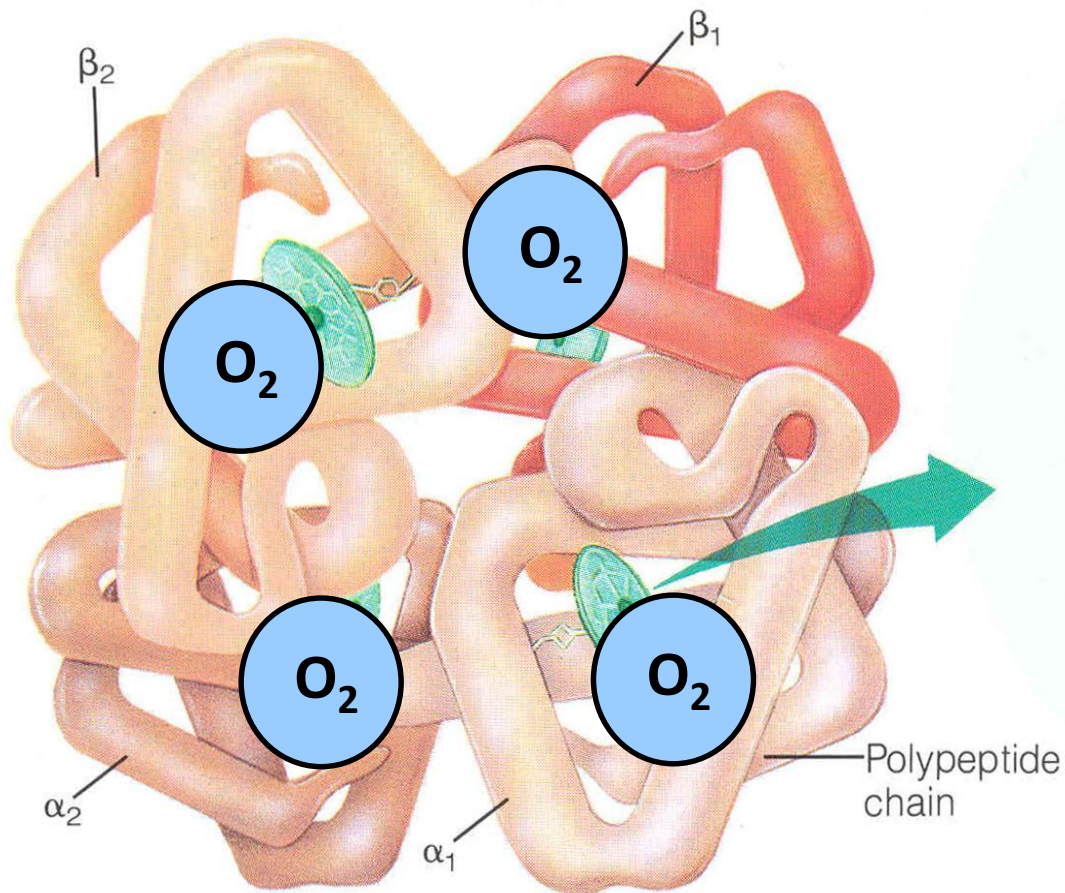


RBC

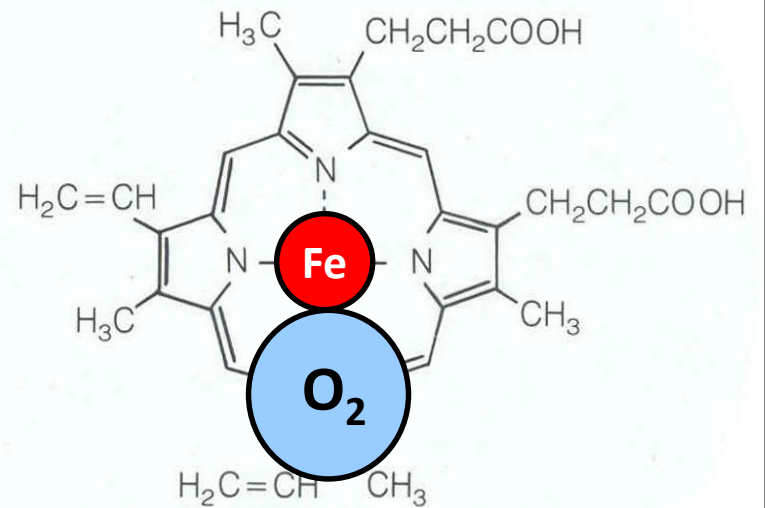




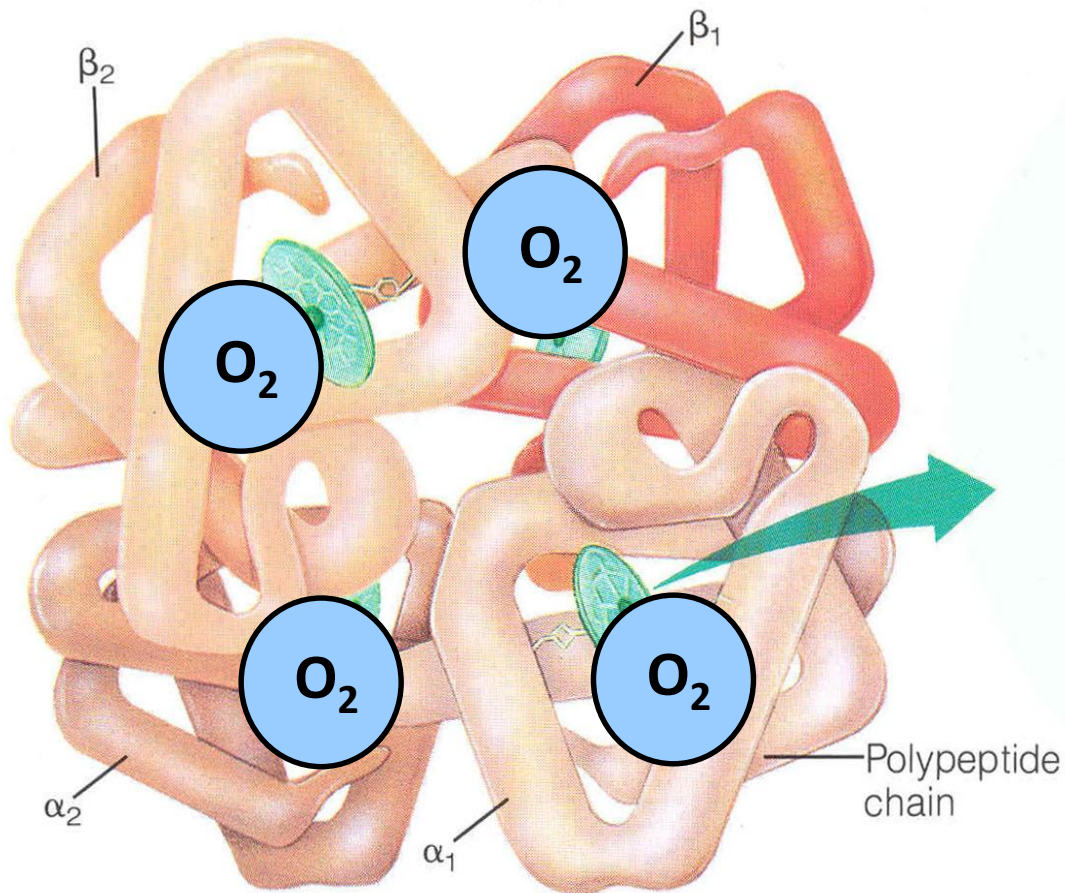
(a) Hemoglobin



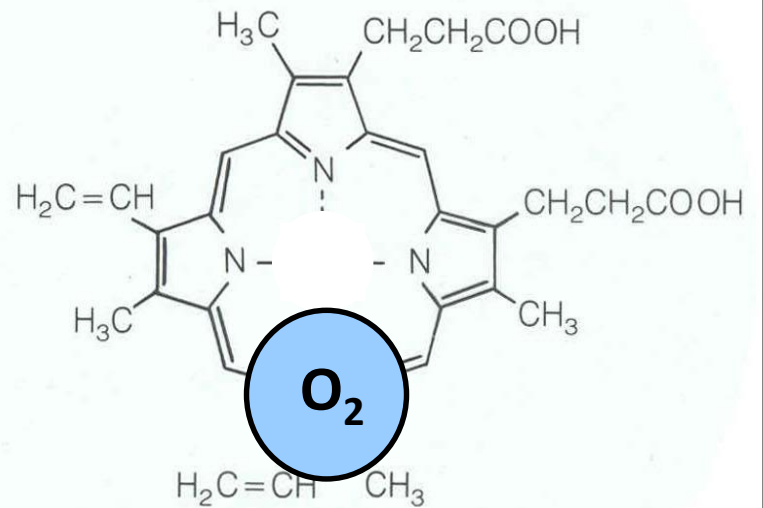
(a) Hemoglobin



(b) Iron-containing heme group



(a) Hemoglobin



(b) Iron-containing heme group

Stage 1: IRON-DEPLETION

COLLECTED: NOT GIVEN

REPORTED: 07/17/2004 PAGE: 1

REQUESTS	RESULTS	UNITS	REFERENCE RANGES	LOC
FERRITIN, SERUM	7 L	NG/ML	10-154	DE
IRON & TIBC, SERUM				
IRON, SERUM	63	MCG/DL	35-175	DE
IRON BINDING CAPACITY	386	MCG/DL	250-400	DE
% SATURATION	16	%	15-50	DE
CBC (INC. DIFF & PLT)				
WBC	6.2	THOUS/MCL	3.8-10.8	DE
RBC	4.50	MILL/MCL	3.80-5.10	DE
HEMOGLOBIN	13.5	GM/DL	11.7-15.5	DE
HEMATOCRIT	40.7	%	35.0-45.0	DE
MCV	90.4	FL	80.0-100.0	DE
MCH	30.1	PG	27.0-33.0	DE
MCHC	33.3	GM/DL	32.0-36.0	DE
RDW	14.9	%	11.0-15.0	DE
PLATELET COUNT	288	THOUS/MCL	140-400	DE
MPV	8.0	FL	7.5-11.5	DE
ABSOLUTE NEUTROPHILS	2790	CELLS/MCL	1500-7800	DE
ABSOLUTE LYMPHOCYTES	2877	CELLS/MCL	850-3900	DE
ABSOLUTE MONOCYTES	434	CELLS/MCL	200-950	DE
ABSOLUTE EOSINOPHILS	87	CELLS/MCL	0-500	DE
ABSOLUTE BASOPHILS	12	CELLS/MCL	0-200	DE
NEUTROPHILS	45.0	%	45-71	DE
LYMPHOCYTES	46.4 H	%	15-45	DE
MONOCYTES	7.0	%	0-15	DE
EOSINOPHILS	1.4	%	0-5	DE
BASOPHILS	0.2	%	0-3	DE



USA CYCLING
Female
22 yr



Stage 2: IRON-DEFICIENT NON-ANEMIA (IDNA)

REQUESTS	RESULTS	UNITS	REFERENCE RANGES
FERRITIN, SERUM	9	L	NG/ML 10-143
IRON & TIBC, SERUM			
IRON, SERUM	32	L	MCG/DL 35-175
IRON BINDING CAPACITY	444	H	MCG/DL 250-400
% SATURATION	7	L	% 15-50
CBC (INC. DIFF & PLT)			
WBC	7.7		THOUS/MCL 3.8-10.8
RBC	4.64		MILL/MCL 3.80-5.10
HEMOGLOBIN	14.1		GM/DL 11.9-15.9
HEMATOCRIT	42.5		% 35.0-49.0
MCV	91.5		FL 80.0-100.0
MCH	30.5		PG 27.0-33.0
MCHC	33.3		GM/DL 32.0-36.0
RDW	15.8	H	% 11.0-15.0
PLATELET COUNT	270		THOUS/MCL 140-400
MPV	7.9		FL 7.5-11.5
ABSOLUTE NEUTROPHILS	4936		CELLS/MCL 1500-7800
ABSOLUTE LYMPHOCYTES	1987		CELLS/MCL 850-3900
ABSOLUTE MONOCYTES	616		CELLS/MCL 200-950
ABSOLUTE EOSINOPHILS	131		CELLS/MCL 0-500
ABSOLUTE BASOPHILS	31		CELLS/MCL 0-200
NEUTROPHILS	64.1		% 45-71
LYMPHOCYTES	25.8		% 15-45
MONOCYTES	8.0		% 0-13
EOSINOPHILS	1.7		% 0-5
BASOPHILS	0.4		% 0-3



USA TRIATHLON
Female
18 yr



IRON SUPPLEMENTATION



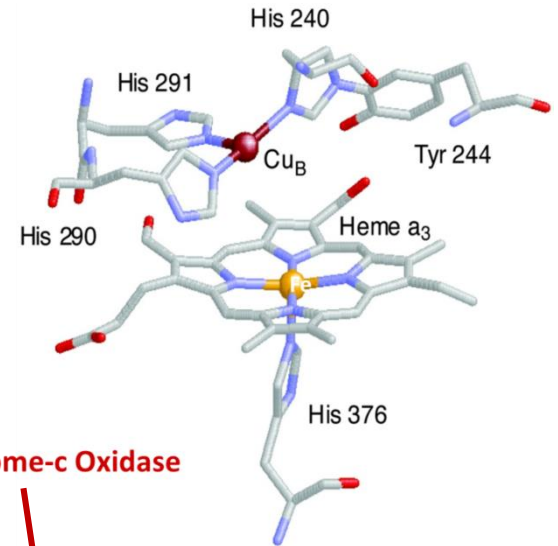
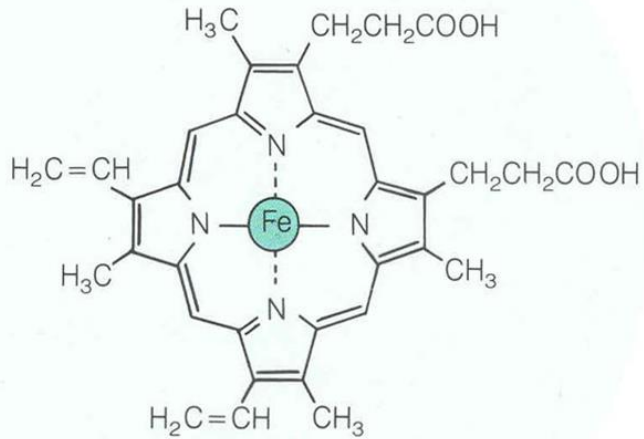
Ferrous sulfate

If serum Ferritin is low:

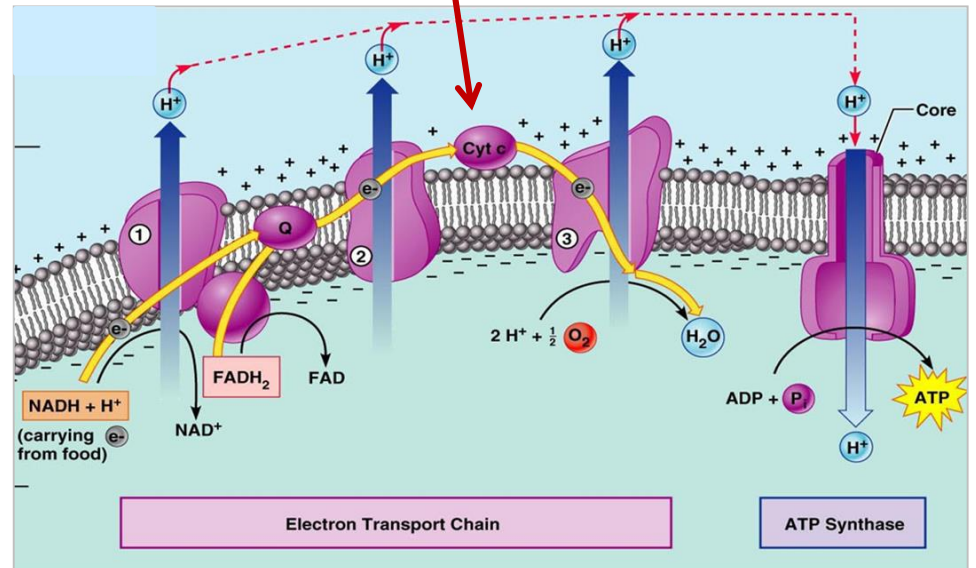
- Attention to “heme Fe” in diet.
- Moderate Fe supplementation
 - 120-130 mg “elemental Fe” divided into 2 doses
 - taken with Vitamin C
 - taken 30 min before or 60 min after meals to increase absorption and decrease GI distress
 - taken daily

IRON SUPPLEMENTATION

Hemoglobin



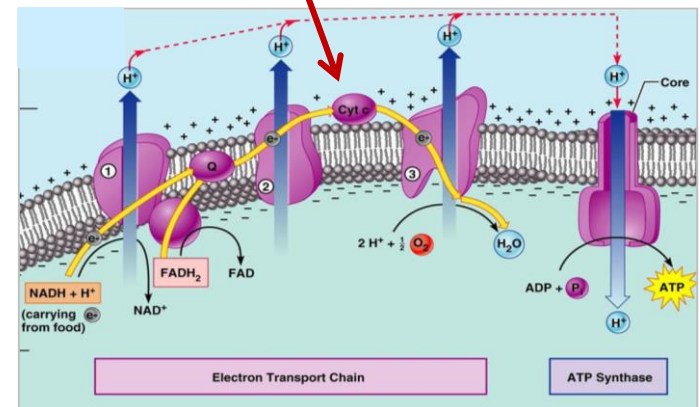
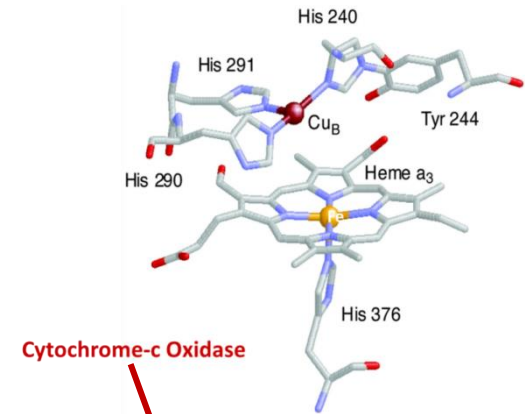
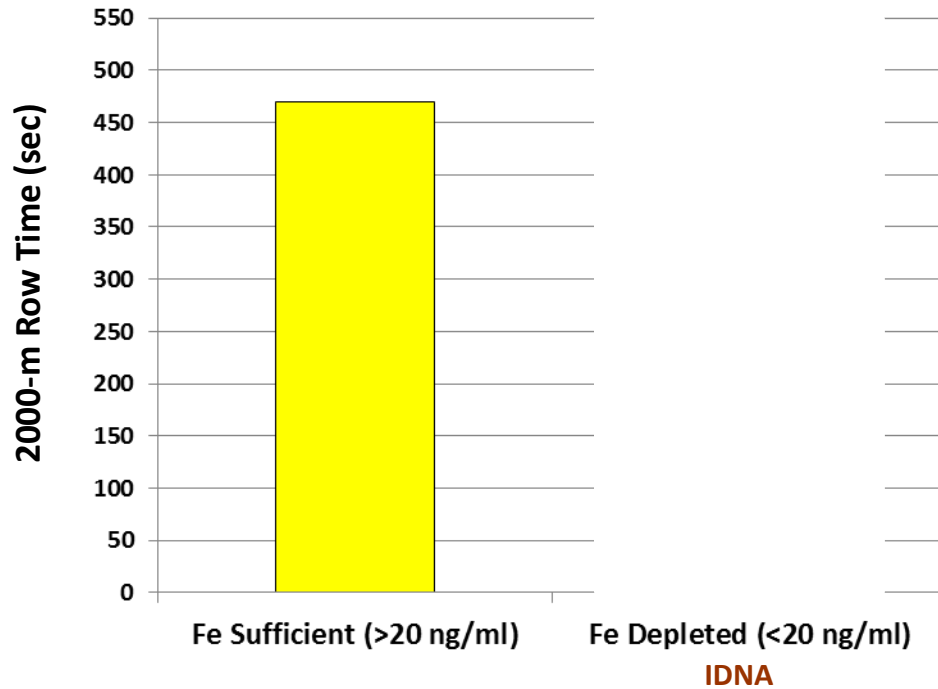
Cytochrome-c Oxidase



Impact of Iron Depletion Without Anemia on Performance in Trained Endurance Athletes at the Beginning of a Training Season: A Study of Female Collegiate Rowers

Diane M. DellaValle and Jere D. Haas

International Journal of Sport Nutrition and Exercise Metabolism, 2011, 21, 501-506



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From CHILDHOOD to ADOLESCENCE to ADULTHOOD



Pre-Puberty Age 6-10

A slow and balanced development when the function of some organs becomes more efficient.



Puberty Age 11-13

Fast growth and development in height, weight, and the efficiency of some organs. Sexual maturation with change in interests and behaviors.



Post-Puberty / Adolescence Age 14-18

A slow, balanced and proportional development with functional maturation.



Young Adulthood Age 19-25

Full growth and maturation of physiological and physical traits. Athletic and psychological potentials are maximized.

From CHILDHOOD to ADOLESCENCE to ADULTHOOD



Pre-Puberty
Age 7-11

A slow and balanced development when the function of some organs becomes more efficient.



Puberty
Age 12-14

Fast growth and development in height, weight, and the efficiency of some organs. Sexual maturation with change in interests and behaviors.



Post-Puberty / Adolescence
Age 15-18

A slow, balanced and proportional development with functional maturation.

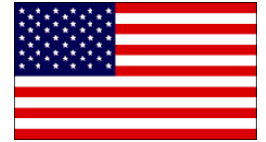


Young Adulthood
Age 19-25

Full growth and maturation of physiological and physical traits. Athletic and psychological potentials are maximized.

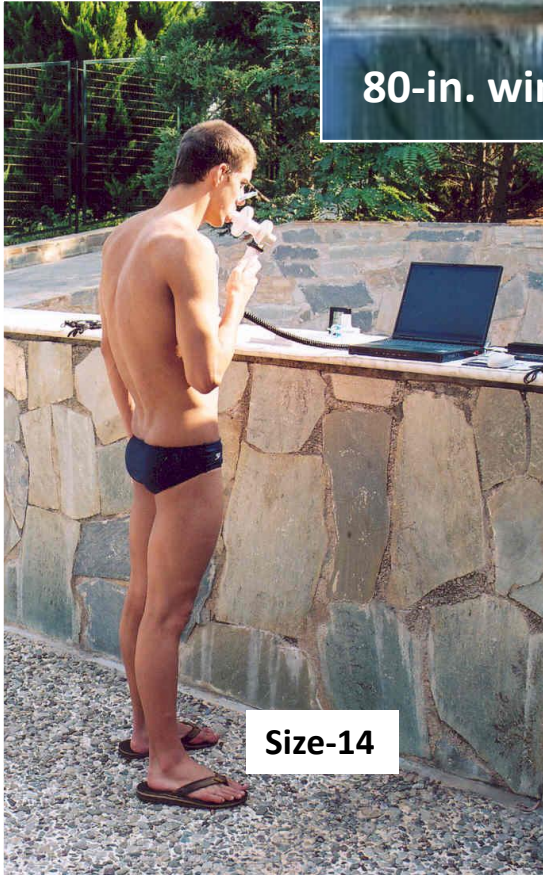
Talent ID + Development

Stage 1: Age-Group Competition





80-in. wingspan

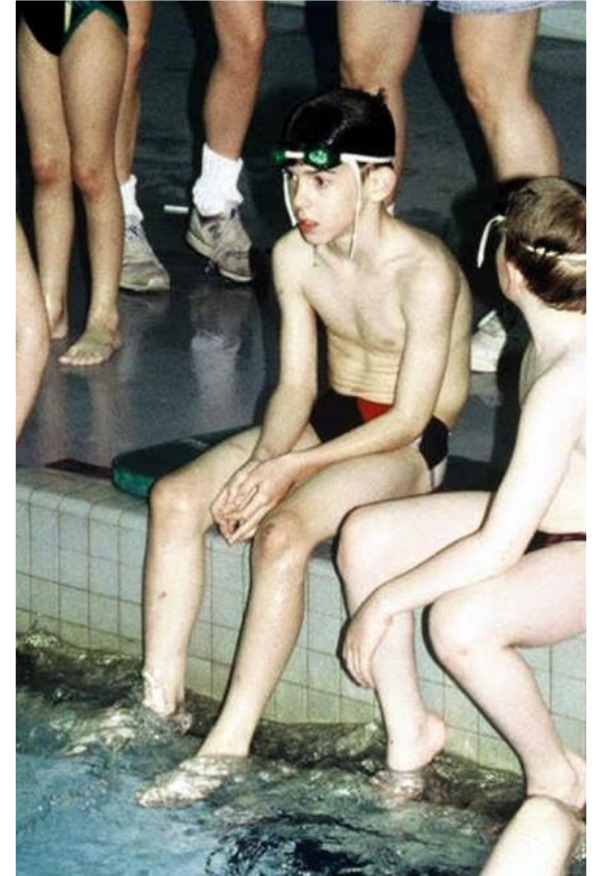


Size-14

Michael Phelps (20 yr)



Coach Bob Bowman



Michael Phelps (10 yr)

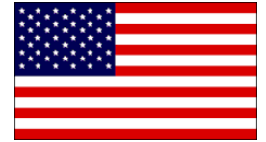
Talent ID + Development

Stage 2: High School (13-17 yr)



Talent ID + Development

Stage 3: University (17-21 yr)

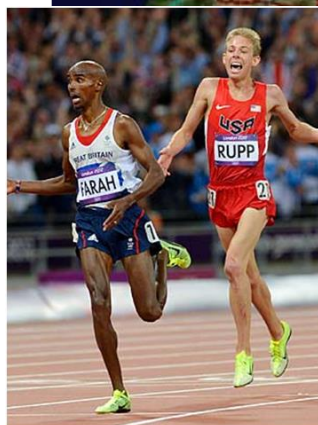


Talent ID + Development

Stage 4: Post-University & Professional (22+ yr)



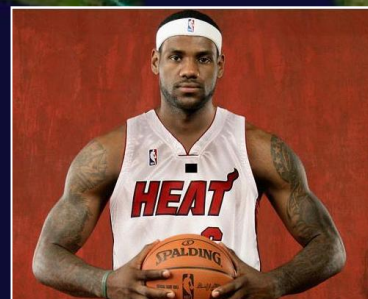
Portland, OR
Galen Rupp



Baltimore, MD
Michael Phelps



Beaumont, TX
W Gymnastics



Miami, FL
LeBron James

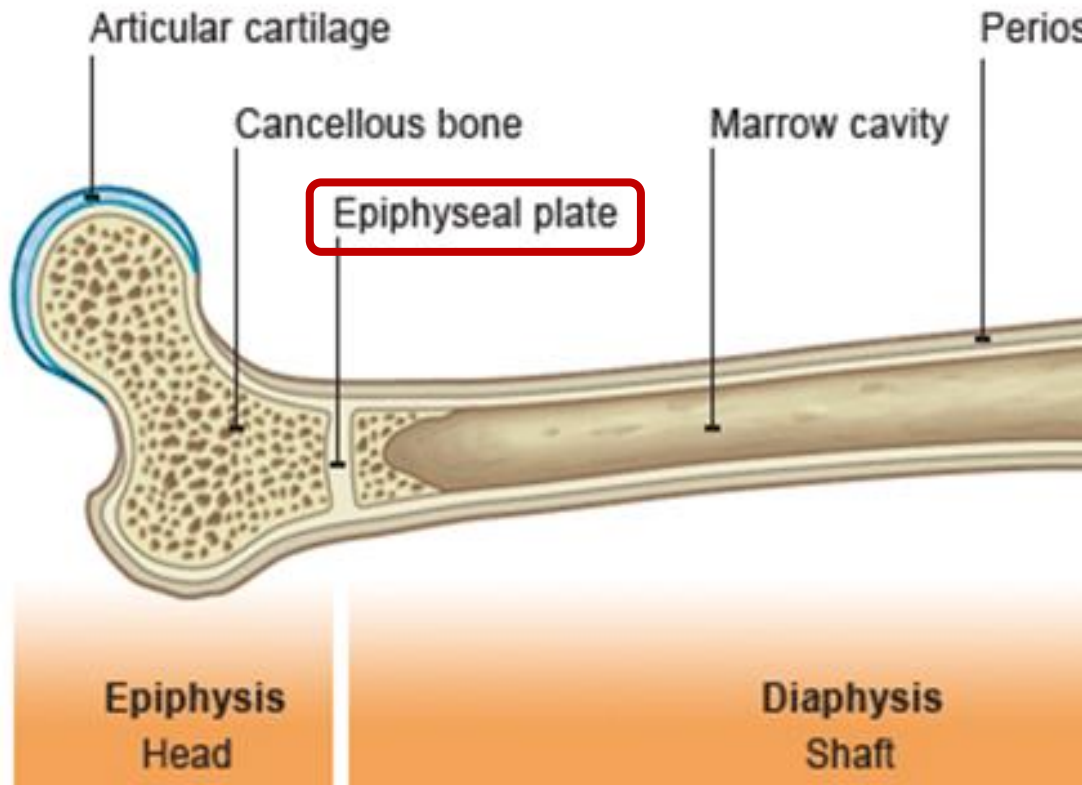
THE JUNIOR ELITE ATHLETE:

Physiological Characteristics and Training Considerations

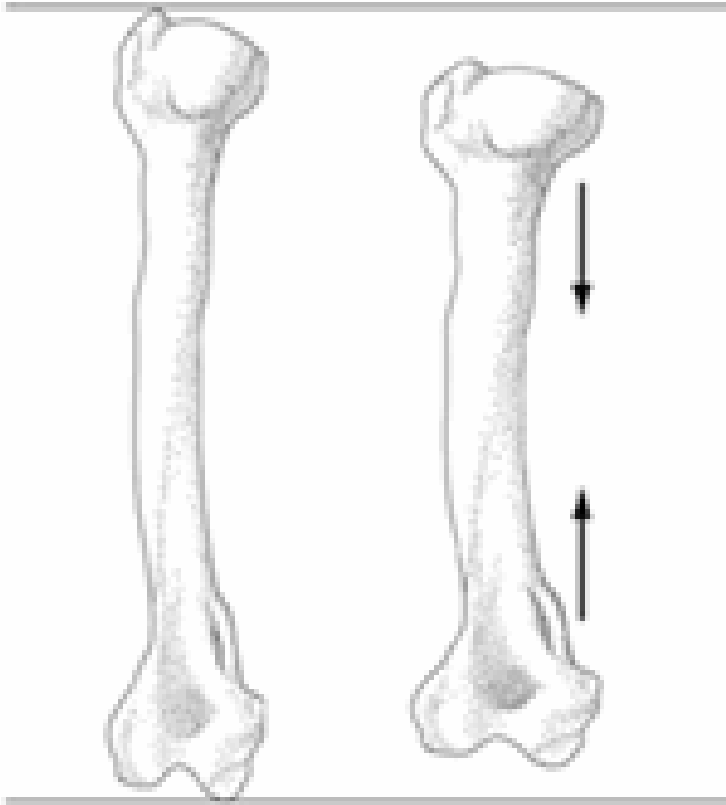
- Introduction / Physiology 101
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- ➔ ■ Increased Risk of Injury →
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Skeletal (Stress Fracture)
Female-Specific (Female AT)
Psychological (Early Drop Out)

INCREASED RISK of INJURY: Skeletal (Stress Fracture)



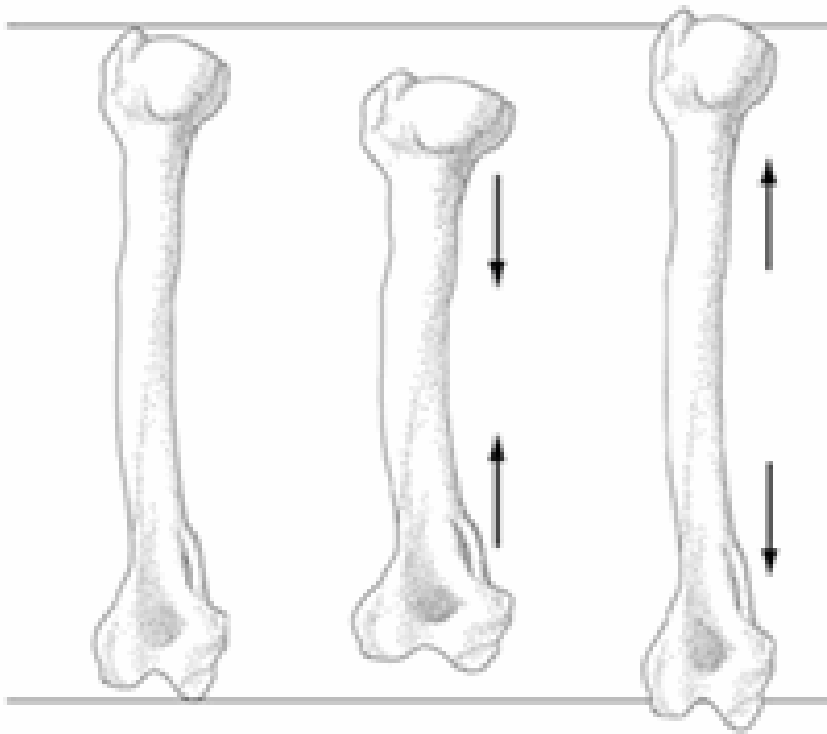
INCREASED RISK of INJURY: Skeletal (Stress Fracture)



COMPRESSION

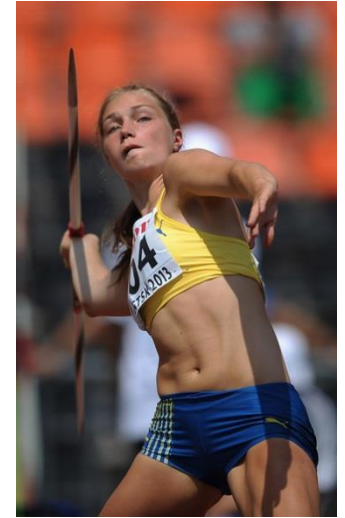


INCREASED RISK of INJURY: Skeletal (Stress Fracture)

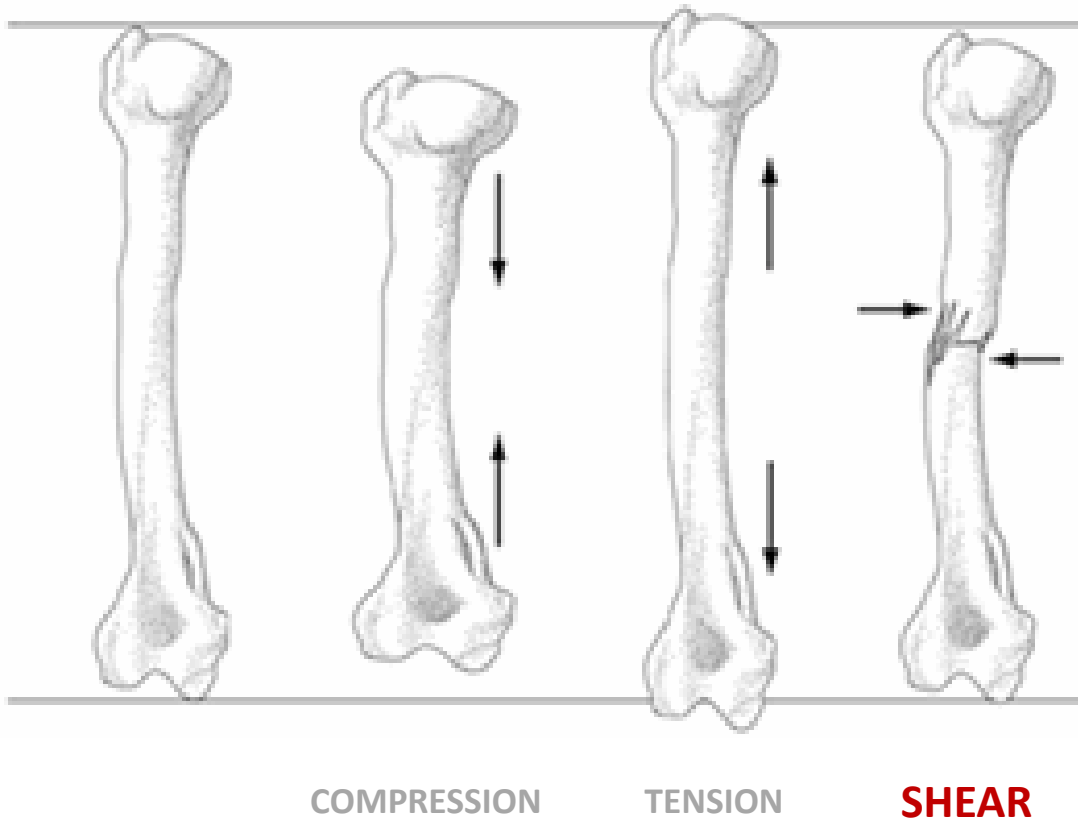


COMPRESSION

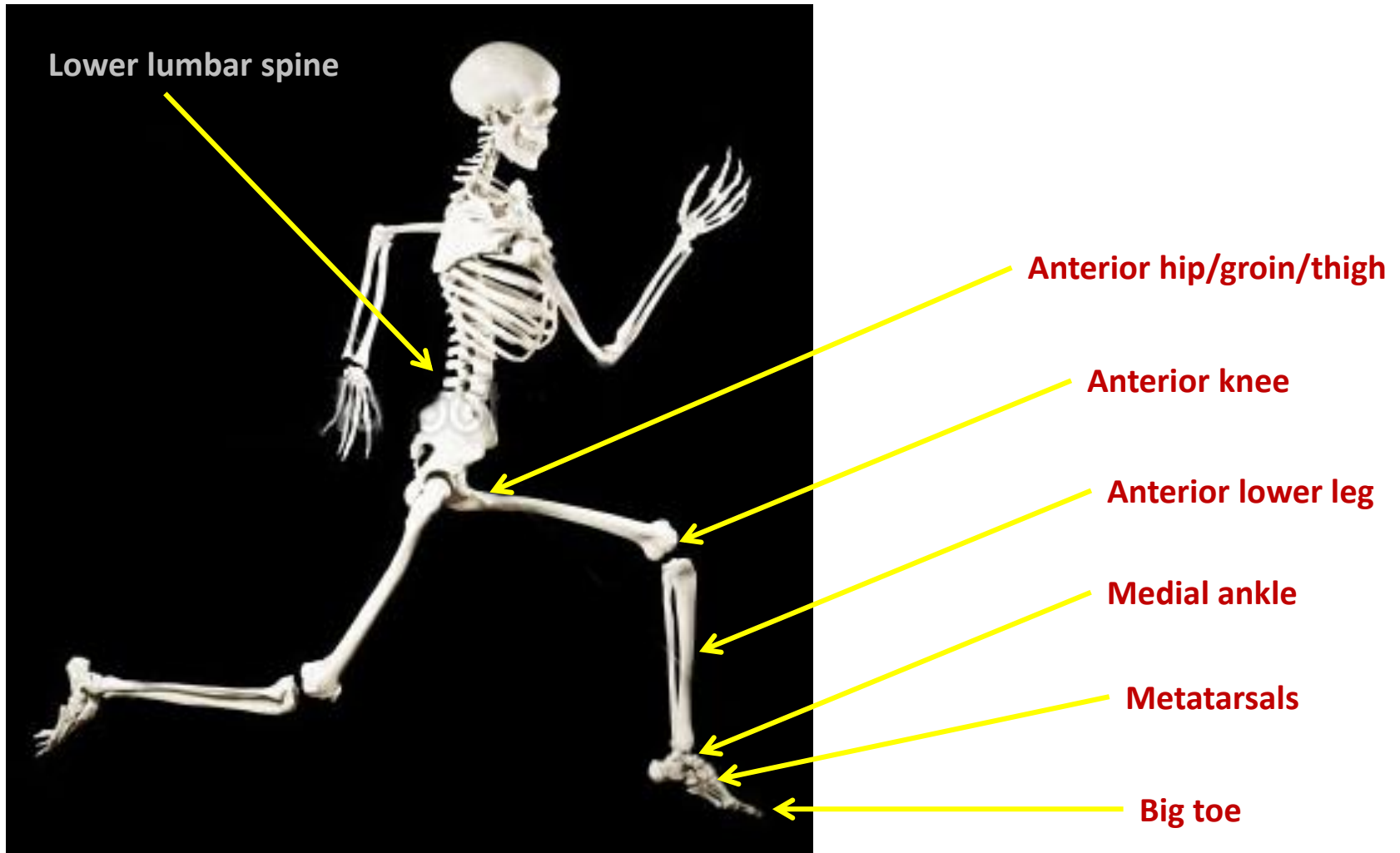
TENSION



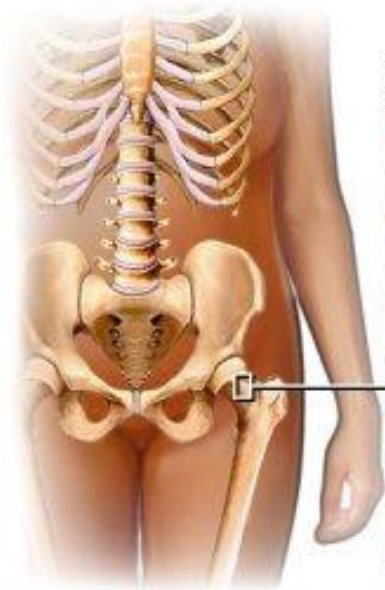
INCREASED RISK of INJURY: Skeletal (Stress Fracture)



INCREASED RISK of INJURY: Skeletal (Stress Fracture)



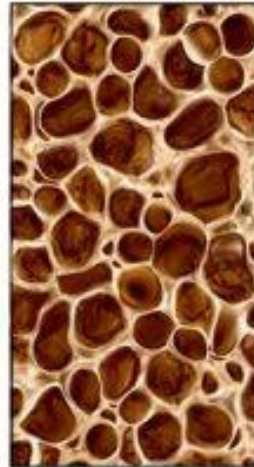
INCREASED RISK of INJURY: Female-Specific



Normal bone matrix



Osteoporosis



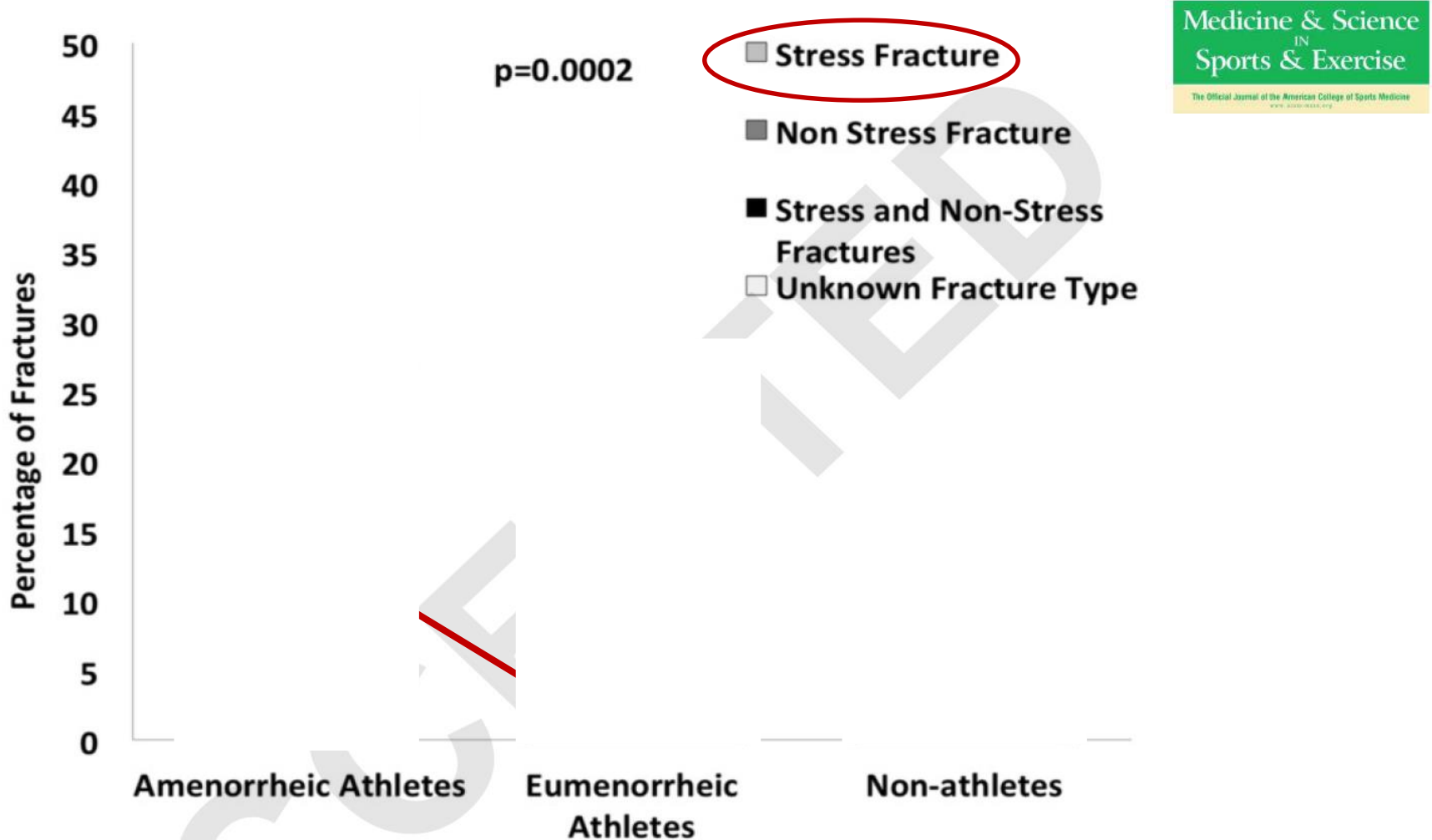
Female
Athlete
Triad



Fractures in Relation to Menstrual Status and Bone Parameters in Young Athletes

Kathryn E. Ackerman, Natalia Cano Sokoloff, Giovanna De Nardo Maffazioli, Hannah Clark, Hang Lee, and Madhusmita Misra

Medicine and Science in Sports and Exercise, 2014.



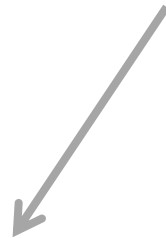
INCREASED RISK of INJURY: Psychological



SPORT SPECIALIZATION



Extremely high training load (adult level)
Extremely high time demands
Frequent competition and travel
High/Unrealistic performance expectations from coach/parents
Perfectionist personality



Injury

and/or



Burnout



“I used to really like this sport . . . but I don’t want to do it anymore!”



INCREASED RISK of INJURY: Prevention



**Conservative Training Load
(Volume, Intensity, # D/W)**



**Don't Overschedule Meets
(or # Events in Meet)**



Soft Surface Training



Aquatic Training



Resistance Training



Be Knowledgeable of Symptoms



Orthotics / Arch Supports



Vitamin D Health

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“Windows of Opportunity”
Specialization vs Diversification
Thermoregulation
Sleep

“WINDOWS OF OPPORTUNITY”

Sport Diversification

Sport Specialization

CHILD

Pre-Puberty
Age 6-10

ADOLESCENT

Puberty
Age 11-14

ADOLESCENT

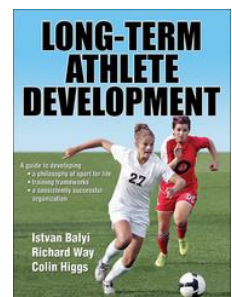
Post-Puberty
Age 15-18

YOUNG ADULT

Maturity
Age 19-25



Speed 1: Central Nervous System (quickness, change of direction, segmental speed, agility)
Speed 2: Anaerobic power + Anaerobic capacity (interval training)



“WINDOWS OF OPPORTUNITY”

Sport Diversification

Sport Specialization

CHILD

Pre-Puberty
Age 7-11

ADOLESCENT

Puberty
Age 12-13

ADOLESCENT

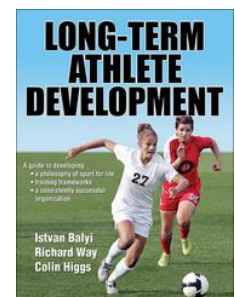
Post-Puberty
Age 13-18

YOUNG ADULT

Maturity
Age 19-25



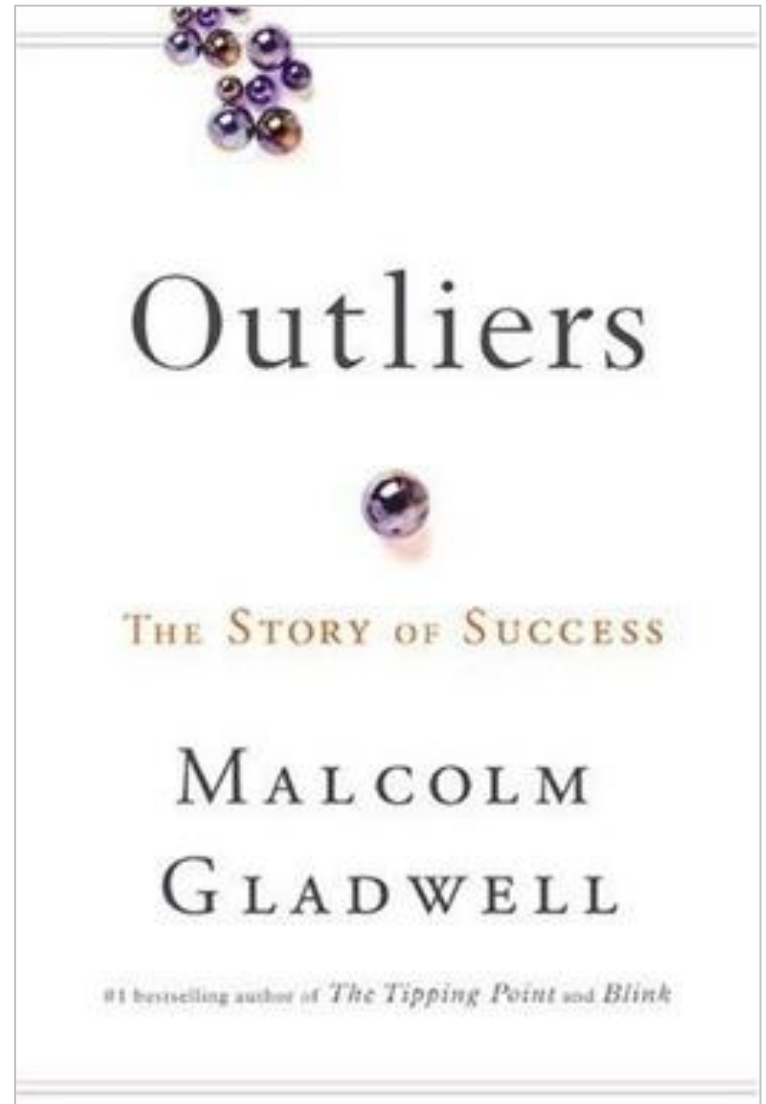
Speed 1: Central Nervous System (quickness, change of direction, segmental speed, agility)
Speed 2: Anaerobic power + Anaerobic capacity (interval training)



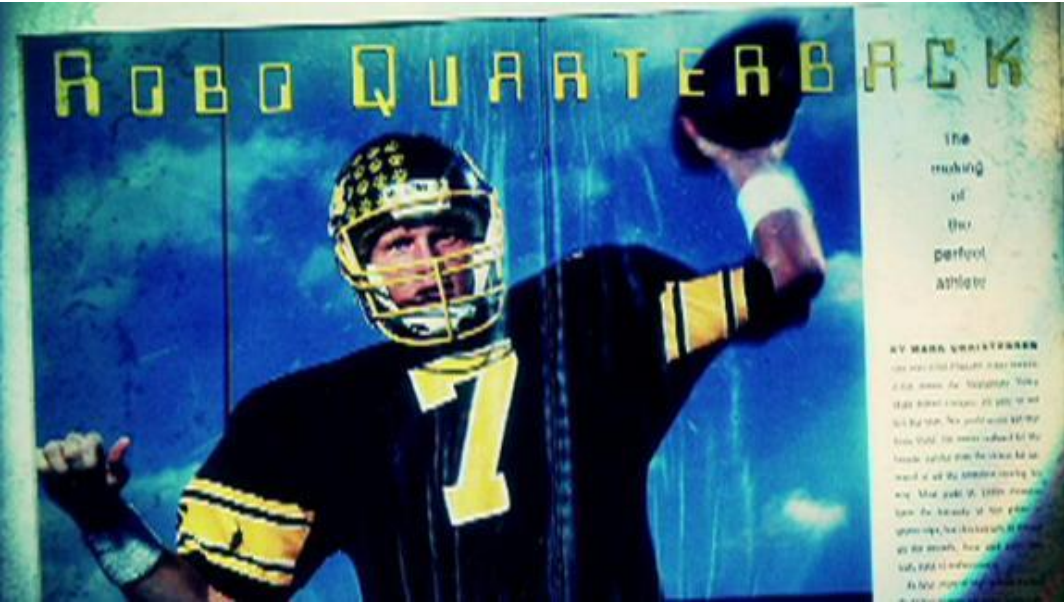
SPECIALIZATION vs DIVERSIFICATION



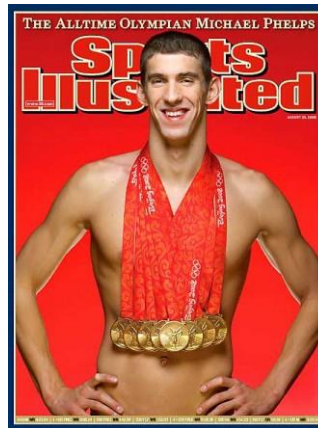
Iten, Kenya



SPECIALIZATION vs DIVERSIFICATION



SPECIALIZATION vs DIVERSIFICATION



1st US Olympic Team: age 15



1st Olympic Medal: age 19



SPECIALIZATION vs DIVERSIFICATION



Team USA swimmers who specialized early (< 12 yr) spent less time on the US National/Olympic Team than swimmers who specialized later (> 16 yr).



Team USA swimmers who specialized early (< 12 yr) retired from the sport significantly earlier than swimmers who specialized later (> 16 yr).

Sports-Specialized Intensive Training and the Risk of Injury in Young Athletes

Am J Sports Med 43:794-801, 2015

Neeru A. Jayanthi,^{††} MD, Cynthia R. LaBella,^{†§} MD, Daniel Fischer,[†] Jacqueline Pasulka,[‡] and Lara R. Dugas,[†] PhD, MPH

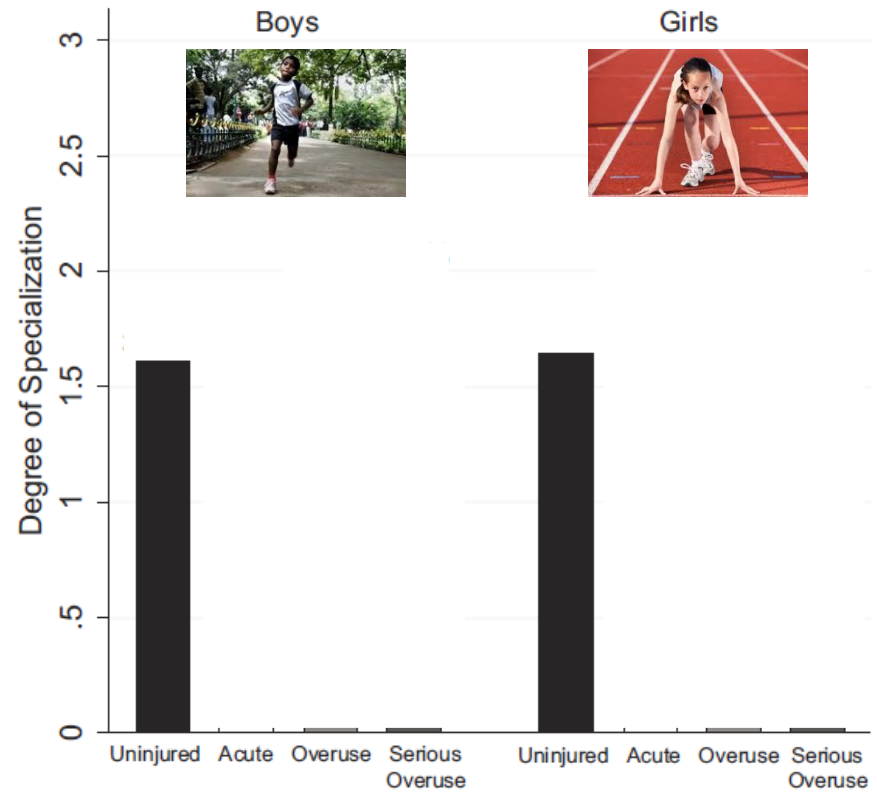
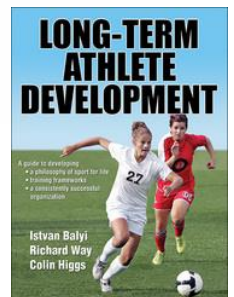
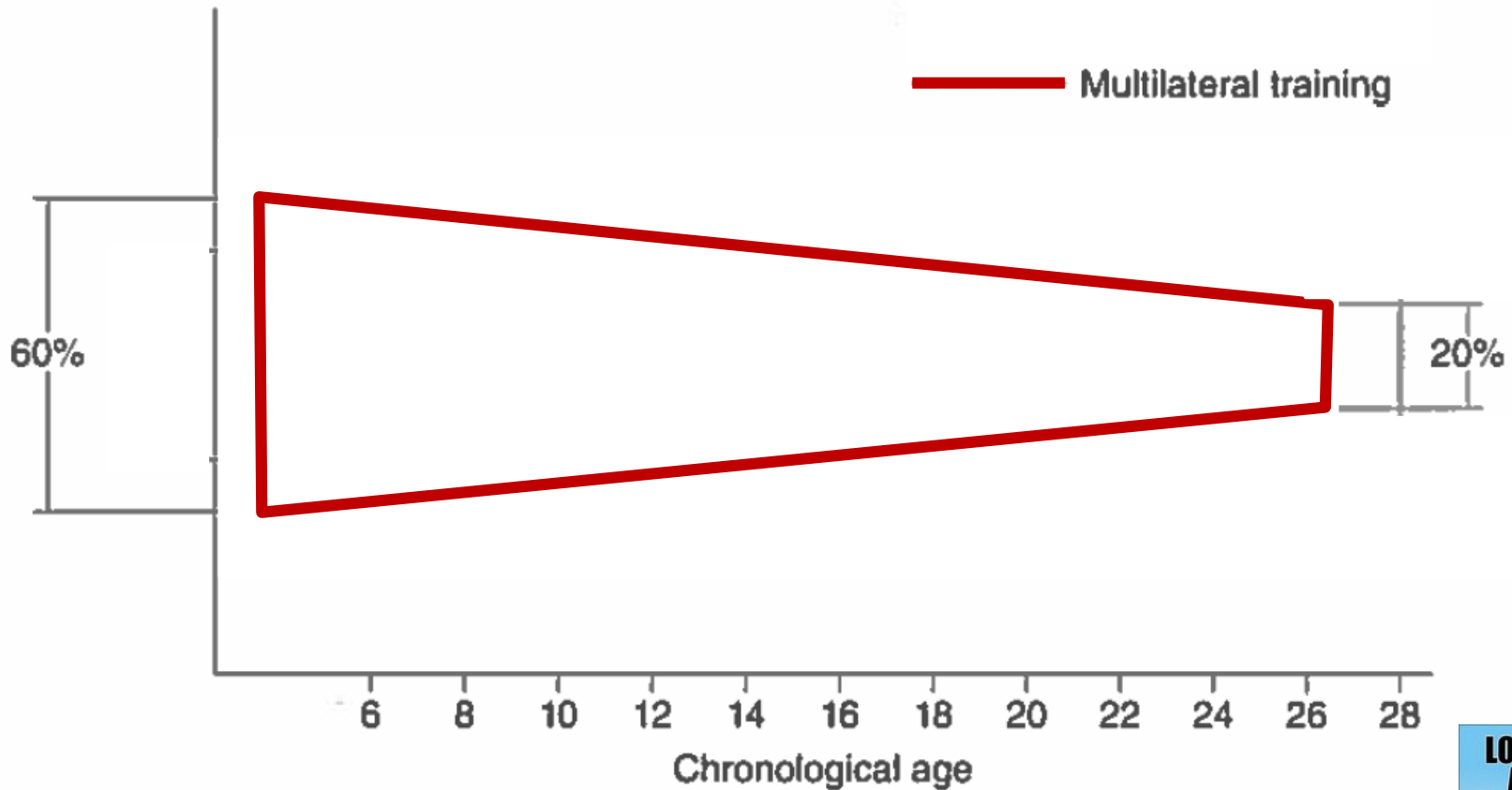
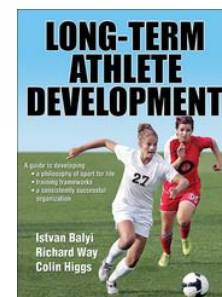
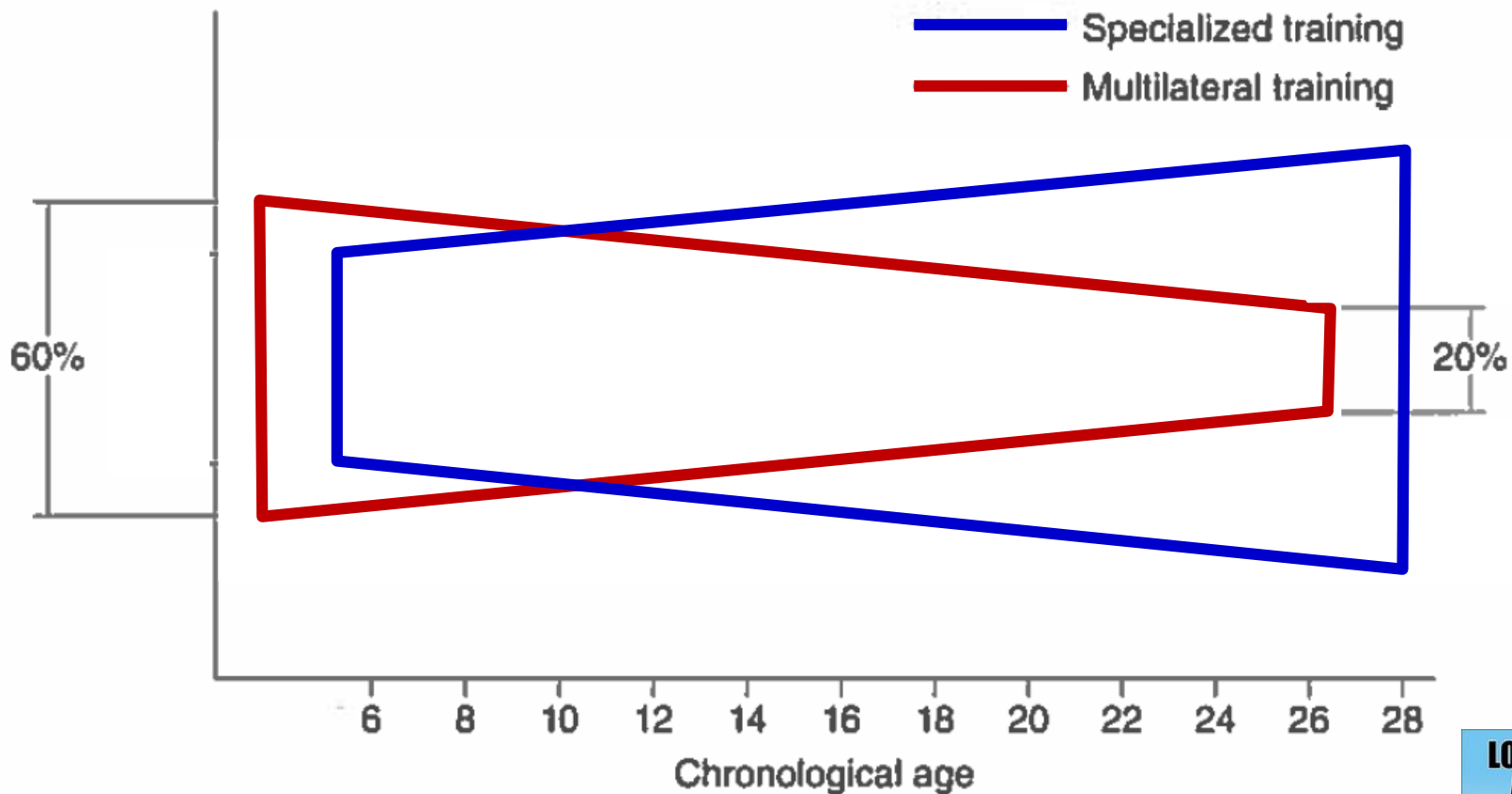


Figure 2. Degree of sports specialization by injury type and sex.

SPECIALIZATION vs DIVERSIFICATION



SPECIALIZATION vs DIVERSIFICATION



SPECIALIZATION vs DIVERSIFICATION



Agility



Balance



Coordination



Speed



Jumping



Climbing

“Diversified sports training during early and middle adolescence appears to be a more effective strategy in ultimately developing elite-level skills in a specific sport/event due to a positive transfer of physical and mental skills.”

Overuse injuries and burnout in youth sports. DiFiori et al. *Clin J Sport Med* 24: 3-20, 2014.



Throwing



Dribbling



Kicking



Throwing



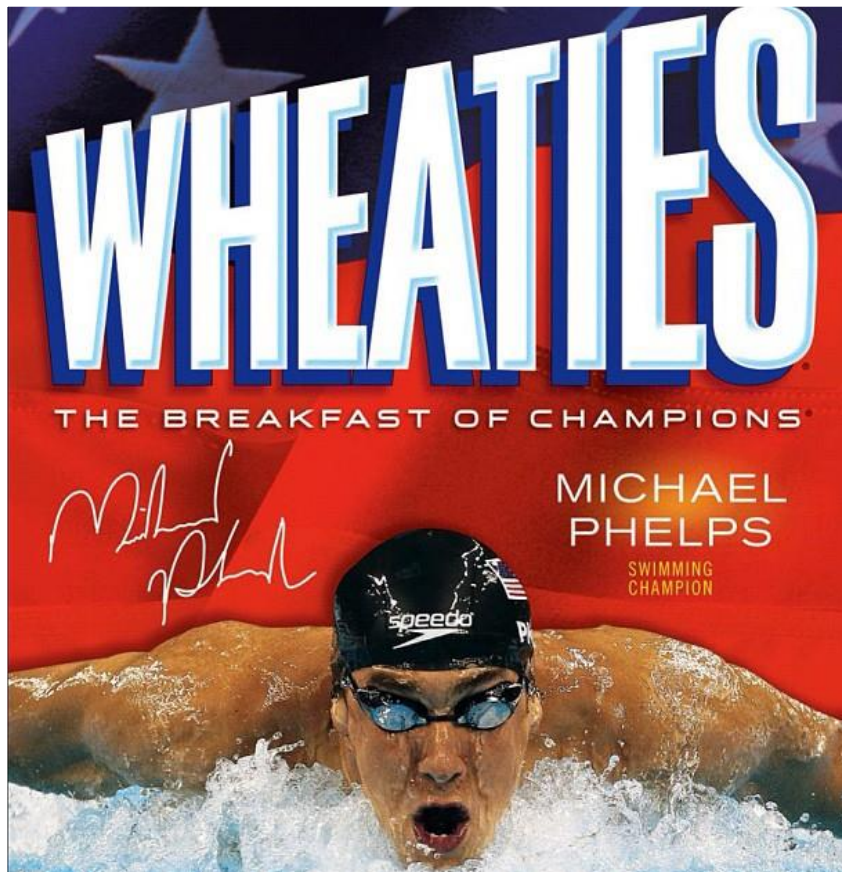
Hitting



Catching

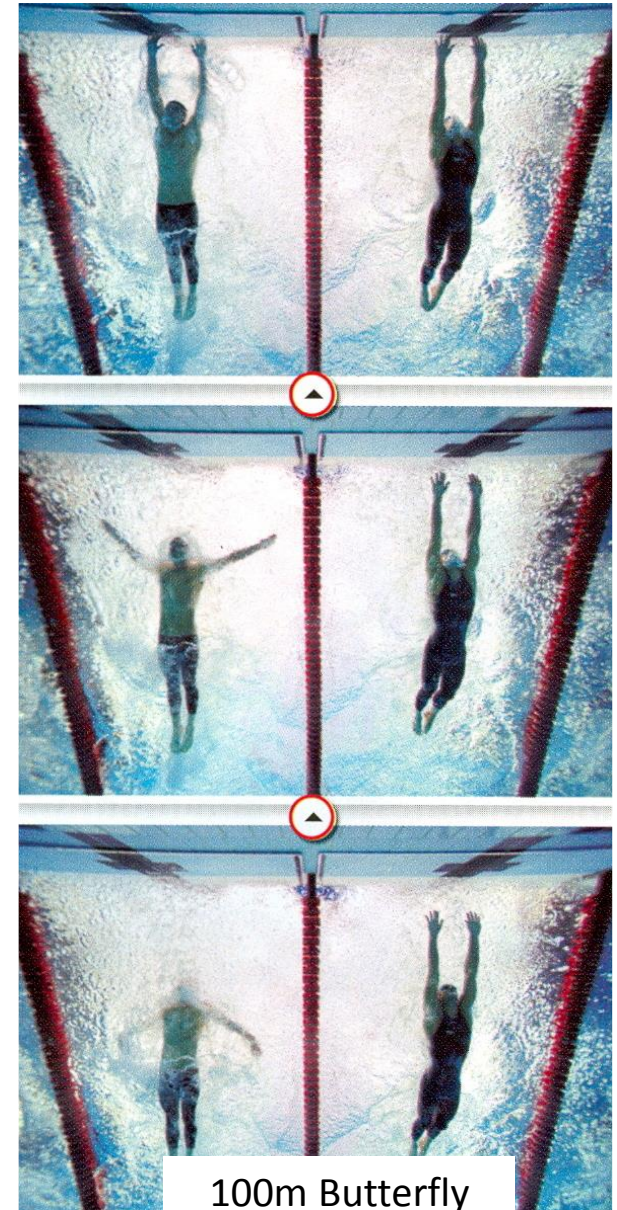


“The difference between being on the Wheaties box . . . and eating from one!”



Michael Phelps
(USA) 50.58

Milorad Cavic
(SER) 50.59



100m Butterfly

SPECIALIZATION vs DIVERSIFICATION

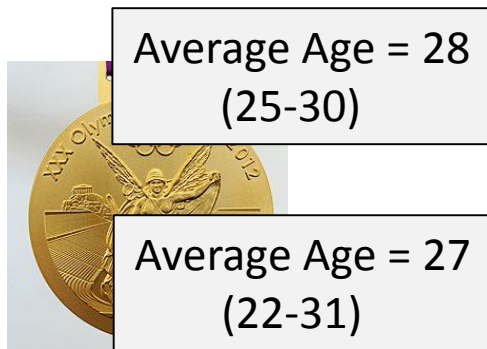


TALENT

TRANSFER

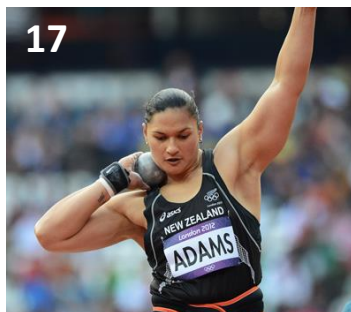


SPECIALIZATION vs DIVERSIFICATION



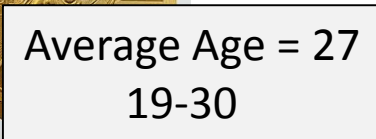
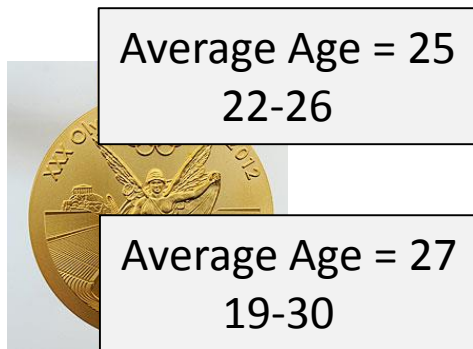
EVENT	WOMEN	Nation	Performance	Age
100	Fraser-Pryce	JAM	10.75	25
200	Felix	USA	21.88	26
400	Richards-Ross	USA	49.55	27
800	Savinova	RUS	1:56.19	27
1500	Cakir Alptekin	TUR	4:10.23	27
3000 SC	Zaripova	RUS	9:06.72	26
5000	Defar	ETH	15:04.25	28
10,000	Dibaba	ETH	30:20.75	27
100 H	Pearson	AUS	12.35	25
400 H	Antyukh	RUS	52.70	31
Marathon	Gelana	ETH	2:23:07	24
20 km RW	Lashmanova	RUS	1:25:02	20
LJ	Reese	USA	7.12	25
TJ	Rypakova	KAZ	14.98	27
HJ	Chicherova	RUS	2.05	30
PV	Suhr	USA	4.75	30
SP	Adams	NZL	20.70	27
D	Perkovic	CRO	69.11	22
J	Spotakova	CZE	69.55	31
HT	Lysenko	RUS	78.18	28
Heptathlon	Ennis	GBR	6955	26

SPECIALIZATION vs DIVERSIFICATION



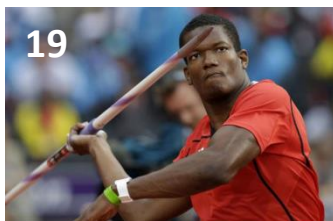
EVENT	WOMEN	Nation	Performance	Age
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Heptathlon	Ennis	GBR	6955	26

SPECIALIZATION vs DIVERSIFICATION



EVENT	MEN	Nation	Performance	Age
100	Bolt	JAM	9.63	26
200	Bolt	JAM	19.32	26
400	James	GRN	43.94	19
800	Rudisha	KEN	1:40.91	23
1500	Makhloufi	ALG	3:34.08	24
3000 SC	Kemboi	KEN	8:18.56	30
5000	Farah	GBR	13:41.66	30
10,000	Farah	GBR	27:30.42	30
100 H	Merritt	USA	12.92	27
400 H	Sanchez	DOM	47.63	34
Marathon	Kiprotich	UGA	2:08:01	23
20 km RW	Chen	CHN	1:18:46	20
50 km RW	Kirdyapkin	RUS	3:35:59	32
LJ	Rutherford	GBR	8.31	25
TJ	Taylor	USA	17.81	22
HJ	Ukhov	RUS	2.38	26
PV	Lavillenie	FRA	5.97	25
SP	Majewski	POL	21.89	30
D	Harting	GER	69.11	27
J	Walcott	TRI	84.58	19
HT	Pars	HUN	80.59	30
Decathlon	Eaton	USA	8869	24


SPECIALIZATION vs DIVERSIFICATION





EVENT	MEN	Nation	Performance	Age
100	Bolt	JAM	9.63	26
200	Bolt	JAM	19.32	26
400	James	GRN	43.94	19
800	Rudisha	KEN	1:40.91	23
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D	Harting	GER	69.11	27
J	Walcott	TRI	84.58	19
HT	Pars	HUN	80.59	30
Decathlon	Eaton	USA	8869	24


TRAINING CONSIDERATIONS: Thermoregulation

The Heat Index																					
Air Temp (°F)	Relative Humidity (percentage)																				
	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100
135°	120	126																			
130°	117	122	131																		
125°	111	116	123	131	141																
120°	107	111	116	123	130	139	148														
115°	105	107	111	115	120	127	135	143	151												
110°	99	102	105	108	112	117	123	130	137	143	150										
105°	95	97	100	102	105	109	113	118	123	129	135	142	149								
100°	91	93	95	97	99	101	104	107	110	115	120	126	132	138	144	150					
95°	87	88	90	91	93	94	96	98	101	104	107	110	114	119	124	130	136	140	150		
90°	83	84	85	86	87	88	90	91	93	95	96	98	100	102	106	109	113	117	122	126	131
85°	78	79	80	81	82	83	84	85	86	87	88	89	90	91	93	95	97	99	102	105	108
80°	73	74	75	76	77	77	78	79	79	80	81	81	82	83	84	85	86	87	88	89	90
75°	69	69	70	71	72	72	73	73	74	74	75	75	76	76	77	77	78	78	79	79	80
70°	64	64	65	65	66	66	67	67	68	68	69	69	70	70	70	70	71	71	71	71	72

 = Heatstroke risk extremely high

 = Heat exhaustion likely, heatstroke possible

 = Heat exhaustion possible

 = Fatigue possible

TRAINING CONSIDERATIONS: Thermoregulation

Cooling Strategies: Hydration for Training/Competition < 1hr

Example: Sprints, Hurdles, Jumps, Throws

Exercise intensity: 80 – 130% VO_{2max}

Primary concern: Fluid:

1. to negate the rise in core temp
2. to replace H_2O lost via sweat

Fluid ingestion: **Pre** = 1 x 8-10 oz. CHO-E
During / After = 2 x 8-10 oz. H_2O (or CHO-E)



CHO-E = Carbohydrate-Electrolyte drink (eg, Gatorade, Powerade)

TRAINING CONSIDERATIONS: Thermoregulation

Cooling Strategies: Hydration for Training/Competition **1-3+ hr**

Example: Middle distance, Long distance

Exercise intensity: 60 – 90% $\text{VO}_{2\text{max}}$

Primary concerns: Fluid:

1. to negate the rise in core temp
2. to replace H_2O lost via sweat

Electrolytes:

1. to replace Na^+ , Cl^- , K^+ lost via sweat

CHO:

1. to maintain blood Glucose for immediate energy
2. to replenish liver/muscle glycogen post-exercise

Fluid ingestion:

Pre = 1 x 8-10 oz. CHO-E ~ 30-60 min pre

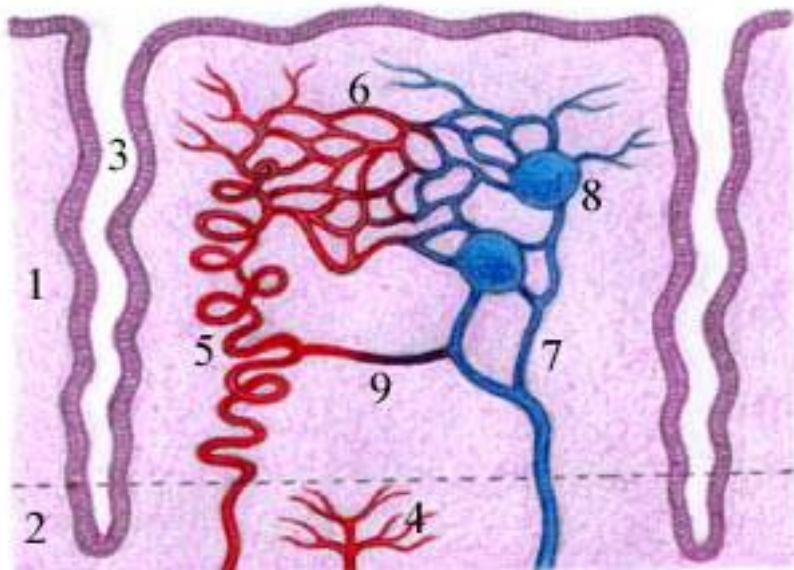
During = 2 x 8-10 oz. CHO-E per hour

After = 1-2 x 8-10 oz. CHO-E-PRO within 30-60 min post



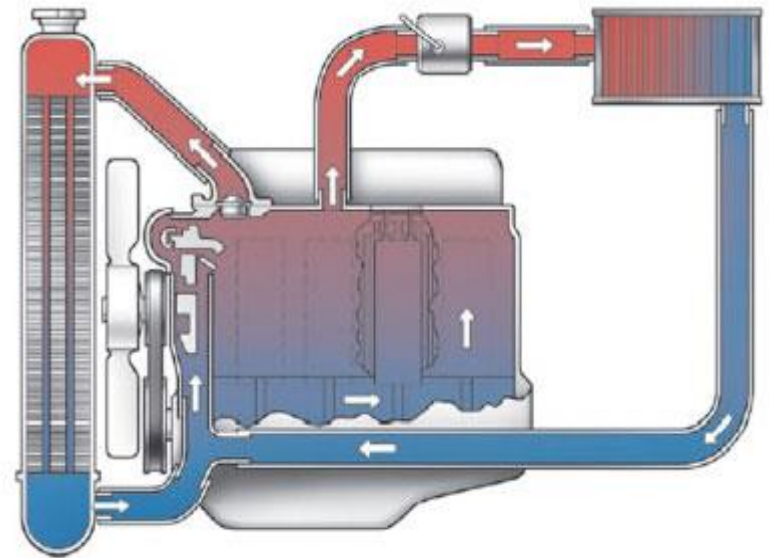
CHO-E-PRO = Carbohydrate-Electrolyte-Protein drink (eg, Gatorade, Powerade, LF chocolate milk)

TRAINING CONSIDERATIONS: Thermoregulation

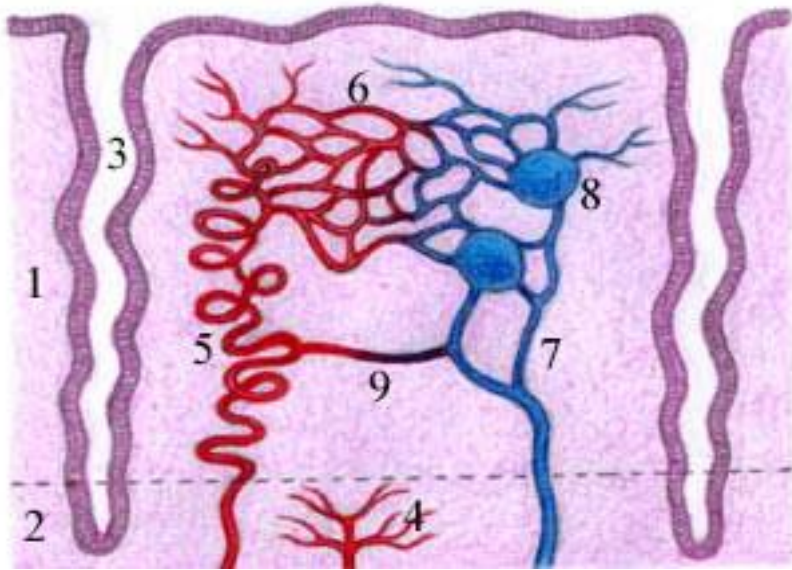


Arterio-venous anastomoses (AVAs)

Automobile cooling system

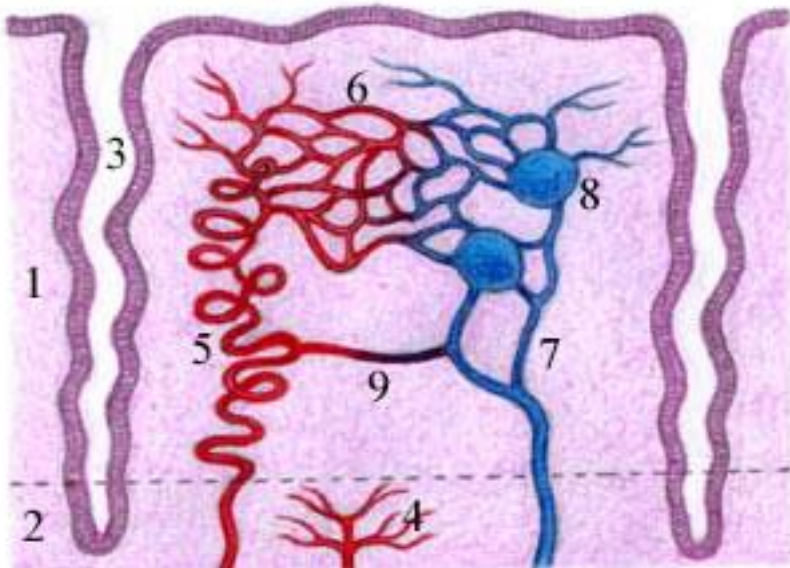


TRAINING CONSIDERATIONS: Thermoregulation

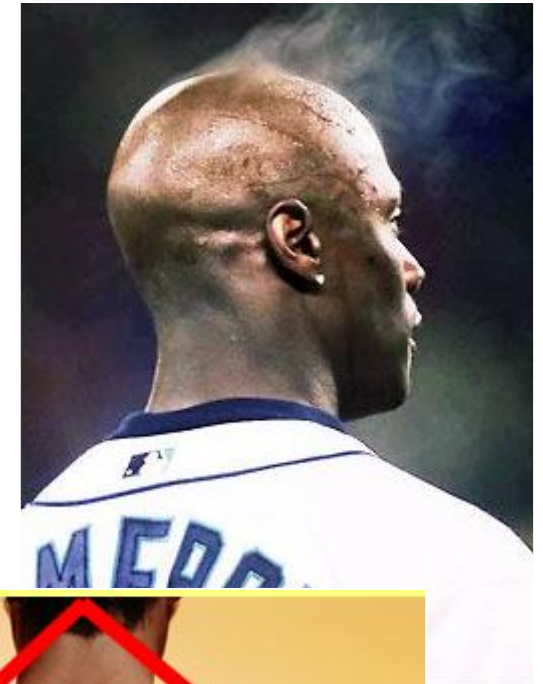


Arterio-venous anastomoses (AVAs)

TRAINING CONSIDERATIONS: Thermoregulation



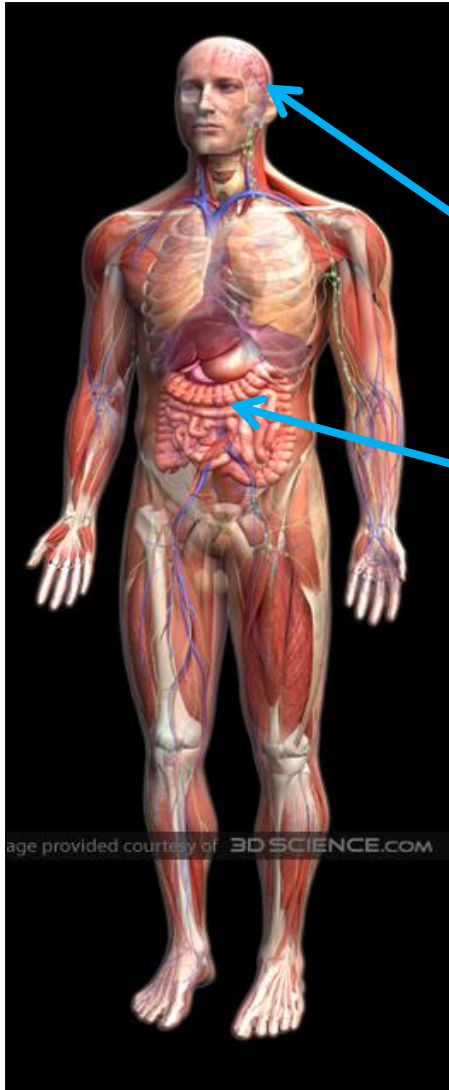
Arterio-venous anastomoses (AVAs)



TRAINING CONSIDERATIONS: Thermoregulation



TRAINING CONSIDERATIONS: Thermoregulation



TRAINING CONSIDERATIONS: Thermoregulation

Ice Slurry Ingestion Increases Core Temperature Capacity and Running Time in the Heat

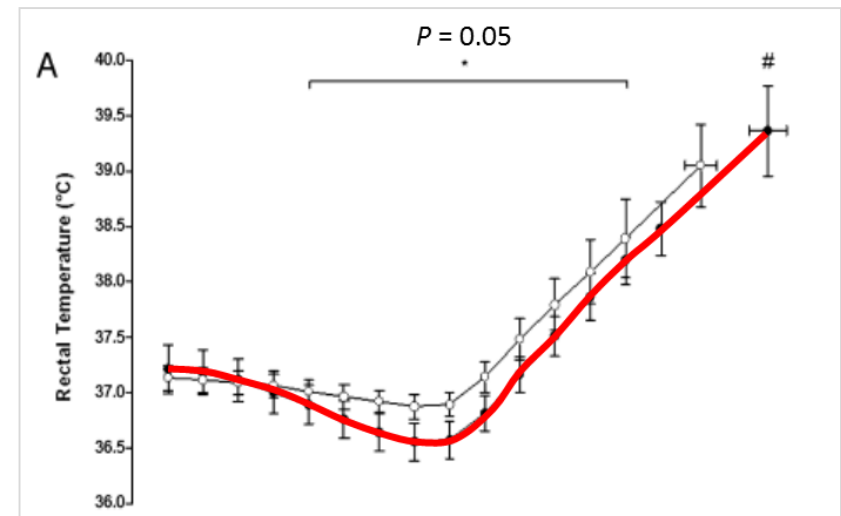
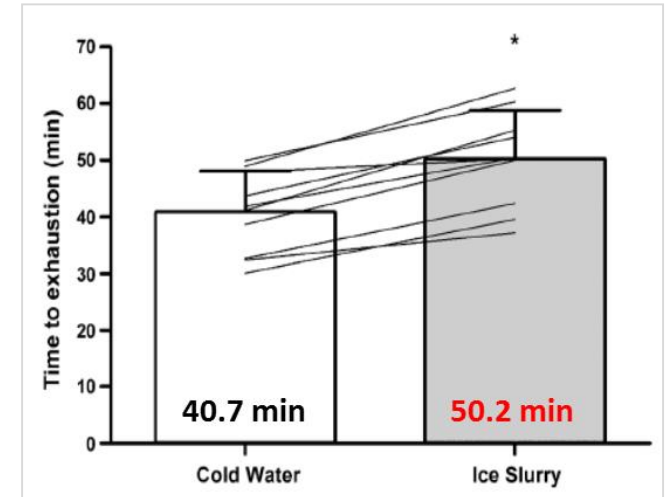
Med Sci Sports Exerc 4:717-725, 2010

SLURPEE = 7.5 g/kg TBM

Ingested equally over 30 min prior to run

Run to exhaustion @ VT1

93° F / 55% RH



TRAINING CONSIDERATIONS: Thermoregulation

Arctic Heat®

\$200

XS, S, M, L, XL

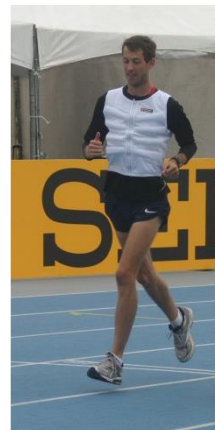


Wear during warmup and cooldown
Stays frozen 30-60 min



Freezer
~3 hr

Transport and keep frozen in ice chest



TRAINING CONSIDERATIONS: Sleep



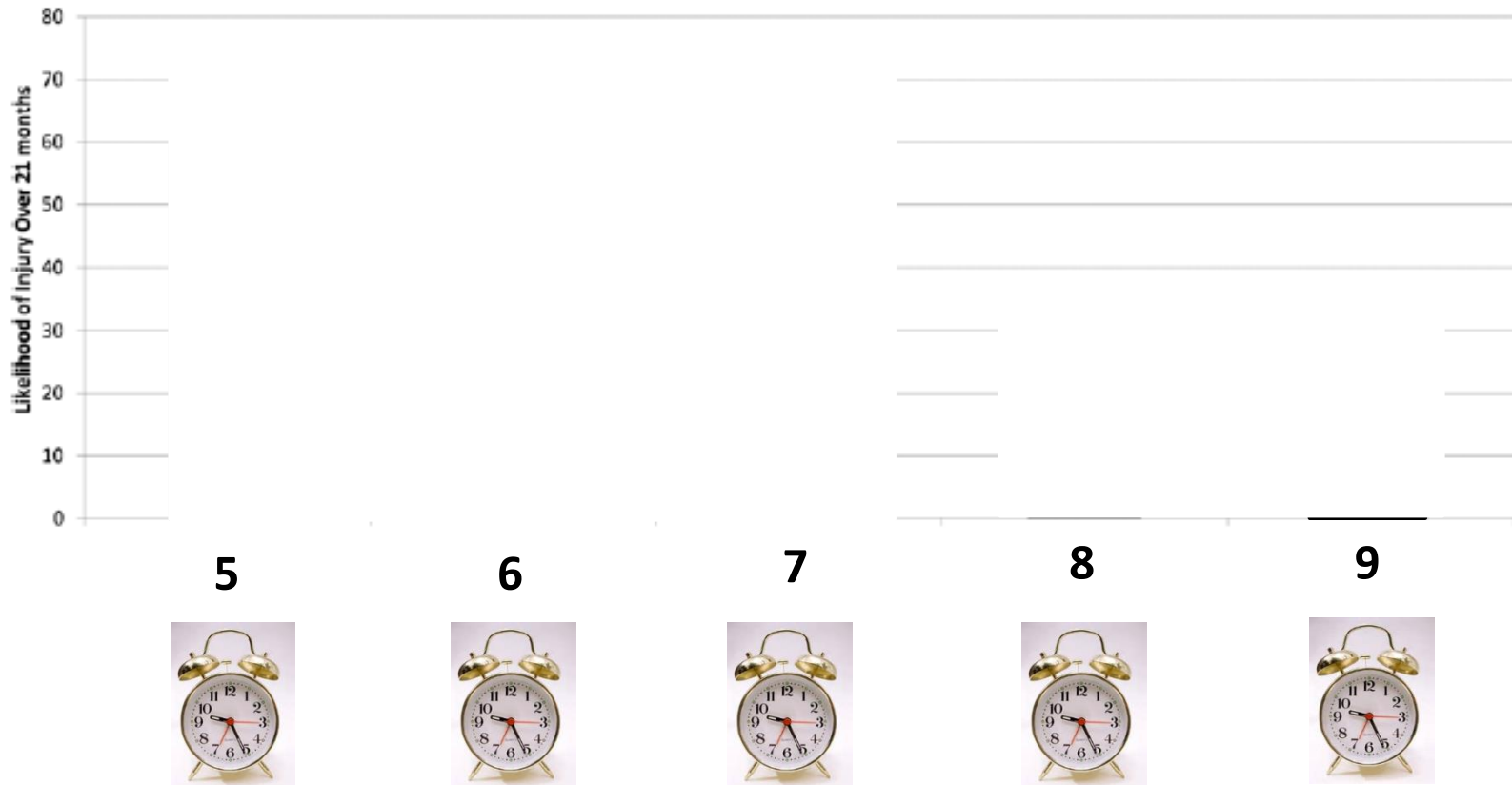
Chronic Lack of Sleep is Associated With Increased Sports Injuries in Adolescent Athletes

Matthew D. Milewski, MD,* David L. Skaggs, MD, MMM,†
Gregory A. Bishop, MS,‡ J. Lee Pace, MD,† David A. Ibrahim, MD,†
Tishya A.L. Wren, PhD,† and Audrius Barzdukas, MEd‡

J Pediatr Orthop 34: 129-133, 2014



Likelihood of Injury Based on Hours of Sleep per Night



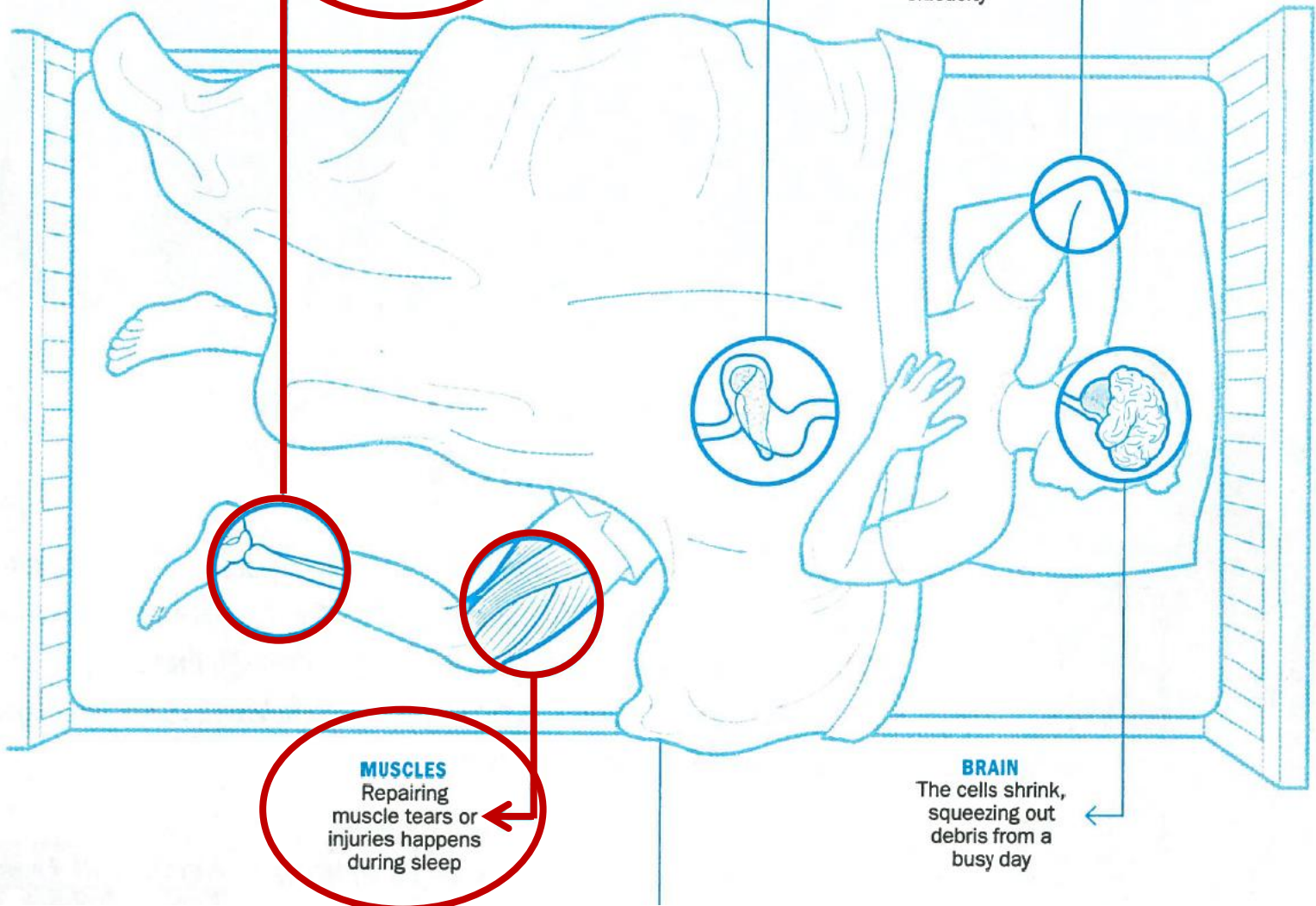
PANCREAS
Without sleep,
we become less
able to break
down sugar from
our diet

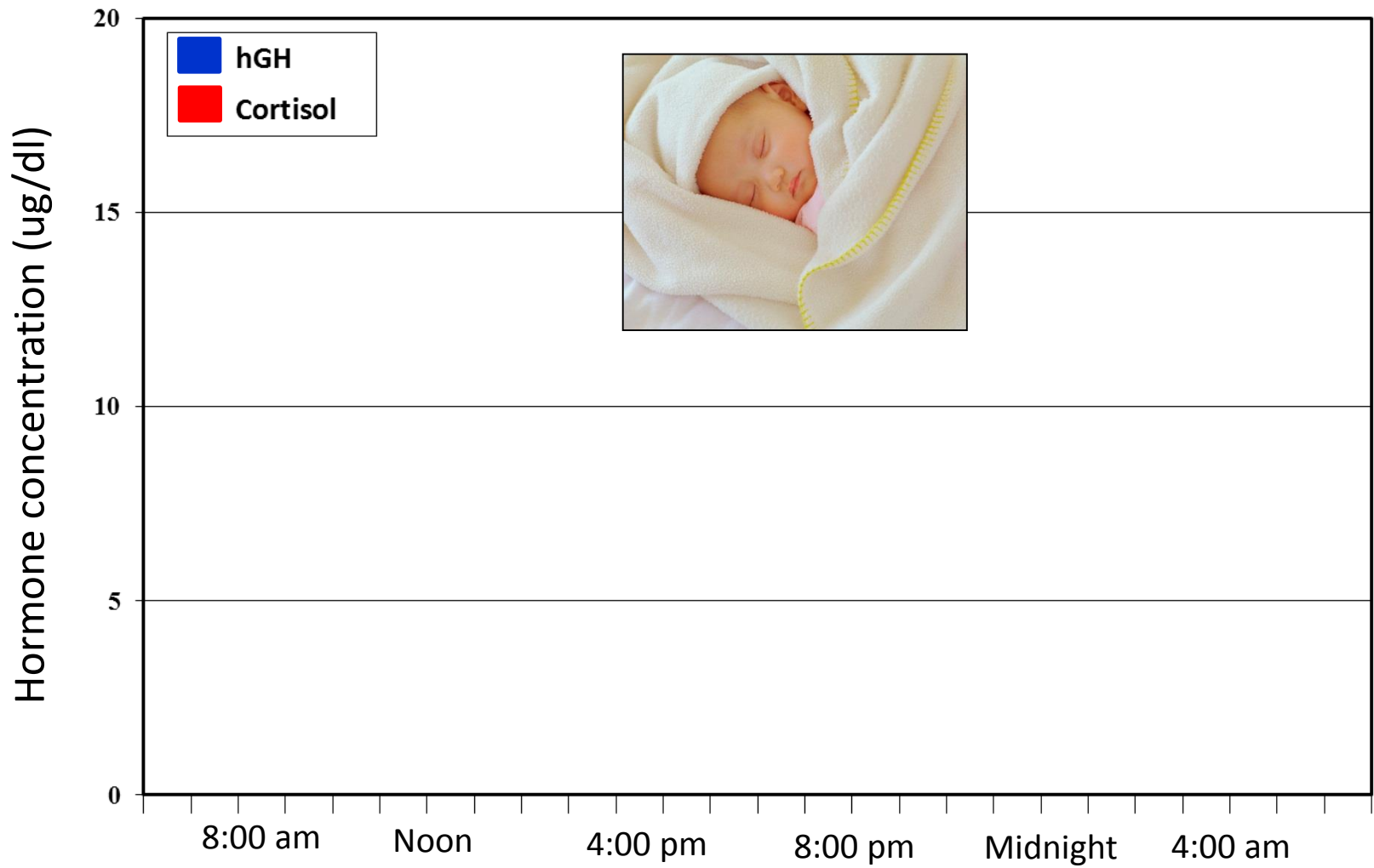
SKIN
Beauty rest is
when cells churn
out growth factors
to repair damage
and maintain
elasticity

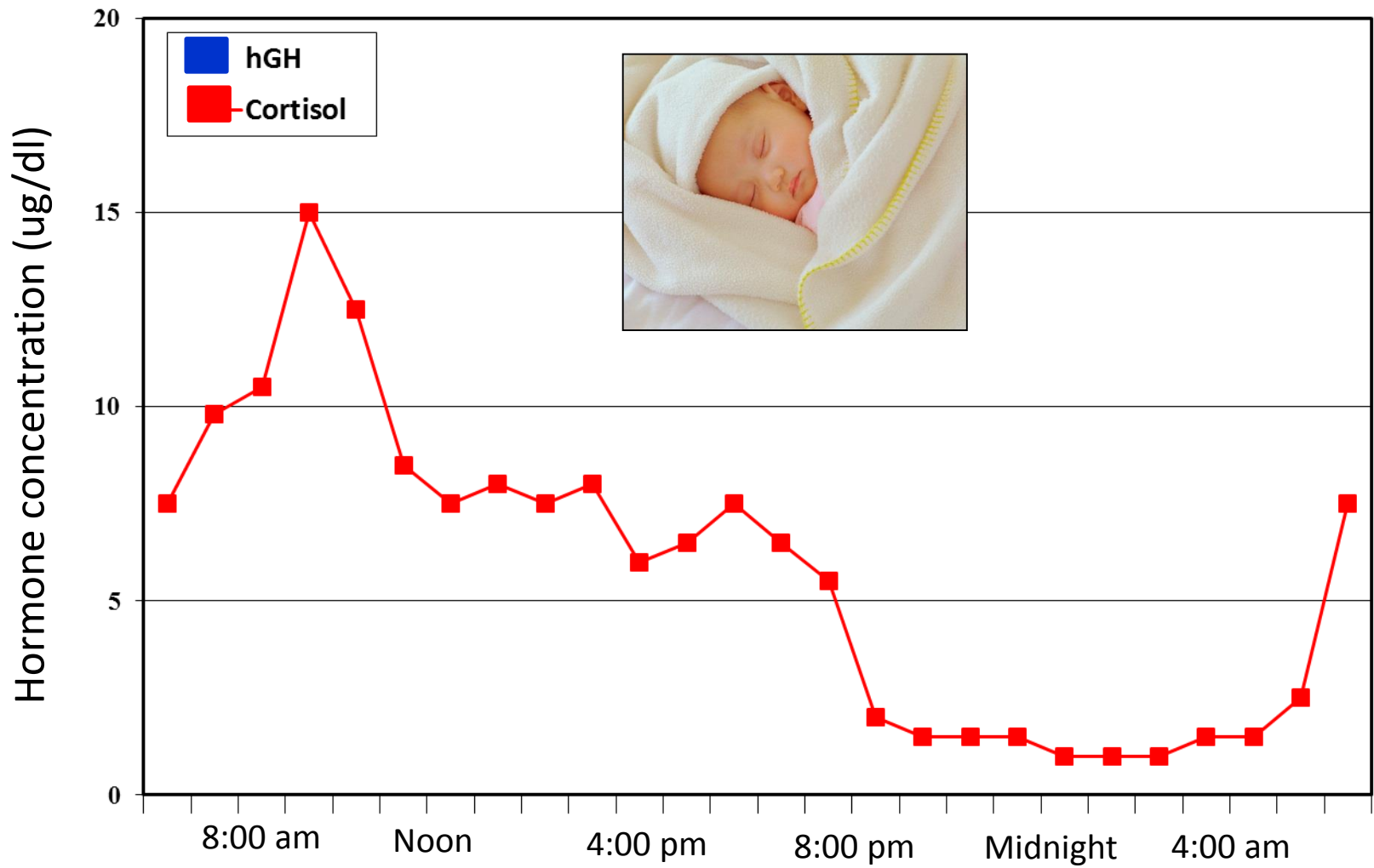
BONE
Wear and tear
is remedied
with intensified
bone building

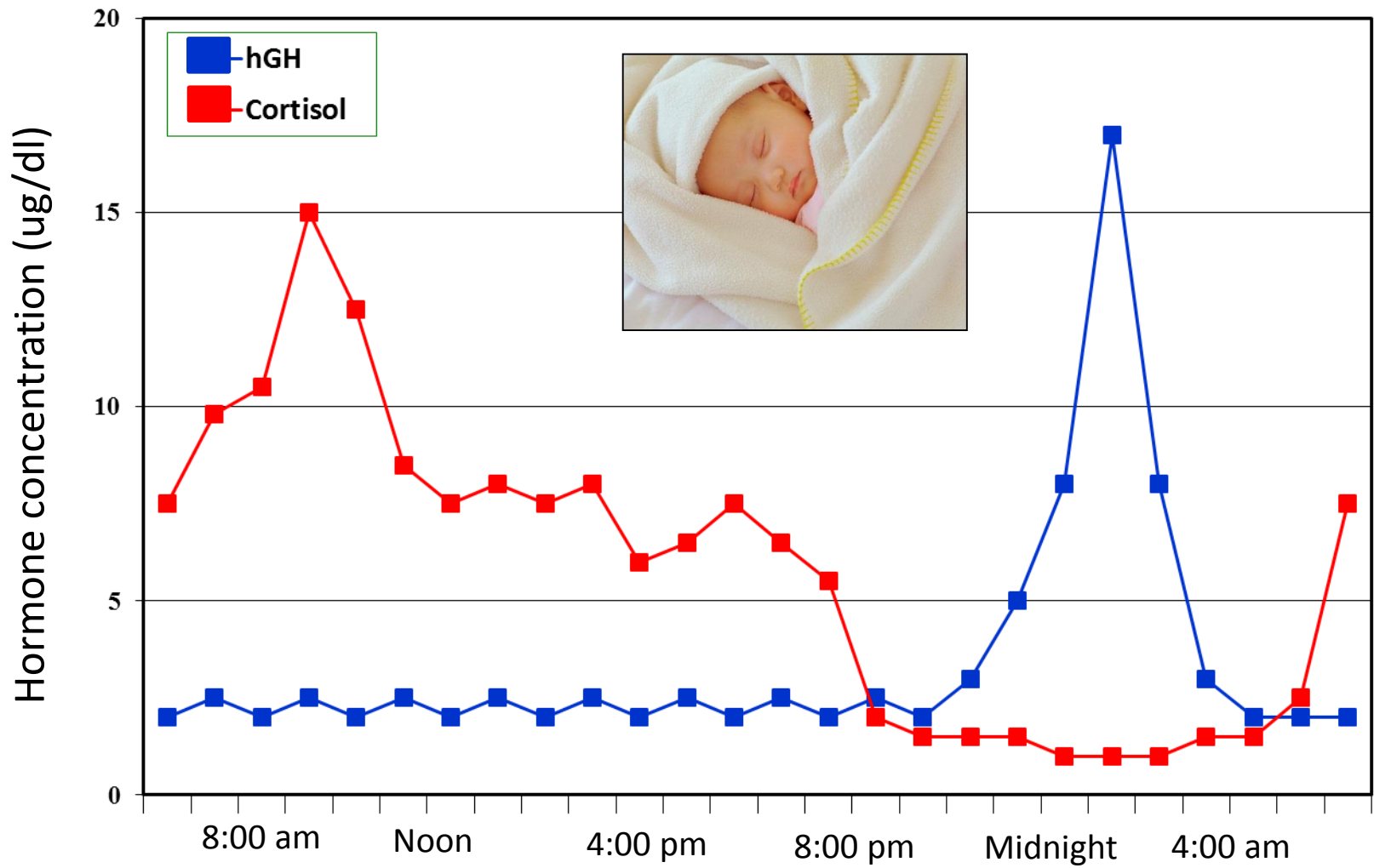
BRAIN
The cells shrink,
squeezing out
debris from a
busy day

MUSCLES
Repairing
muscle tears or
injuries happens
during sleep











==

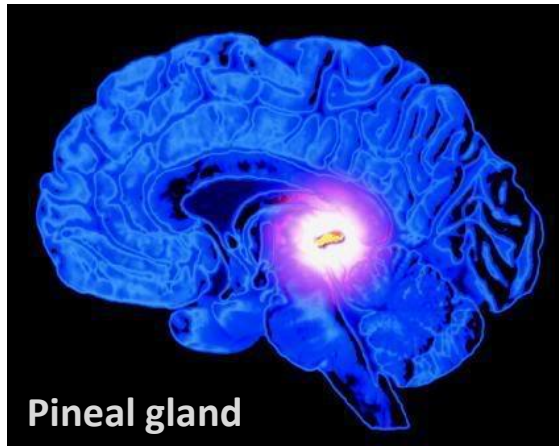


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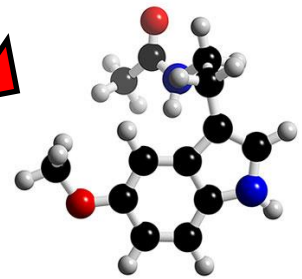


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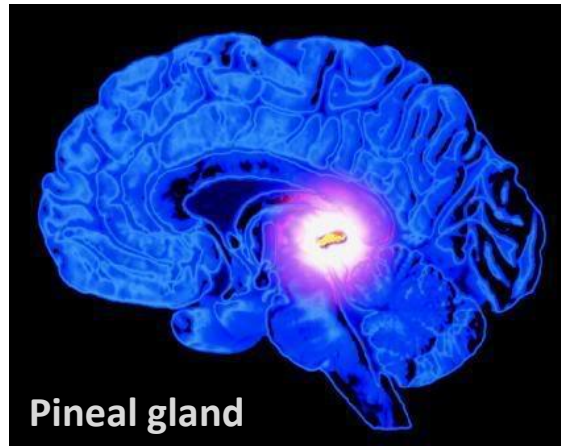




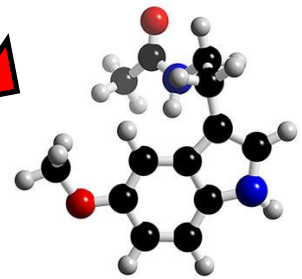
Pineal gland



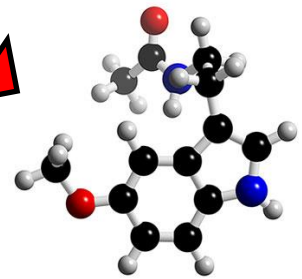
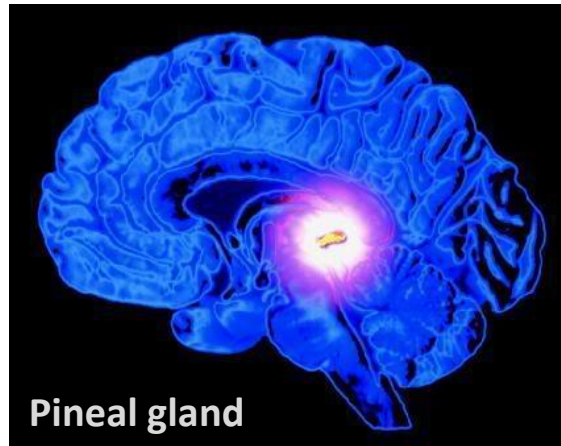
Melatonin



Pineal gland

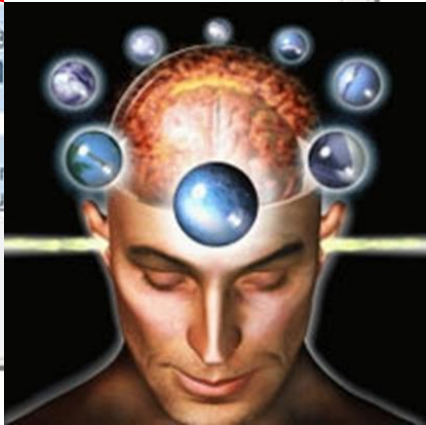
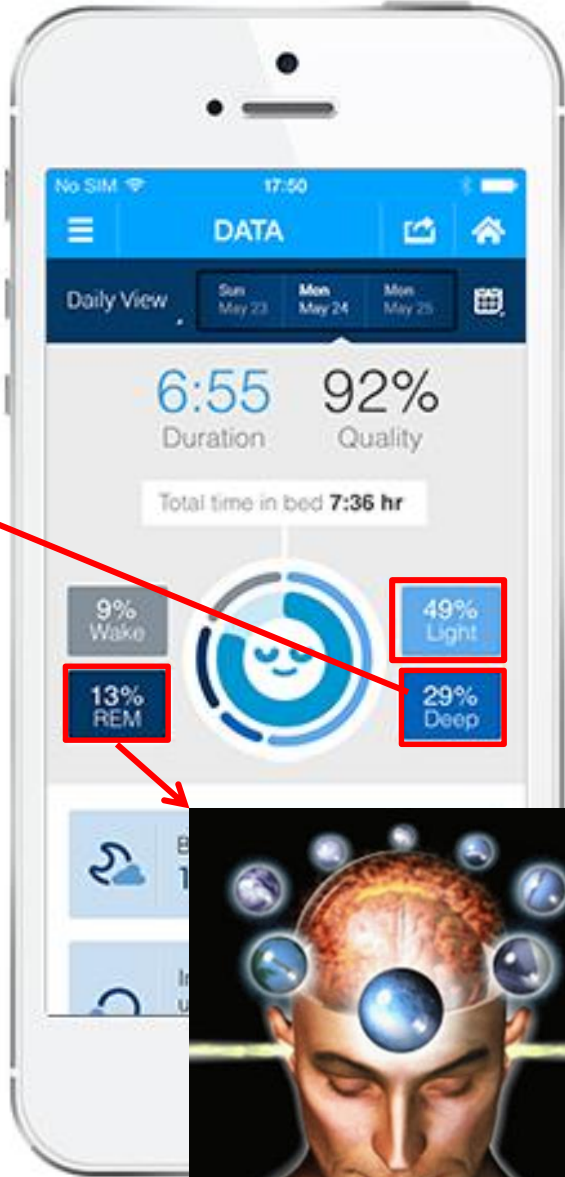
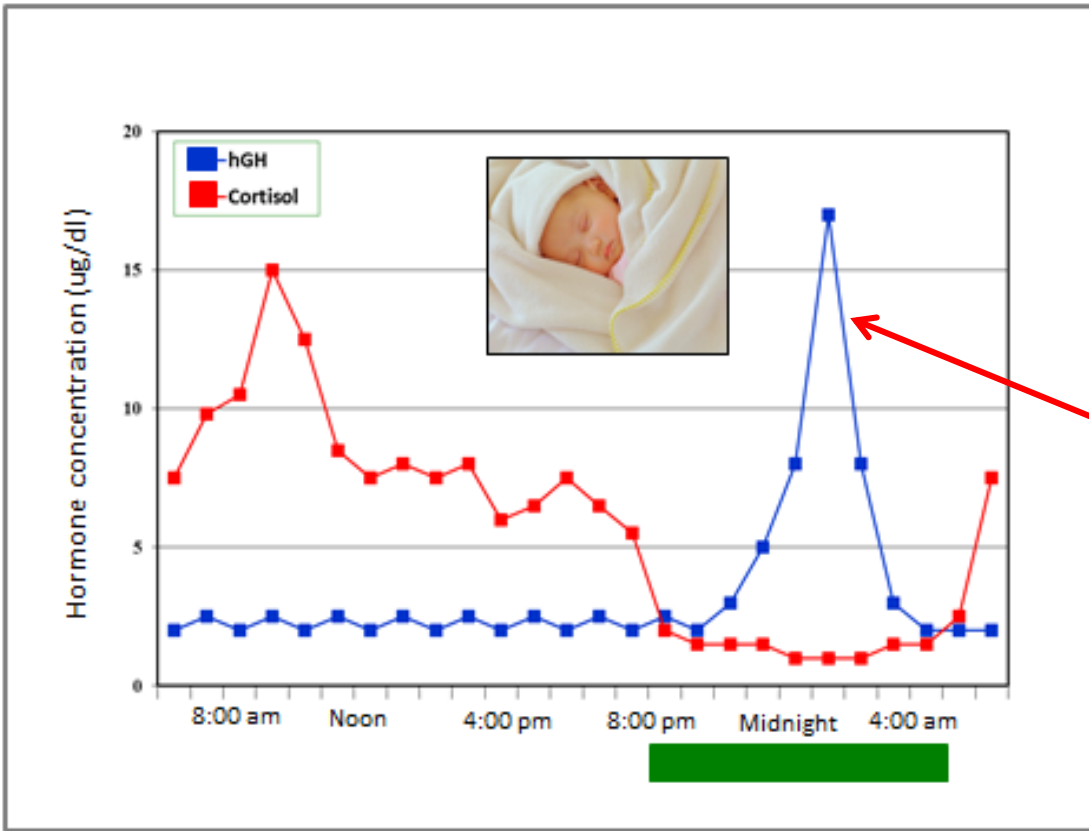


Melatonin



Melatonin





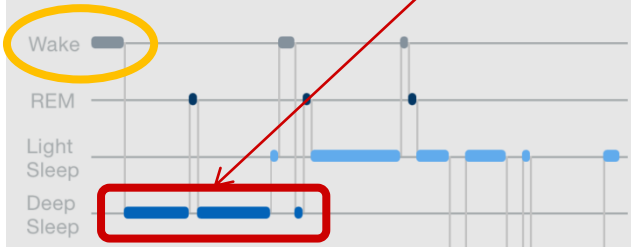
USA SWIMMING
 LTE 9:44 AM 35%
 DATA
 Home icon
 Daily View Fri Feb 27

7:37 87
 Duration Quality

Total Time Tracked 8:31 Hours



SLEEP STAGES



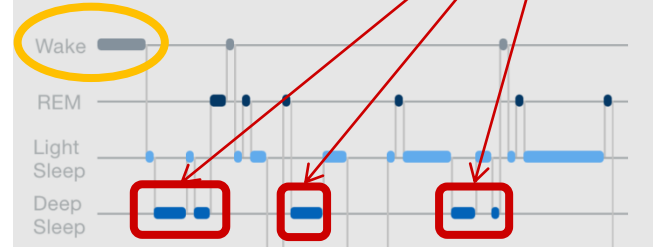
USA SWIMMING
 8:41 AM 100%
 DATA
 Home icon
 Daily View Thu Feb 26

6:37 85
 Duration Quality

Total Time Tracked 7:34 Hours



SLEEP STAGES

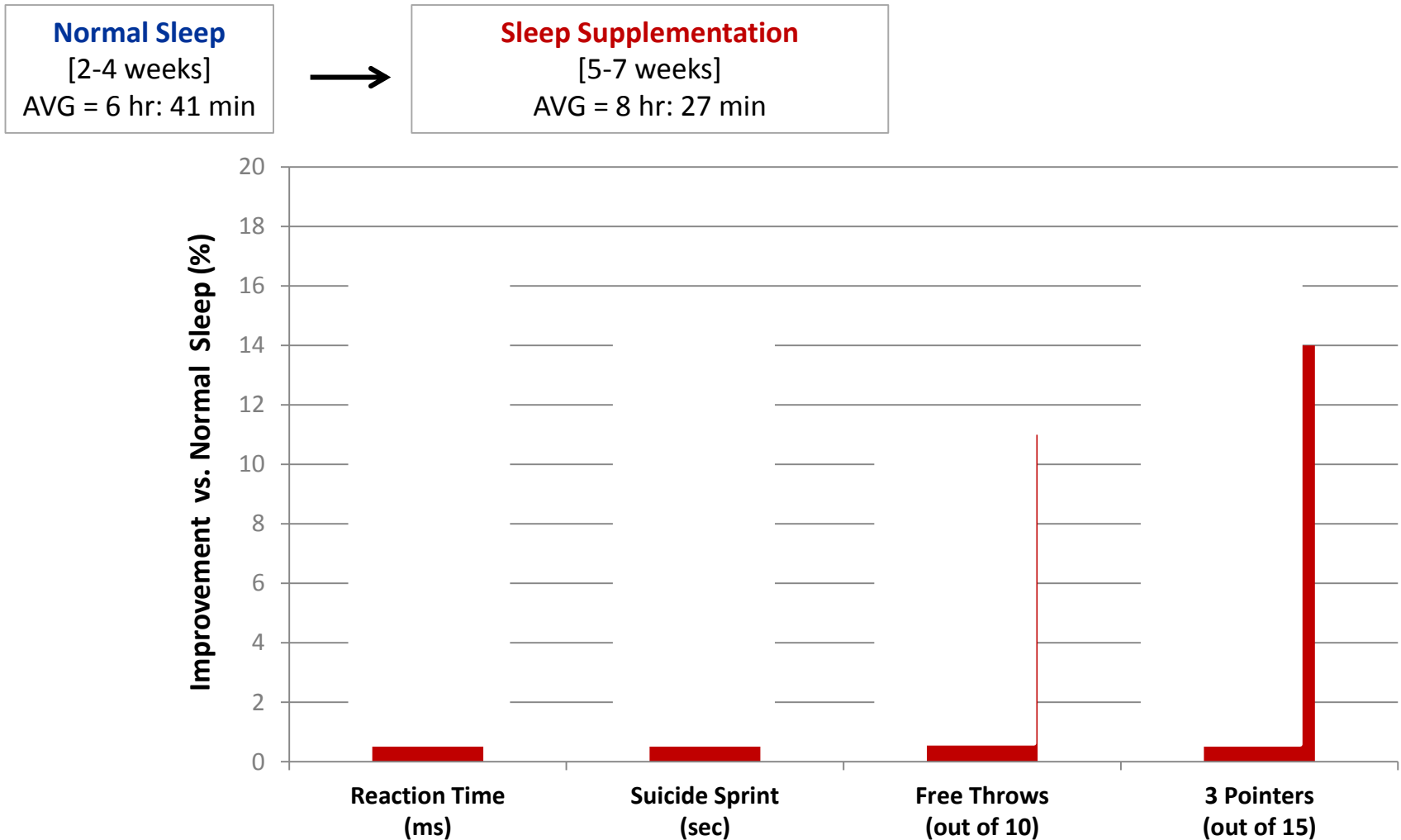


The Effects of Sleep Extension on the Athletic Performance of Collegiate Basketball Players



Cheri D. Mah, MS¹; Kenneth E. Mah, MD, MS¹; Eric J. Kezirian, MD, MPH²; William C. Dement, MD, PhD¹

Sleep 34: 943-950, 2011



THE JUNIOR ELITE ATHLETE:

Physiological Characteristics and Training Considerations

- Introduction / Physiology 101
- From Childhood to Adolescence to Adulthood
- Increased Risk of Injury
- Training Considerations
- ➔ ■ Overtraining
- Summary and Q/A



Cytokine Hypothesis of Overtraining / Under-Performance

Dr. Lucille Lakier Smith

BASIC SCIENCES
Reviews

Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress?

LUCILLE LAKIER SMITH

Department of Health, Leisure, and Exercise Science, Appalachian State University, Boone, NC 28608

ABSTRACT

SMITH, L. L. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? *Med Sci Sports Exerc*, Vol. 32, No. 2, pp. 317-331, 2000. Overtraining syndrome (OTS) is a condition wherein an athlete is training excessively, yet performance deteriorates. This is usually accompanied by mood/behavior changes and a variety of biochemical and physiological alterations. Presently, there is no global hypothesis to account for OTS. The present paper will attempt to provide a unifying paradigm that will integrate previous research under the rubric of the cytokine hypothesis of overtraining. It is argued that high volume/intensity training with insufficient rest will produce muscle and/or skeletal and/or joint trauma. Correlating symptoms are then attributed by injury-related cytokines, and as time passes large quantities of proinflammatory IL-1 β and IL-6, and/or TNF- α produce systemic inflammation. Elevated circulating cytokines then coordinate the whole-body response by (a) communicating with the CNS and inducing a set of behaviors referred to as "altered" behaviors, which involve mood and behavior changes that suggest resolution of systemic inflammation; (b) adjusting liver function, to support the up-regulation of glycoproteins, as well as *de novo* synthesis of acute phase proteins, and a concomitant hypercatabolic state; and (c) impacting on immune function. Thereafter, OTS is viewed as the final stage of what's termed adaptation syndrome, with the focus being on recovery/repair, and not adaptation, and is deemed to be "protective," occurring in response to excessive physical/physiological stress. Recommendations are made for potential markers of OTS, based on a systemic inflammatory condition. Key Words: INTERLEUKIN-1 β , INTERLEUKIN-6, TUMOR NECROSIS FACTOR- α , ACUTE PHASE PROTEIN, TISSUE TRAUMA

The purpose of this paper is to integrate available information pertaining to the overtraining syndrome (OTS) into one paradigm, which will be referred to as the cytokine hypothesis of overtraining. The following hypothesis is not presented as complete but is advanced in an attempt to focus future research efforts. For brevity, references are generally limited to review articles. The predominant focus of this paper will be on the systemic immune/inflammatory response. These terms are frequently used interchangeably due to their extensive overlap; for consistency, the term systemic inflammation will be used.

Athletes train hard to optimize performance. Inherent in all training programs is the application of the progressive overload principle, which implies working beyond a comfortable level in order to maximize athletic ability

(26,27,45,91). Unfortunately, there is a fine line between improved performance and deterioration. When deterioration in performance occurs in association with an arduous training schedule, it is referred to as overtraining, staleness, or burnout (66).

The universal criterion associated with overtraining is a decrease in performance. However, not all aspects of performance are affected simultaneously nor are they impacted to the same degree, making prediction and/or interpretation confusing (66). It is also probable that other signs/symptoms typically associated with overtraining are evident before a deterioration in performance. These might include generalized fatigue, depression, muscle and joint pain, and loss of appetite. However, it is the decline in performance frequently associated with an increased volume or load of training, that captures the attention of the athlete and coach. A large number of symptoms associated with overtraining, have been reported in the literature. Fry et al. (27) have categorized these according to physiological performance, psychological information processing, immunological, and biochemical parameters (see Table 1). However, there is no

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REVIEW ARTICLE

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Overtraining, Excessive Exercise, and Altered Immunity Is This a T Helper-1 Versus T Helper-2 Lymphocyte Response?

Lucille Lakier Smith

Department of Sport and Physical Rehabilitation Sciences, Technikon Pretoria, Pretoria, South Africa

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Abstract

Overtraining syndrome (OTS) occurs where an athlete is training vigorously, yet performance deteriorates. One sign of OTS is suppressed immune function, with an increased incidence of upper respiratory tract infections (URTI). An increased incidence of URTI is also associated with high volume/intensity training, as well as with excessive exercise (EE), such as a marathon, manifesting between 3-72 hours post-race. Presently, there is no encompassing theory to explain EE and altered immune competence. Recently, it has been conclusively established that T helper lymphocytes (T $_H$), a crucial aspect of immune function, represent two distinct functional subsets: T $_H$ 1 and T $_H$ 2 lymphocytes. T $_H$ 1 lymphocytes are associated with cell-mediated immunity (CMI) and the killing of intracellular pathogens, while T $_H$ 2 lymphocytes are associated with humoral immunity and antibody production. When T $_H$ 1 precursor cells are activated, the balance is tipped in favour of one or the other. Furthermore, the most appropriate means of determining the T $_H$ 1-subset, is by the prevailing cytokine "pattern". This paper hypothesizes that exercise-related immunosuppression is due to tissue trauma sustained during intense exercise, producing cytokines, which drive the

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Brief Review

TISSUE TRAUMA: THE UNDERLYING CAUSE OF OVERTRAINING SYNDROME?

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ABSTRACT: Smith, L.L. Tissue trauma: the underlying cause of overtraining syndrome? *J Strength Cond Res* 18(1):185-193, 2004.—An athlete who trains intensely, yet consistently underperforms, is considered to be suffering from overtraining syndrome (OTS). OTS is a complex state that involves a large variety of signs and symptoms. Symptoms include changes in mood or behavior, decrease or increase in concentration of different blood molecules, and alterations in immune function. Although several hypotheses have been proposed, each only explains a selective aspect of OTS. Presently, the sole agreement is that OTS is associated with excessive training and insufficient rest and recovery. The hypothesis proposed in this paper suggests that excessive training/competing causes repetitive tissue trauma, either to muscle and/or connective tissue and/or to bony structures, and that this results in chronic inflammation. It is further proposed that traumatized tissue synthesizes a group of inflammatory molecules, cytokines. Cytokines have been shown to coordinate the different systems of the body to promote recovery. Suggestions are made to detect, prevent, and rehabilitate the overtrained athlete.

Key Words: burnout, staleness, interleukin, tumor necrosis factor, depression

PURPOSE

The purpose of this paper is to propose an underlying mechanism that drives the condition referred to as overtraining syndrome (OTS). This will be achieved by integrating information from a variety of different areas of research and proposing a unifying hypothesis that will attempt to explain many of the biochemical, psychological, physiological, hormonal, and immune changes that have previously been associated with OTS. For the sake of brevity, where possible, review articles are referenced.

Although a majority of studies have demonstrated OTS in endurance-trained athletes (27, 29, 35, 38), this condition may equally impact anaerobically/resistance-trained athletes, with some variations (28, 27, 64, 73), although this issue remains uncertain (1, 29, 35, 73). However, since a typical training program incorporates many different aspects of training, including high-intensity resistance exercise, high-volume resistance exercise, interval/sprint training, and endurance training, a distinction will not be drawn between different aspects of training/competing and the induction of OTS. Additionally, it is proposed that OTS occurs in athletes as well as in lay athletes, but, for obvious reasons, is not closely monitored in these cases.

INTRODUCTION

OTS, also known as "staleness" or "burnout" is a serious condition that affects many athletes, often in the prime

of their athletic careers. By definition, it occurs when an athlete is training intensely, but, instead of improving, shows a deterioration in performance, even after an extended rest period (1, 26, 27, 29, 47, 55, 74). This sports-specific decrease in performance, be it in individual or team sports (56), is regarded as the gold standard, and may extend over a period of weeks or months (39), while in some instances, the athlete may never recover (1, 27, 55). There are numerous signs and symptoms associated with OTS (Table 1) (1, 29, 55, 73, 75). These may be grouped into four categories: psychological, physiological, biochemical, and immunological. However, athletes manifest different combinations of these signs and symptoms, with varying degrees of severity (56). Presently, there do not appear to be specific patterns of signs and symptoms that can be associated with specific sporting events (56). However, it has been suggested that there may be distinct differences between sports regarding which signs and symptoms predominate, although to date this has not been clarified (27, 73). Generally, the first indication of impending OTS is a change in mood (1, 45, 47, 73), although it is the decline in performance that usually captures the attention of the athlete and coach.

OTS should be distinguished from the condition of short-term overtraining, typically referred to as "overreaching" or "supercompensation training" (27, 38, 73). "Overreaching" describes an initial temporary deterioration in performance, usually lasting a few days (27). With sufficient rest, athletic performance is regained and frequently there is improvement (26, 27, 73). "Overreaching" is regarded by many as part of the training stimulus/adaptation (27, 73).

CAUSES OF OVERTRAINING SYNDROME?

Notwithstanding the seriousness and prevalence of OTS, and despite the fact that this condition has been recorded as far back as the 1920s (quoted in 55, 73), the underlying causes of OTS remain unclear. A number of hypotheses have been proposed, but for the most part each explains only one aspect of OTS. For example, the diurnal hypothesis suggests that general fatigue and complaints of heavy legs are due to reduced muscle glycogen (13, 70). The central fatigue hypothesis suggests that general complaints of fatigue are due to an excess of a specific amino acid, tryptophan, in the central nervous system (50). The glutamine hypothesis suggests that the prevalence of immune-related disorders is due to reduced blood levels of glutamine, an amino acid crucial to optimal functioning of immune cells (51, 76). There are also additional hypotheses concerning the causes of OTS, including involvement of the sympathetic and parasympathetic nervous system

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MSSE 32: 317-331, 2000

Sports Med 33: 347-364, 2003

JSCR 18: 185-193, 2004



Excessive Musculoskeletal Stress

[2-3 training sessions / day]

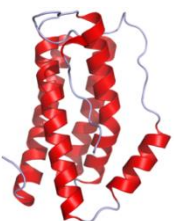


Insufficient Recovery

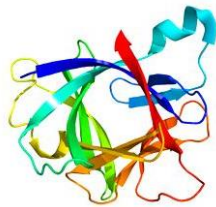
[sleep, nutrition, psychological down time]



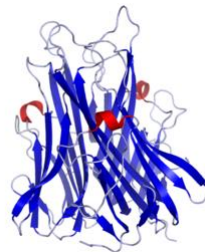
Pro-Inflammatory Cytokines



IL-6



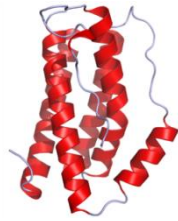
IL-1β



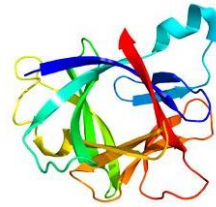
TNF-α

Chronic Musculoskeletal Inflammation

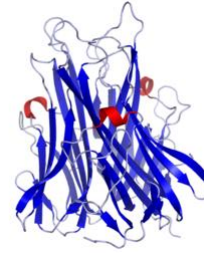
Pro-Inflammatory Cytokines



IL-6

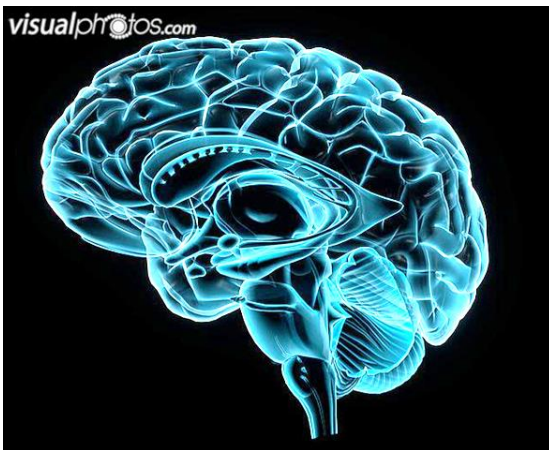


IL-1β



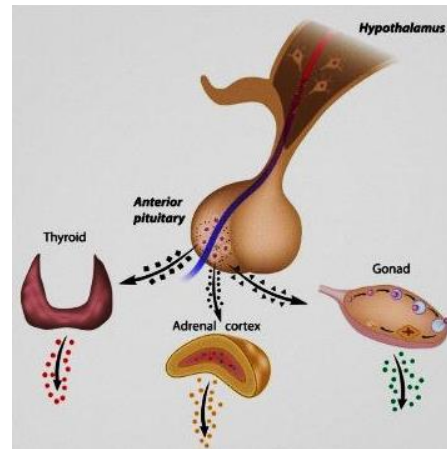
TNF-α

CNS



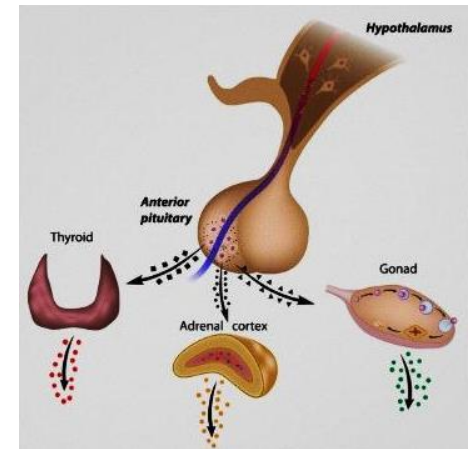
↓ Appetite
Sleep disturbances
Apathy / Depression

HPA axis



↑ Cortisol, E, NE
Immunosuppression

HPG axis



↓ Testosterone, LHRH
↓ Muscle anabolism
↓ Reproductive function

OVERTRAINING Continuum





OVERTRAINING Continuum



Active Recovery

Acute TL

Chronic TL

Chronic TL

OVERTRAINING

Rest
&
Cross-training

WO

Mesocycle
[Moderate V / I]

Mesocycle
[High V / I]

TS = R

TS > R

+

+

+

+ / -

-

FOR

NFOR

OTS

LEGEND

TL = Training Load; WO = Workout; V = Volume; I = Intensity; TS = Training Stimulus; R = Recovery; FOR = Functional Overreaching; NFOR = Non-Functional Overreaching; OTS = Overtraining Syndrome; + = Positive Training; - = Negative Training



SCENARIO 1

“I’m currently under-performing . . . what can I do to get back to 100% before the 2015 season ends?”

ACTION PLAN = Reactive / Therapeutic

1. Comprehensive Health Screen
2. Modify training back to “Active Recovery” Phase
3. Monitor conservative progression from “Active Recovery” back to “Chronic TL”
4. Retroactive inspection of Training Log



SCENARIO 2

“I think I might have overtrained in 2014
... how can I prevent that from happening
again in the 2015 season?”

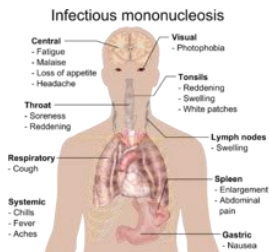
ACTION PLAN = Proactive / Preventive

1. Recognition of OT “risk factors”
2. Robust DB to monitor negative response to “Chronic TL”
3. Meticulous attention to Recovery
4. Execute a scientifically-based taper

2012

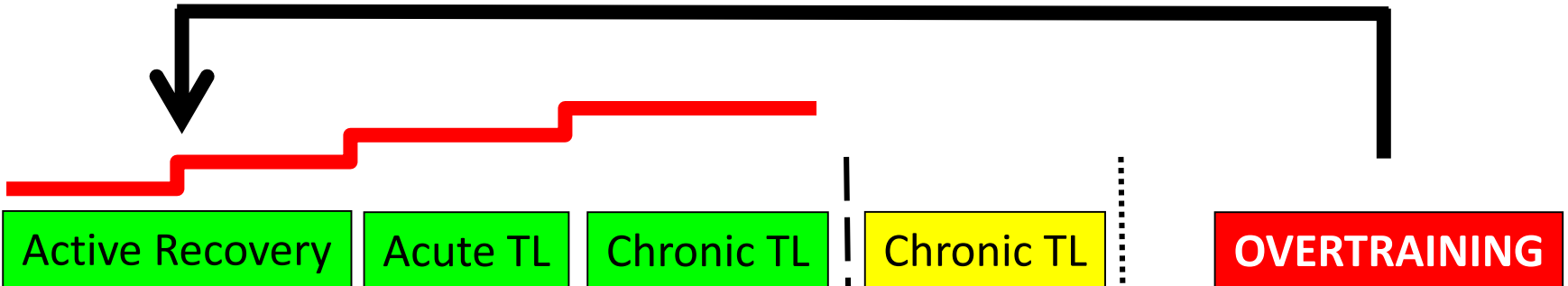


January
Chronic “under performance”
reported by Coach and Athlete



February
Diagnosed with IM

Female
20 yr
2008 Olympian
Modern Pentathlon



Active Recovery

Acute TL

Chronic TL

Chronic TL

OVERTRAINING

Rest
&
Cross-training

WO

Mesocycle
[Moderate V / I]

Mesocycle
[High V / I]



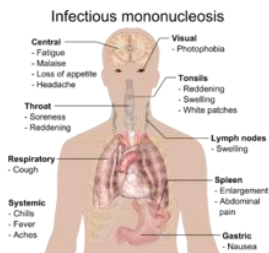
2012



January
Chronic “under performance”
reported by Coach and Athlete



Female
20 yr
2008 Olympian
Modern Pentathlon



February
Diagnosed with IM



June
Resumed normal training



August
4th Place (<1 sec out of Bronze)



September
Junior World Champion: INDIVIDUAL
Junior World Champion: TEAM



THE JUNIOR ELITE ATHLETE:

Physiological Characteristics and Training Considerations

- Introduction / Physiology 101
- From Childhood to Adolescence to Adulthood
- Increased Risk of Injury
- Training Considerations
- Overtraining
- ➔ ■ Summary and Q/A



SUMMARY

1. Diversification . . . not Specialization



SUMMARY

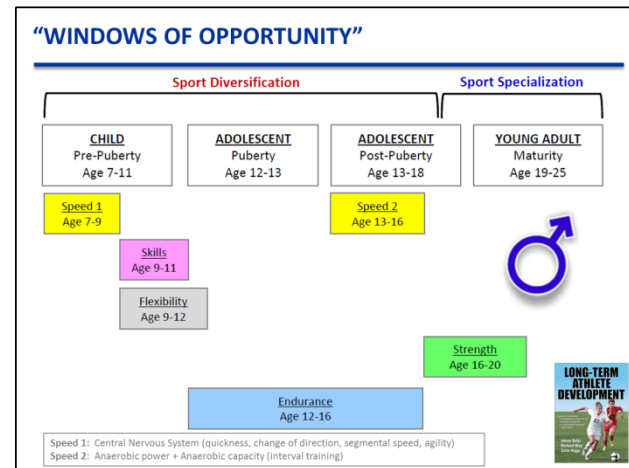
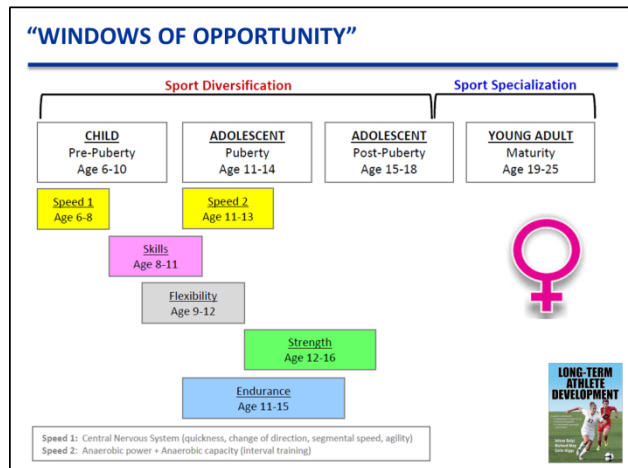
1. Diversification . . . not Specialization

2. Conservative training load (volume, intensity, # days per week)



SUMMARY

1. Diversification . . . not Specialization
2. Conservative training load (volume, intensity, # days per week)
3. Female vs Male “windows of opportunity”



SUMMARY

1. Diversification . . . not Specialization
2. Conservative training load (volume, intensity, # days per week)
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4. Individual variability in growth/maturation . . . “late bloomers”



SUMMARY

1. Diversification . . . not Specialization
2. Conservative training load (volume, intensity, # days per week)
3. Female vs Male “windows of opportunity”
4. Individual variability in growth/maturation . . . “late bloomers”
5. Strategies for injury prevention



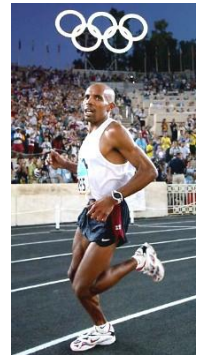
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2. Conservative training load (volume, intensity, # days per week)
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5. Strategies for injury prevention
6. Focus on LT skill development vs ST “instant gratification” in performance



SUMMARY

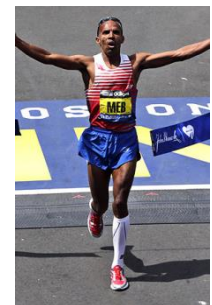
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4. Individual variability in growth/maturation . . . “late bloomers”
5. Strategies for injury prevention
6. Focus on LT skill development vs ST “instant gratification” in performance
7. FUN . . . nurture that original “passion”



Athens
2004

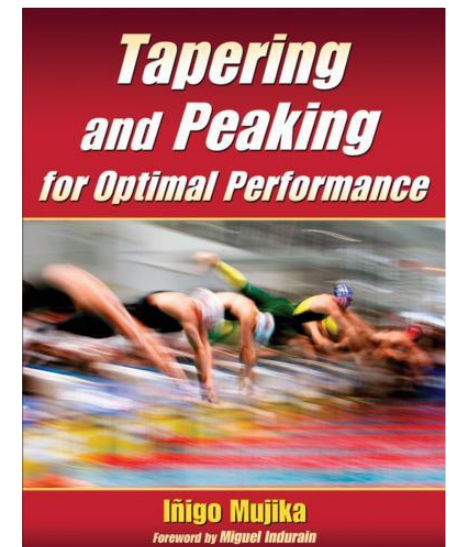
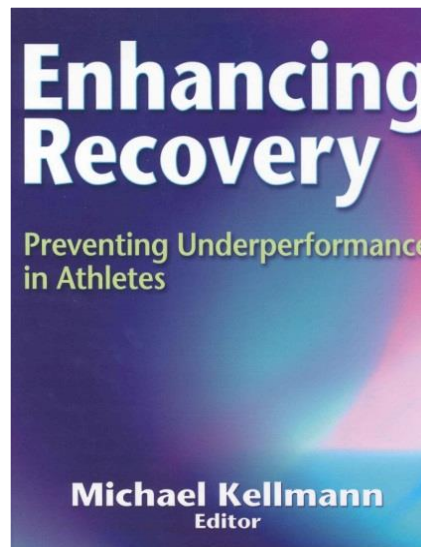
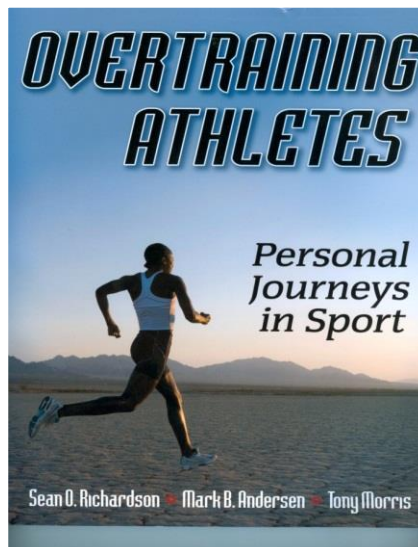
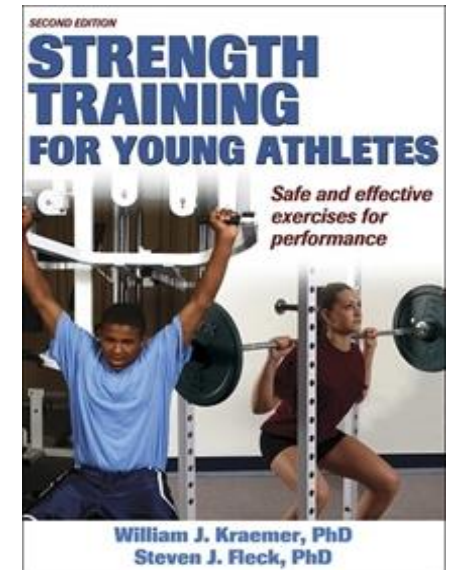
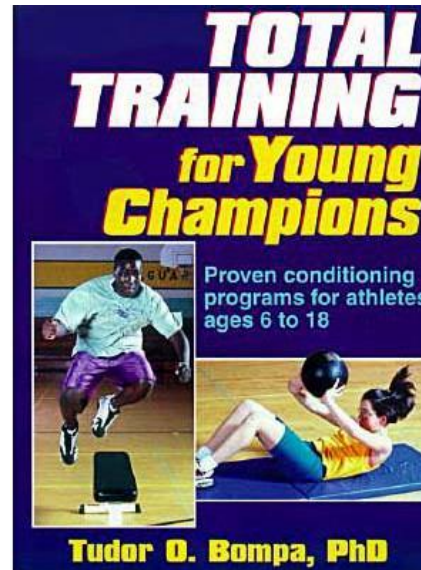
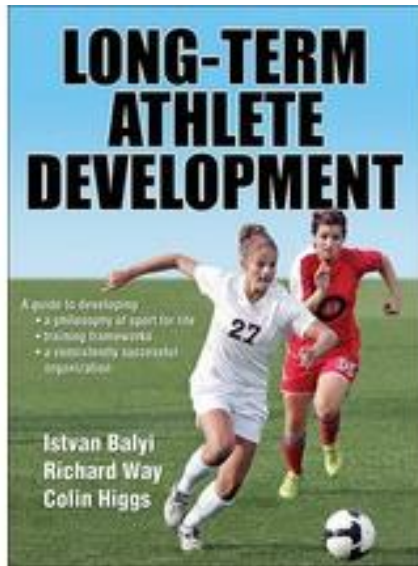


NYC
2009



Boston
2014

RESOURCES





Brenda Martinez

800 m
Bronze
MOSCOW 2013

Matthew Centrowicz

1500 m
Silver
MOSCOW 2013



QUESTIONS?

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