Instagram: Humanhealth.ir

کامل‌ترین دایره‌المعارف شناخت زنان درجهان

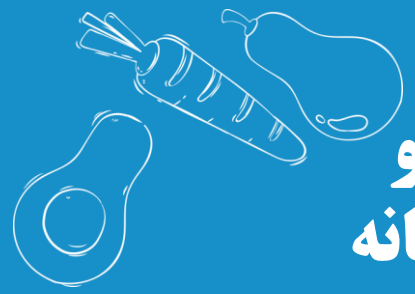
از زبان بسیار ساده تا بسیار پیشرفته

دکتر آرمان رستگاری
محقق حوزه سلامت انسان

بسیار مهم

این وبینار و محتوای ارائه شده، **صرفاً جهت آموزش و اطلاع‌رسانی بوده و به هیچ عنوان جایگزین تشخیص، درمان، یا مشاوره پزشکی نیست.** مسئولیت هرگونه رژیم غذایی، توصیه درمانی، یا مداخلات پزشکی و ورزشی بر عهده متخصصان مربوطه است. اینجانب آرمان رستگاری به عنوان فیزیولوژیست ورزشی و متخصص تغذیه ورزشی، هیچ‌گونه تشخیص پزشکی، نسخه‌نویسی، یا مسئولیت مستقیم در قبال برنامه‌های ورزشی ندارم. لطفاً قبل از هر اقدام، با پزشک یا متخصص تغذیه یا فیزیولوژیست ورزشی مشورت نمایید.

فهرست مطالب



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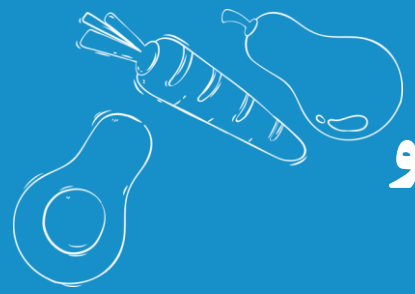

۱۳ ایمنی، غربالگری و الگوریتم ارجاع

انواع ابزار آماده ارائه به پزشک و متخصص روان
انواع چک لیست های روزانه
انواع فرم های غربالگری
آموزش و توانمندسازی
ابزارهای عملی PAR-Q+ ویژه زنان
چک لیست مراقبت بارداری
و ده‌ها فرم قابل چاپ دیگر

فرم غربالگری و ارجاع به فیزیولوژیست ورزشی

اختلالات لیپیدی

این فرم توسط پزشک تکمیل و به فیزیولوژیست ورزشی بالینی ارجاع می‌شود

هدف :تعیین ایمنی، اهداف درمانی لیپیدی و برنامه‌ریزی فعالیت بدنی و ورزشی

مشخصات بیمار

_____ :نام و نام‌خانوادگی _____ :کدملی/شناسه

تاریخ تولد _____/____/____ :سن ____ :جنس :□ زن □ مرد □ دیگر

____ BMI: قد ____ :سانتی‌متر وزن ____ :کیلوگرم

____ :دور کمر ____ :سانتی‌متر دور باسن ____ :سانتی‌متر نسبت کمر/باسن

____ bpm :)میانگین دو بار (نبض استراحت ____ mmHg / ____ :فشار خون نشسته

_____ :آدرس/تلفن

تشخیص و سطح خطر قلبی–عروقی

_____ :دیگر □ HyperTG □ E78.5 □ E78.2 □ E78.1 □ E78.0 □ :ICD-10 (E78.x) تشخیص

ASCVD: □ نوع| □ خیر □ بله :□ MI □ PCI/CABG □ PAD □ Stroke/TIA سابقه

HbA1c: ____ % | دیابت :□ نوع 1 □ نوع 2

بله □ خیر |کبد :□ بله □ خیر □ :(eGFR <60) بیماری مزمن کلیه

خطر کلی :□ خیلی بالا □ بالا □ میانه □ پایین

_____ :معیار/یادداشت

مقدمه

برای سلامت یک زن به کدام سمت باید نگاه کرد؟

Table 1 Female health domains and their definitions

Health domain	Domain abbreviation	Definition
Menstrual and other gynaecological health	D-MG	The health of the menstrual cycle and female reproductive organs and tract.
Preconception/Assisted reproduction	D-AR	Undergoing treatments to assist in becoming pregnant without sexual intercourse.[33]
Pregnancy	D-PR	The condition of being pregnant.[34]
Postpartum	D-PO	Immediately follows childbirth until 2 years* postchildbirth.
Menopause	D-ME	The transitional time between perimenopause and postmenopause, when menstruation surceases.[7]
Breast health	D-BH	The health of the mammary glands.[34]
Pelvic floor health	D-PF	The physical and functional integrity of the pelvic floor unit through the life stages of an individual (male or female).[35]
Breast feeding, parenting and caregiving	D-BP	Providing direct care for another individual who needs help taking care of themselves (eg, a baby, child, the elderly, chronically ill), including suckling milk from a mother's breast.
Mental health†	D-MH	The psychological, emotional and social well-being[36] of an athlete.
Sport environment†	D-SE	The physical and social context within which athletes train and compete.

*Based on WHO breastfeeding recommendation[37] and mental health outcomes.[38]
†This health domain is particularly prevalent but not unique to female athletes only. Consideration should be given to all athletes.

تفاوت‌های فیزیولوژیکی و پاسخ به عوامل محیطی در زنان

- سلامت زنان تنها به دستگاه باروری محدود نمی‌شود؛ در تمام سیستم‌های بدن و درک محیط، تفاوت‌های زیستی و رفتاری میان زنان و مردان وجود دارد که بر پاسخ‌های فیزیولوژیک و نیازهای بهداشتی تأثیر می‌گذارد. تفاوت‌های جنسی (Sex) به ساختار ژنتیکی و هورمون‌ها مربوط است، در حالی‌که جنسیت (Gender) به نقش‌های اجتماعی و الگوهای رفتاری اشاره دارد. در این وبینار، عوامل محیطی مانند فعالیت بدنی، تغذیه، نور، صدا، آلودگی، رنگ، طبیعت و سایر تحریک‌ها بررسی می‌شوند و تفاوت‌های فیزیولوژیکی زنان در هر عامل با دلایل علمی بیان می‌شود تا مخاطبان درک کنند چرا نیازمند رویکرد اختصاصی هستند.

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تفاوت‌های فیزیولوژیکی

سیستم قلبی-عروقی

اندازه قلب و عروق

خروجی قلبی

پاسخ عروقی

ریسک اختلالات

سیستم اسکلتی-عضلانی

جرم عضلانی و تارهای عضلانی

استخوان و عضله

آسیب و بیماری

بیماری‌های خودایمنی

فعالیت ورزشی و سازگاری

پاسخ به تمرین قدرتی

تمرینات استقامتی

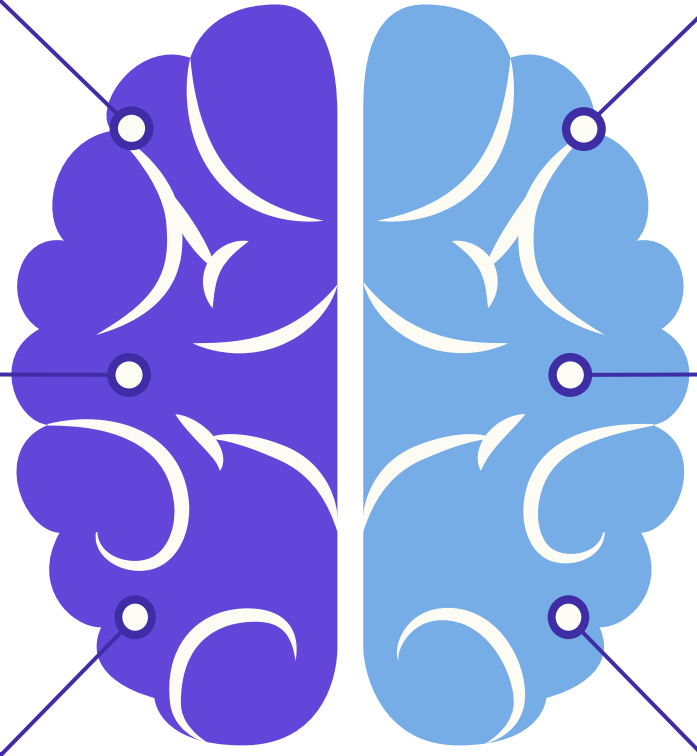
هایپوکسی و ارتفاع

ذخایر آهن

سیستم هورمونی و پاسخ‌های هومورال

دوره‌های قاعدگی

محور HPA و استرس



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تفاوت‌های فیزیولوژیکی

سیستم ایمنی

تغذیه و پاسخ‌های سوخت‌وساز

سوخت‌وساز چربی و گلوکز
درک مزه

حس‌ها و ادراک

بویایی
چشایی
دید رنگی
شنوایی و صدا
درد و درد مزمن

خواب، ریتم شبانه‌روزی و نور

ریتم شبانه‌روز و ملاتونین
حساسیت به نور

تفاوت‌های فیزیولوژیکی

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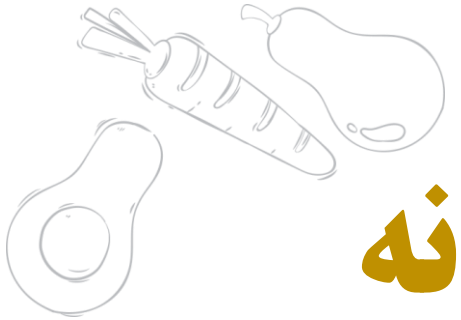
تنظیم حرارت و واکنش به گرما و سرما

- گرما و عرق
- سرما و پاسخ‌های خودکار

آلودگی هوا و عوامل محیطی

طبیعت و فضای سبز



بخش ۱
مبانی فیزیولوژی و نوروواندوکرین زنانه

- در طول تاریخ مطالعات فیزیولوژی و پزشکی اغلب بر مردان متمرکز بوده‌اند و داده‌های زنانه کمتر بوده‌اند. به همین دلیل، بسیاری از استانداردهای پزشکی بر مبنای داده‌های مرد پایه‌گذاری شده‌اند که ممکن است در زنان دقیق نباشند. (مثلاً در تمرین‌درمانی، دارورسانی، الگوی هورمونی)

محورهای اصلی نوروآندوکراین در زنان

Hypothalamus – Pituitary – somatotropic

Hypothalamus – Pituitary – thyroid

Hypothalamus – Pituitary – Adrenal

Hypothalamus – Pituitary – Gonadal

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Hypothalamus – Pituitary – Gonadal

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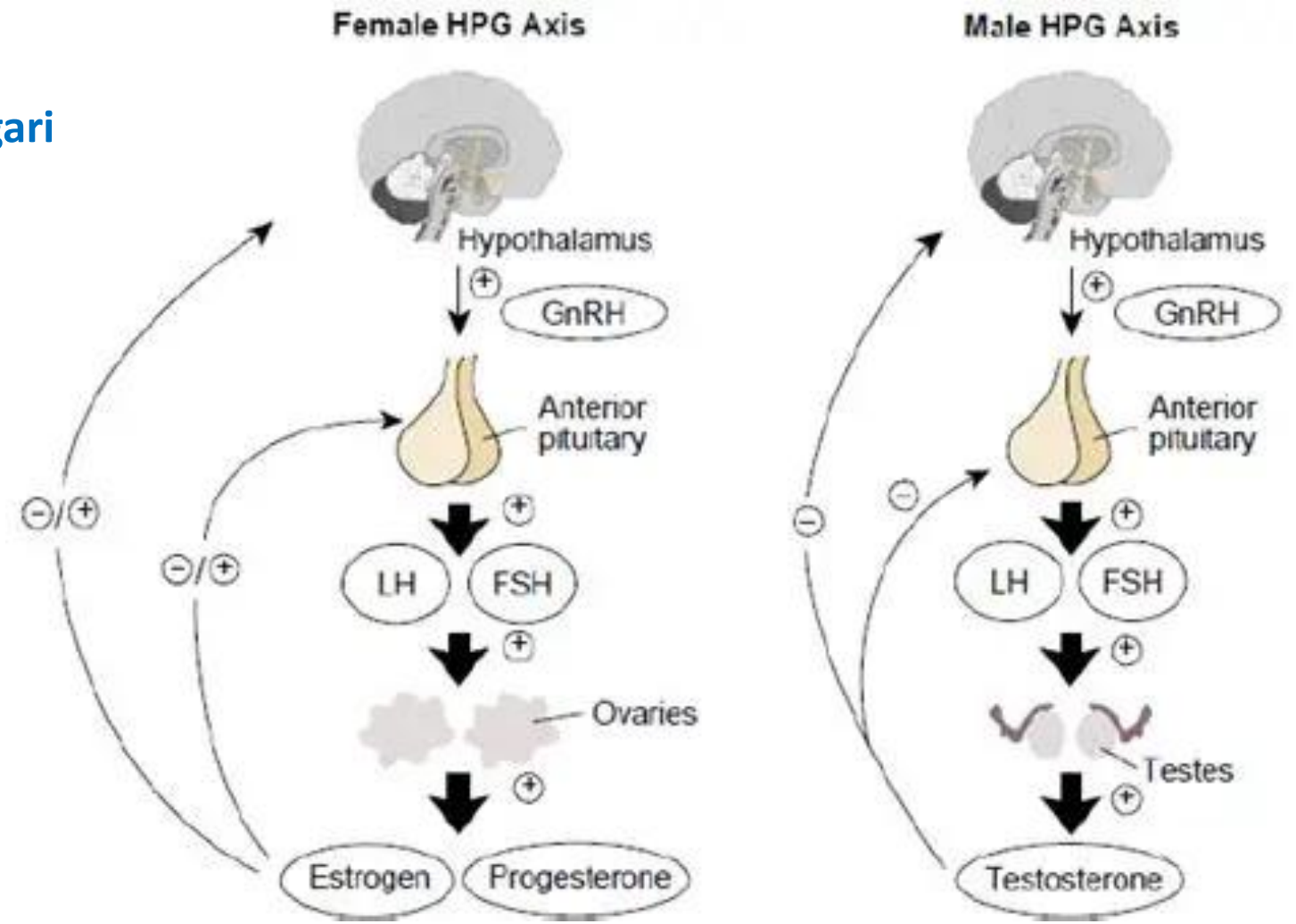
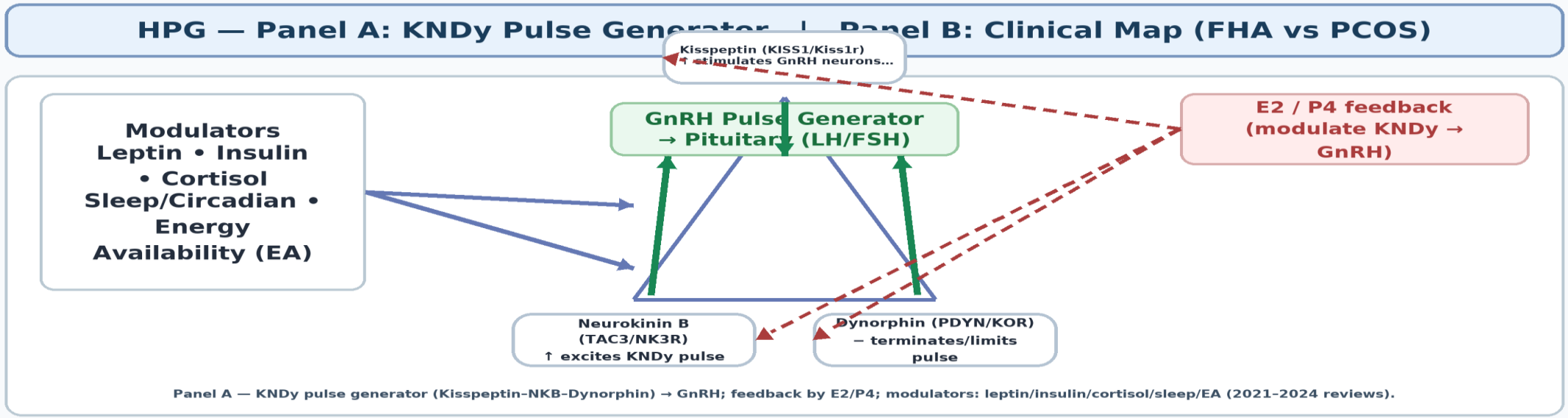
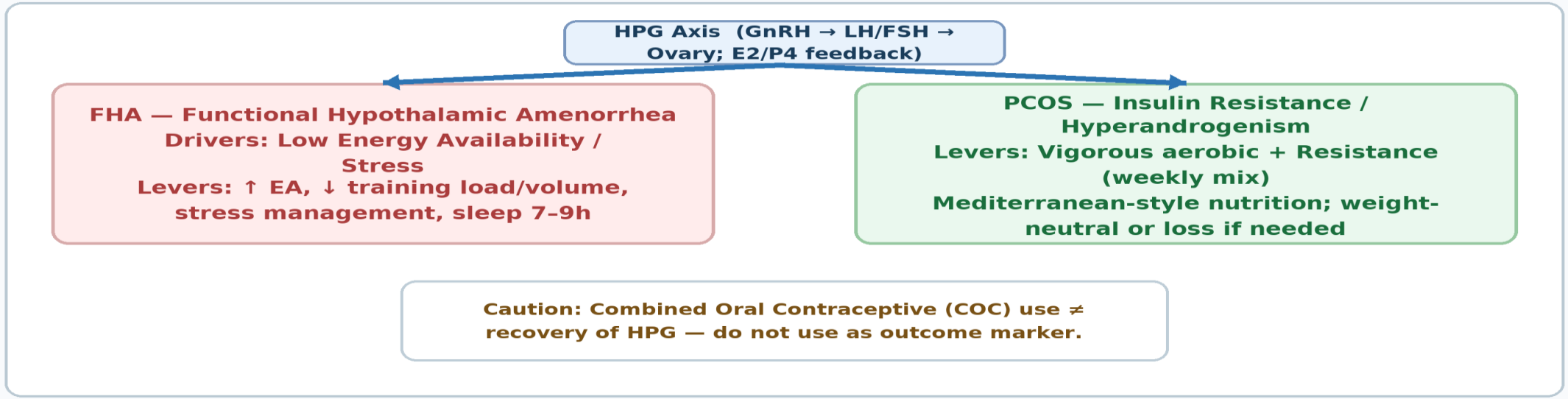


فیزیولوژی محور HPG





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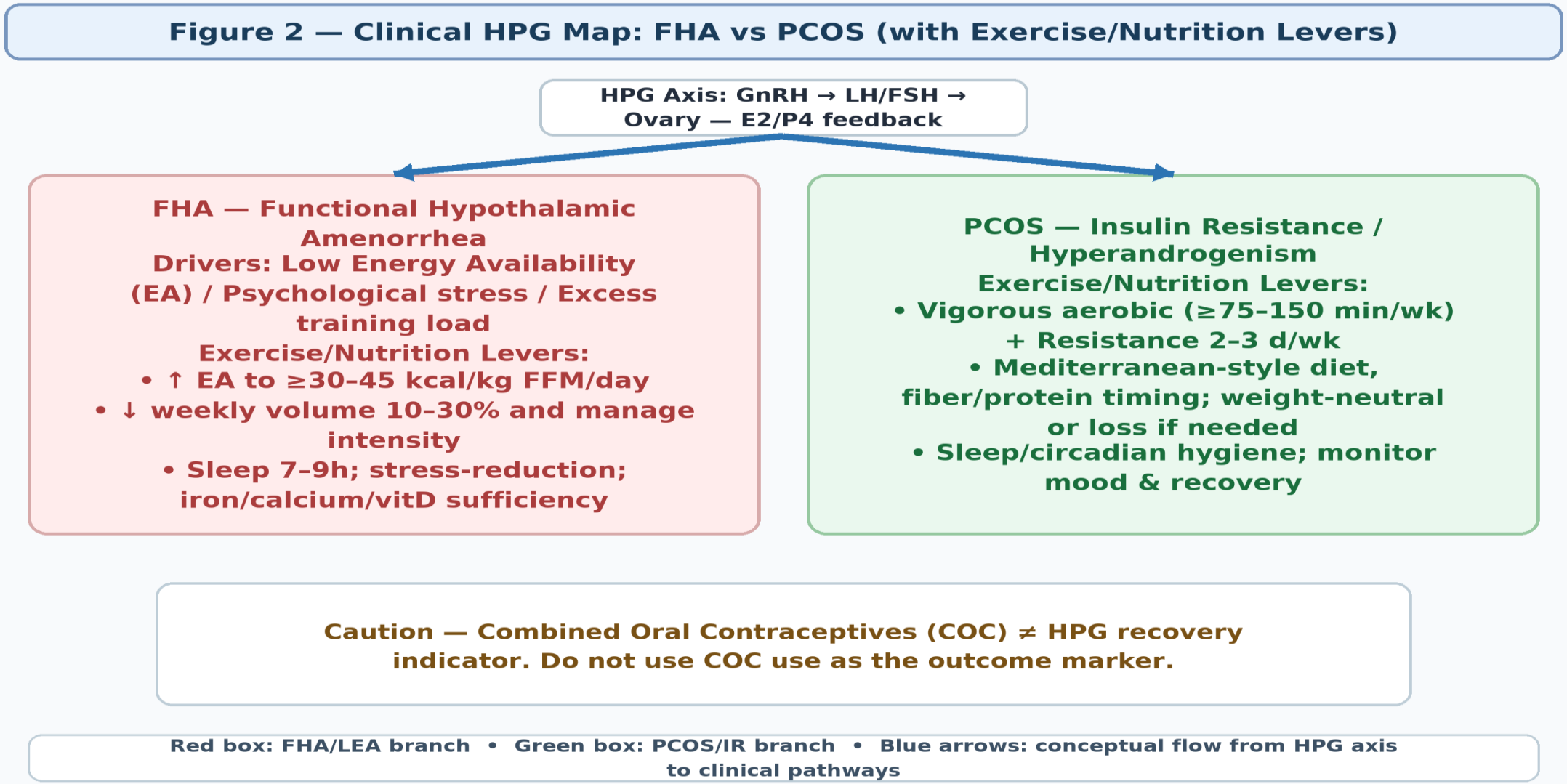
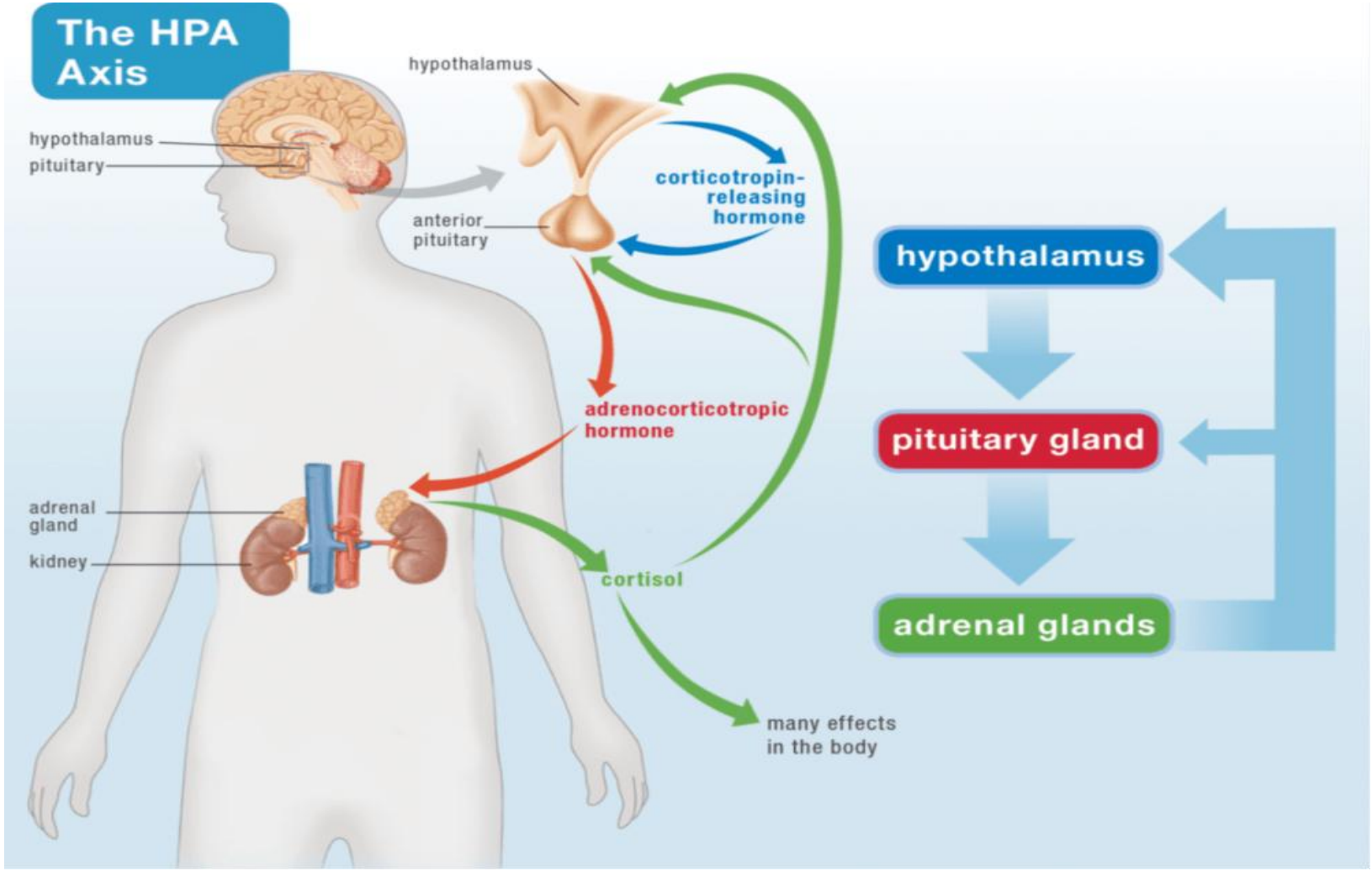
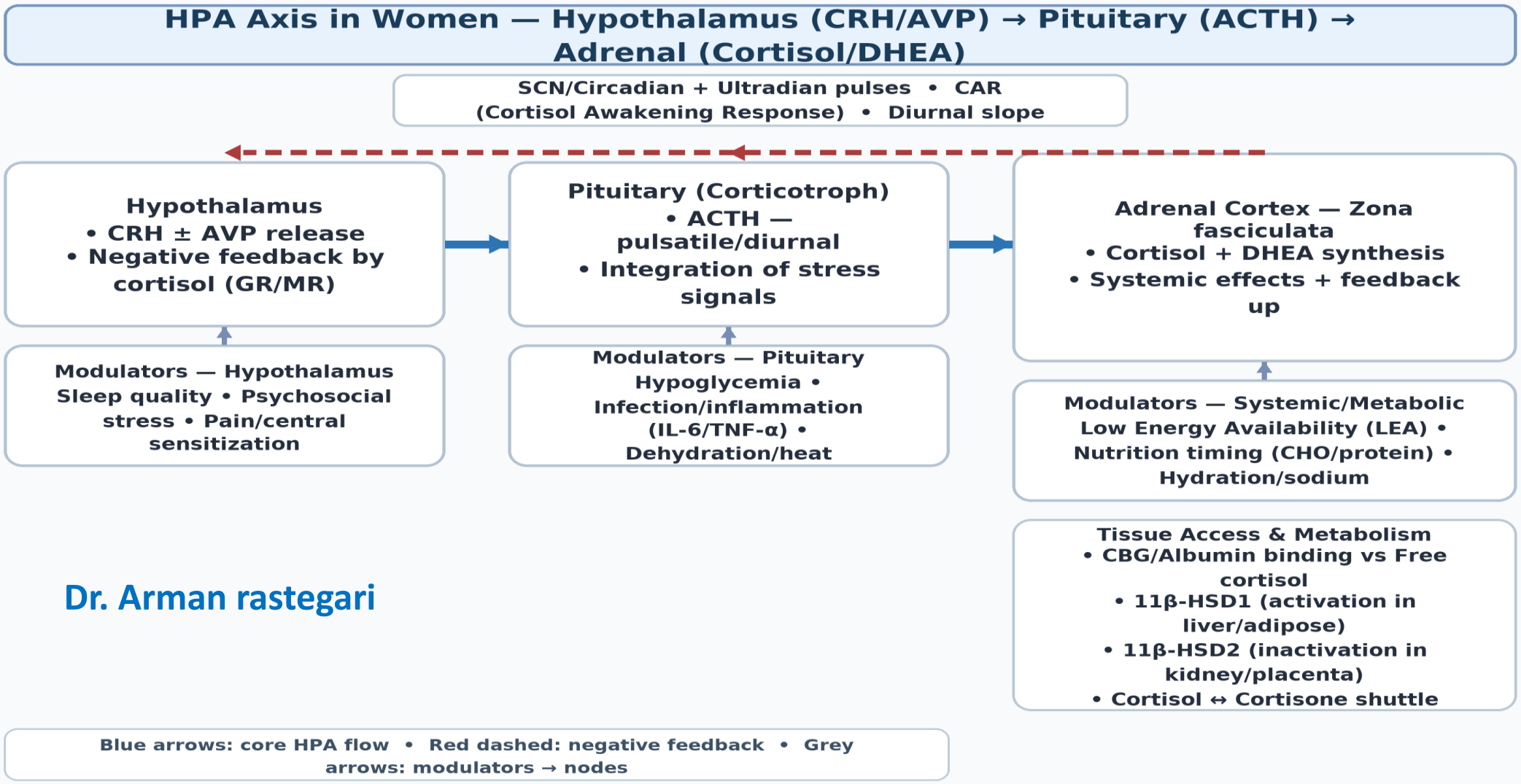
نقشه بالینی PCOS و آمنوره عملکردی با ورزش و تغذیه



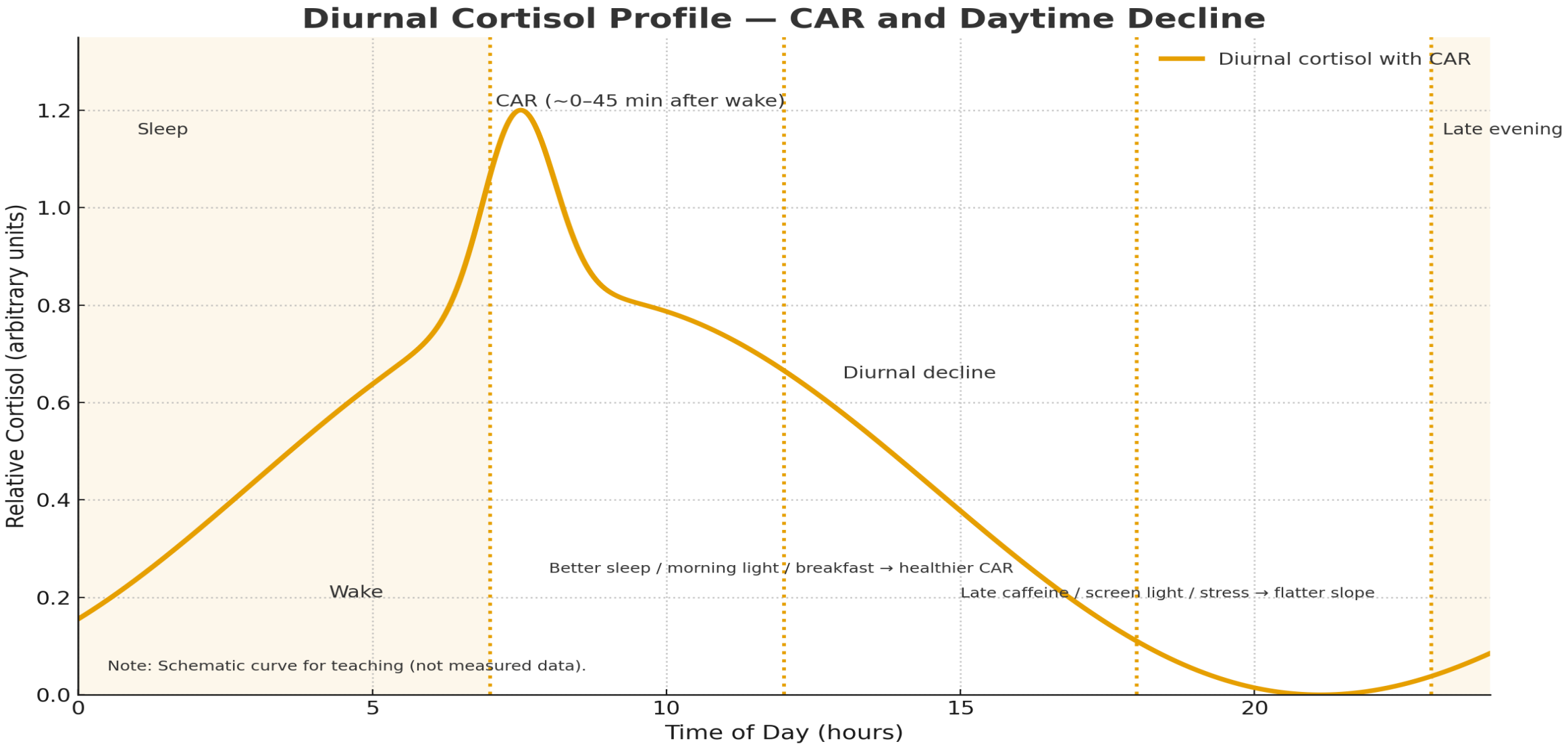
Hypothalamus – Pituitary – Adrenal



نقشه محور HPA در زنان



پروفایل کورتیزول روزانه



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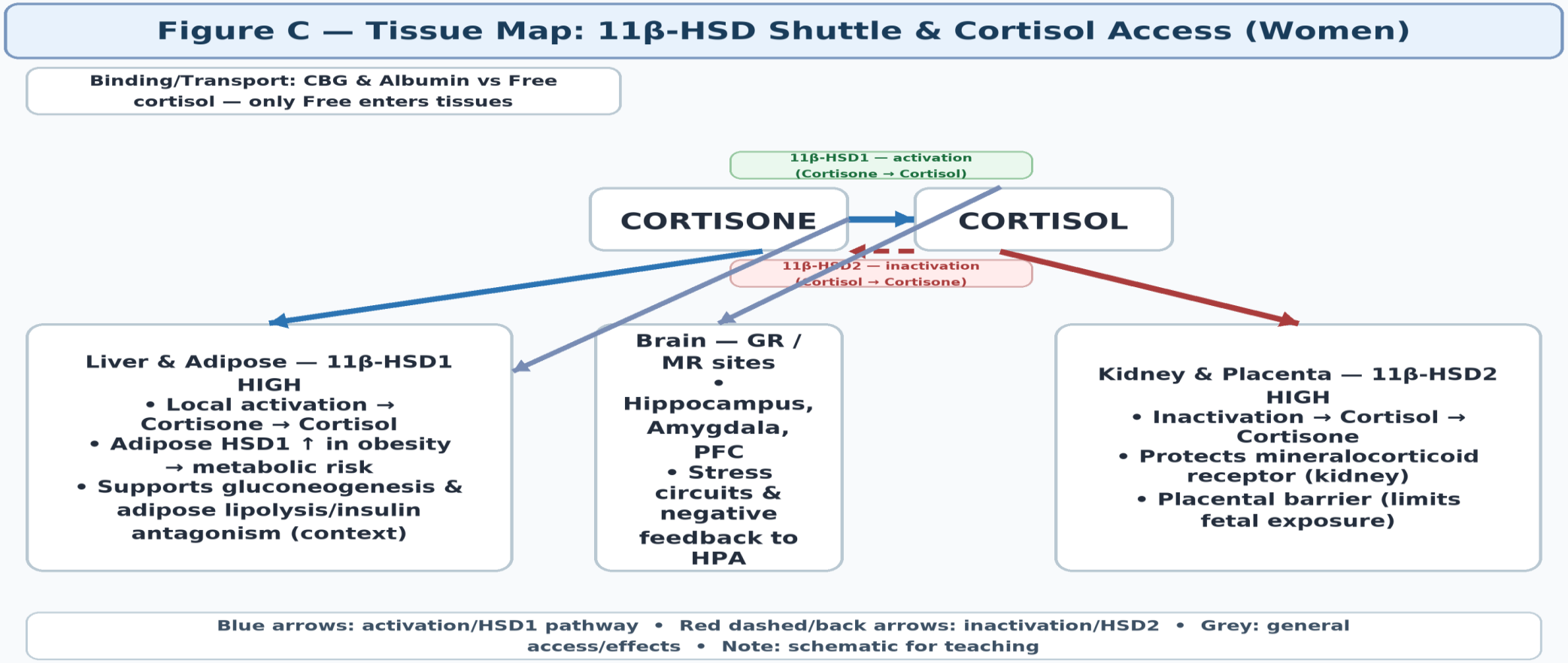
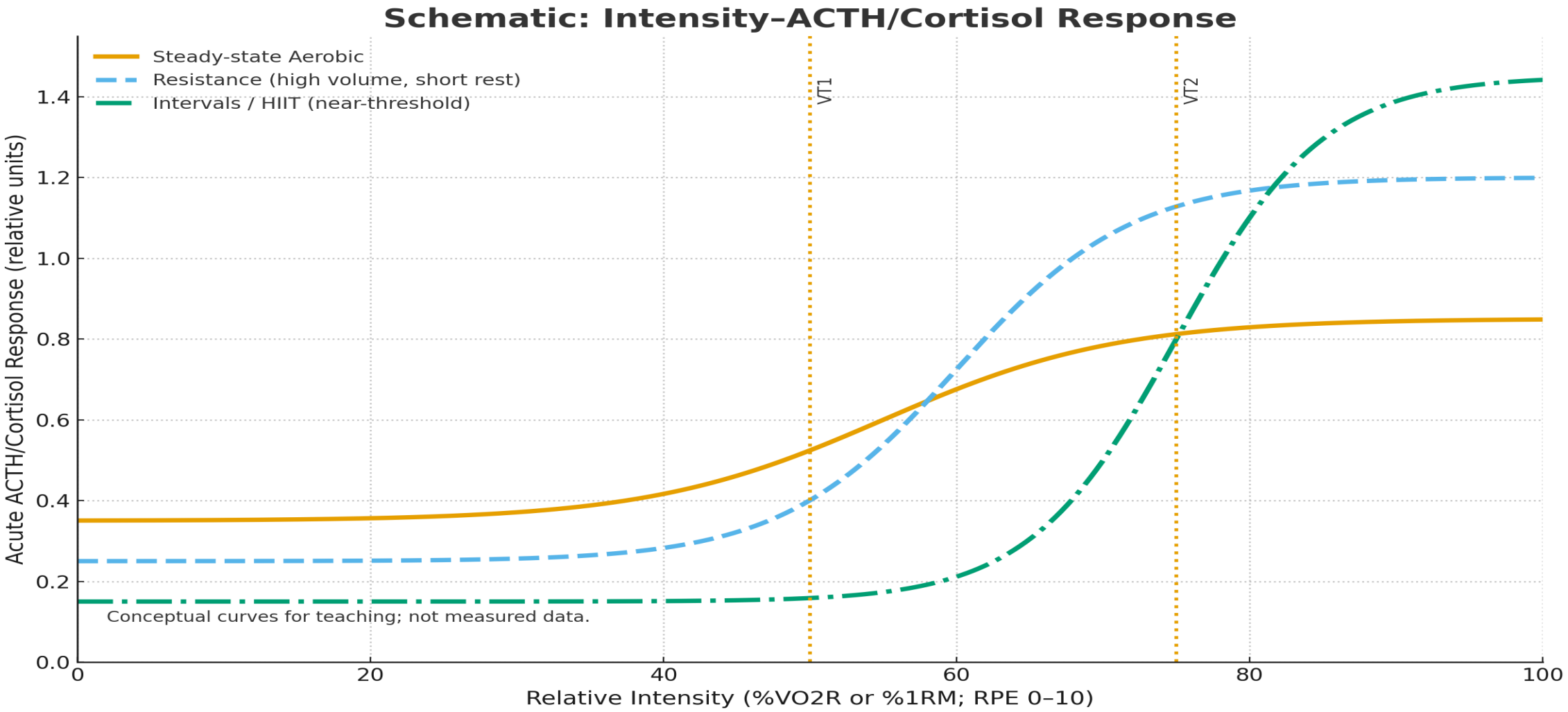
اثر اندوکراین کورتیزول



پاسخ ACTH و کورتیزول به شدت ورزشی



نردبان مداخله در به هم ریختگی محور HPA

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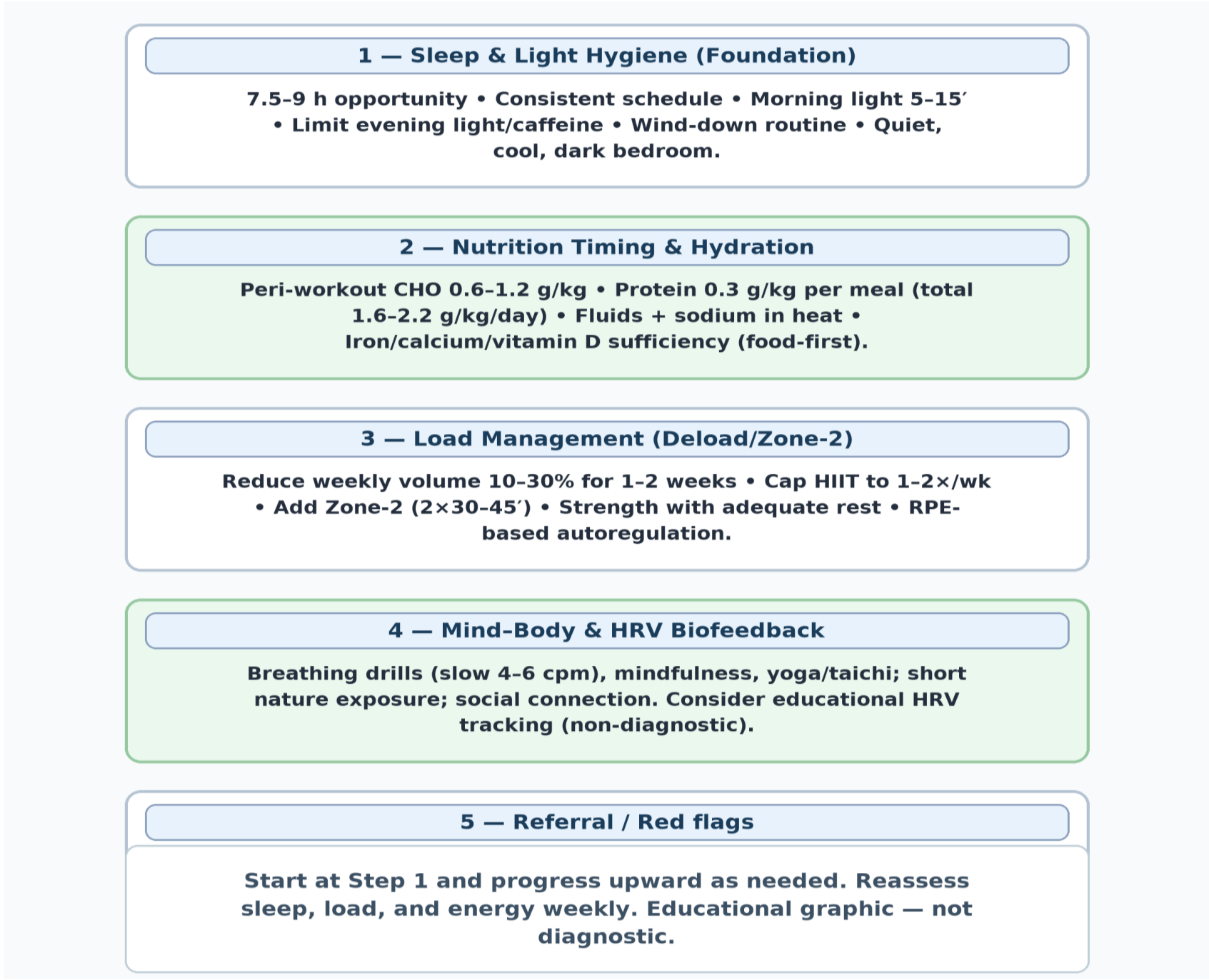

محور HPA در حاملگی، پساحاملگی و منوپاز

Figure F — HPA Axis Across Female Life Stages: Pregnancy, Postpartum, Menopause

Pregnancy

- Placental CRH & ACTH ↑
- CBG & total cortisol ↑, free stable
- Fetal protection via 11β-HSD2
- Maternal adaptation: ↑ energy & stress tolerance
- Exercise: moderate, thermoregulated, hydration emphasis

Postpartum

- Placental CRH removed → axis reset
- Blunted CAR common weeks 1–6
- Sleep fragmentation → stress reactivity ↑
- Gradual load return, social support, morning light
- Nutrition: iron, DHA, hydration, small meals

Menopause

- Estrogen ↓ → GR sensitivity ↑ (variable)
- Sleep/thermoregulation disturbances
- Cortisol rhythm flattening possible
- Exercise: combined aerobic+resistance; mind-body adds resilience
- Nutrition: protein adequacy, phytoestrogens, vitamin D

Each stage modifies HPA drive, cortisol metabolism, and exercise/nutrition priorities. Educational schematic, not diagnostic.

چک‌لیست اجرا

- پایش (هفتگی):
- خواب: زمان ثابت خواب/بیداری ۳۰±min؛ کیفیت (۱–۵)
- استرس ادراک‌شده (۰–۱۰) + یادداشت محرک اصلی
- RPE تمرین، HRV یا روند نبض صبح
- کافئین: مقدار/ساعت آخر مصرف
- «علائم چرخه» (PMS/PMDD) یا وضعیت پسازایمان
- نسخه‌ی تمرین (۳–۵ روز/هفته):
- ۲–۴× MICT (20–40) دقیقه، RPE (11–14)

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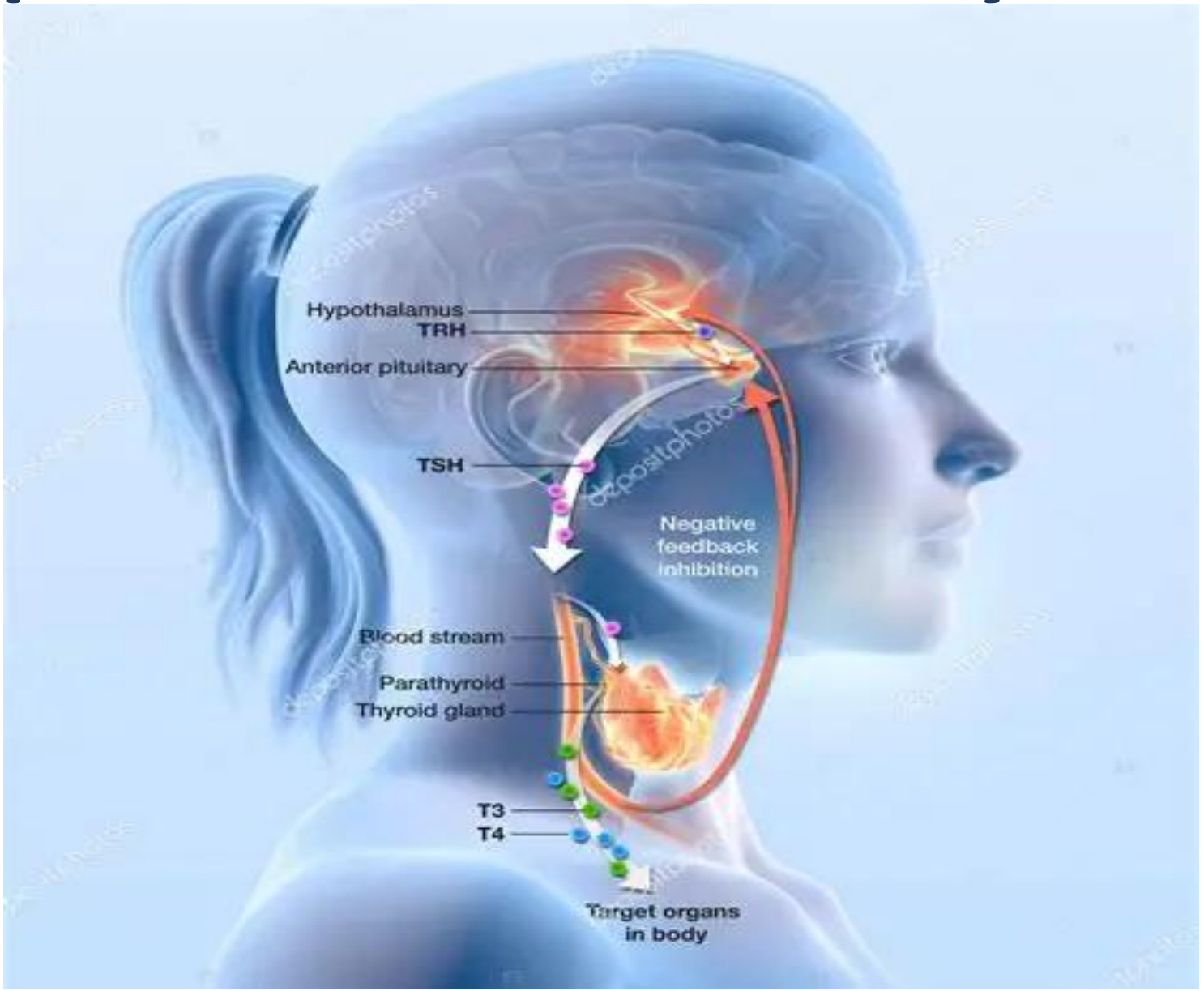
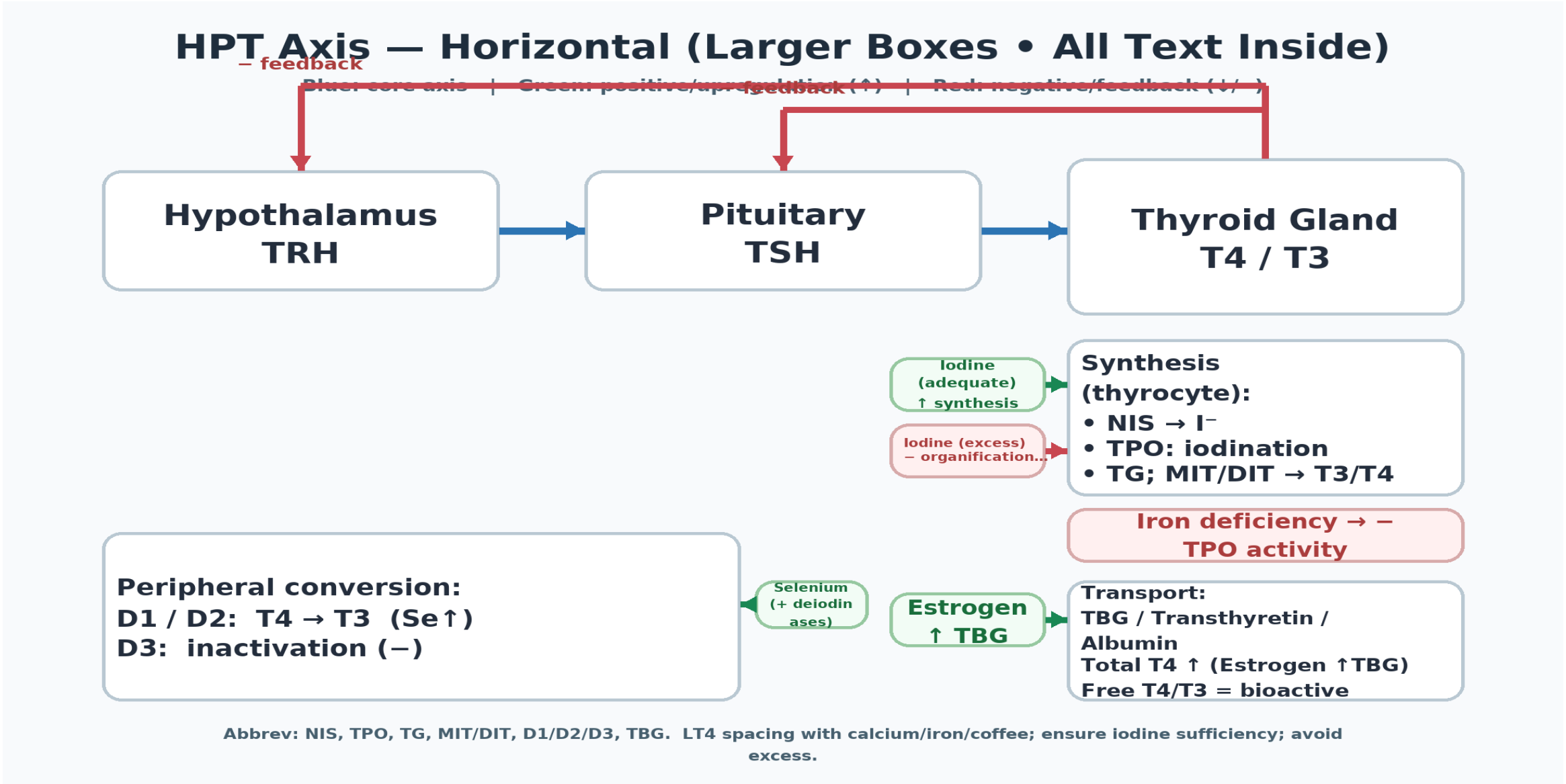
- ۲× مقاومتیِ تمام‌بدن (۶–۸ حرکت، ۲–۳ ست)، RIR≈2
- 2–3×یوگا/تنفس ۱۰–۲۰ دقیقه (روزهای لوتئال/استرس بالا)
- Deload برنامه‌ریزی‌شده هر ۴–۸ هفته (کاهش حجم ۲۰–۴۰٪)
- غذا–مایعات:
- مدیترانه‌ای/غذاهای کم‌فرآوری‌شده؛ EPA/DHA از ماهی کم‌جیوه ۲–۳ وعده/هفته؛ منیزیم غذایی؛ کربوهیدرات کافی پیرامون تمرین؛ کافئین نهایت تا ظهر
- پرچم‌های ارجاع:
- افسردگی/اضطراب شدید، افکار خودآسیب‌رسان، بی‌خوابی مقاوم، علائم کوشینگی/آدرنال، کاهش وزن بی‌دلیل، آمنوره طولانی‌مدت.

Hypothalamus – Pituitary – thyroid



فیزیولوژی محور HPT



مخصوص پزشک محترم

- TRH→TSH→T4/T3 فعال می‌شود؛ کیفیت سنتز وابسته به یُد/آهن/سلنیوم و TSH است.
- تفسیر آزمایش‌ها را بر Free T4/T3 و TSH بسپارید— Total ممکن است با TBG گمراه‌کننده باشد.
- در برنامه‌ریزی تمرین/تغذیه زنان: مراقب کمبود آهن/سلنیوم باشید؛ مصرف افراطی ید (مثلاً جلبک) را اجتناب کنید؛ در بارداری/OCP اثر TBG را در ذهن داشته باشید.

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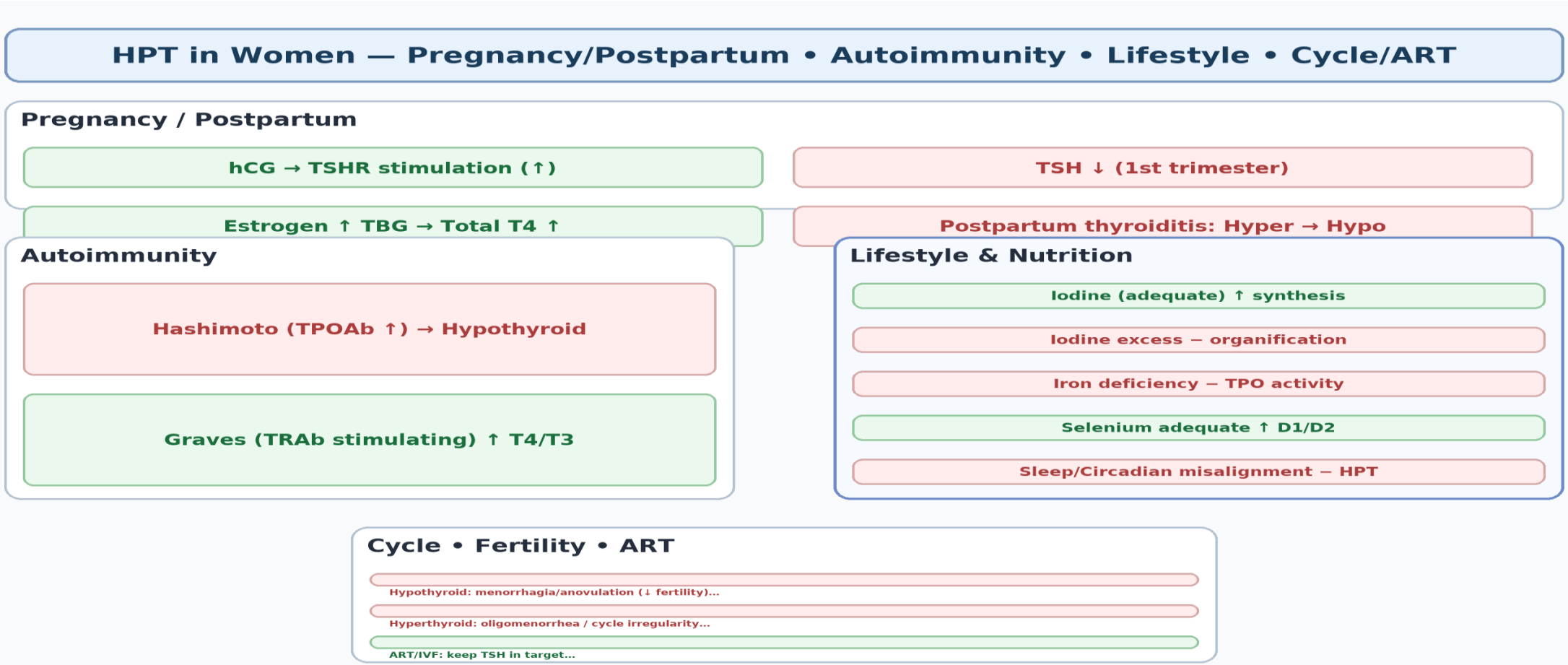
HPT در حاملگی، خودایمنی، سبک زندگی



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ورزش در تیروئید کم‌کار و پرکار

Exercise Traffic Light — Thyroid Conditions

Hypothyroid Training

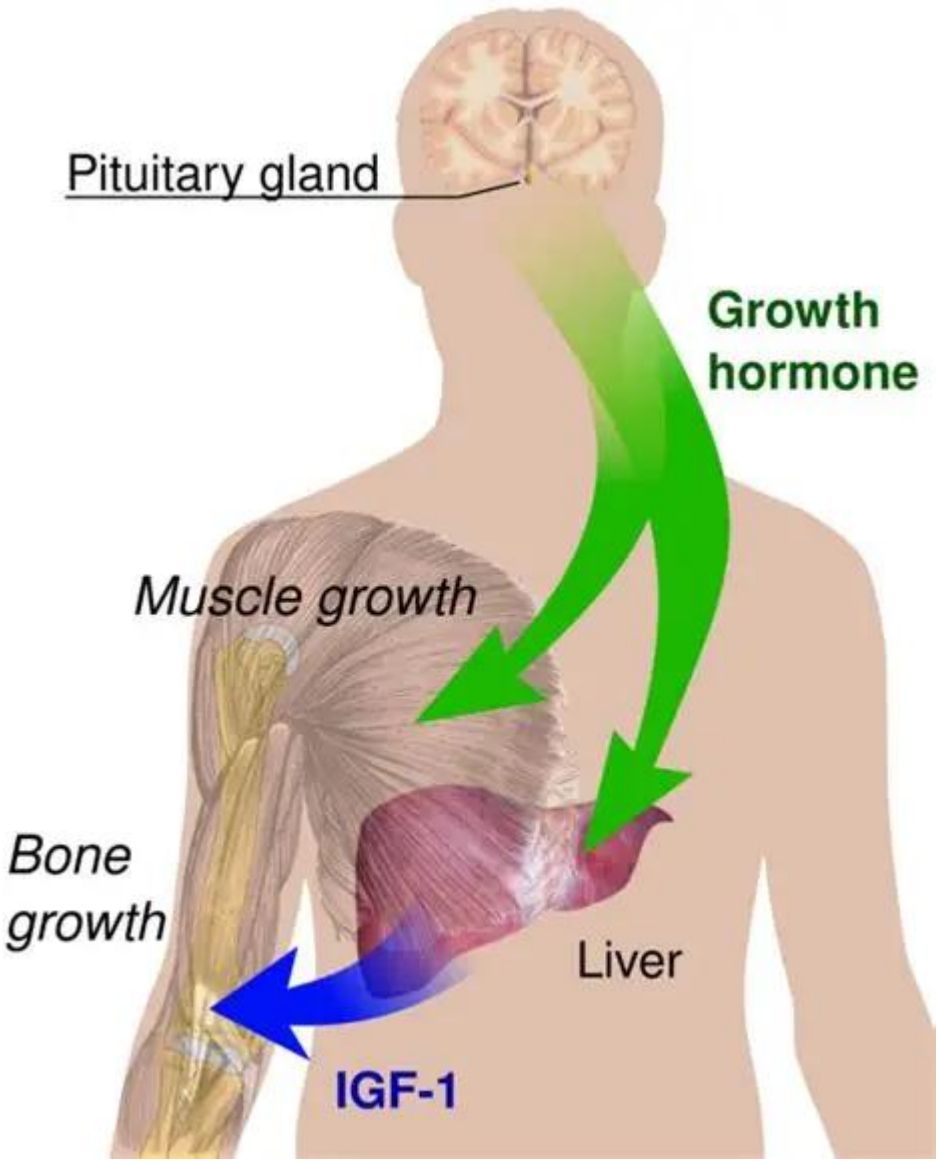
- Aerobic: Low → Moderate Intensity
- Progressive Resistance Training
- Long Warm-up (10–15 min)
- Focus: Thermogenesis & Energy Drive
- Caution: Fatigue → increase slowly

Hyperthyroid Training

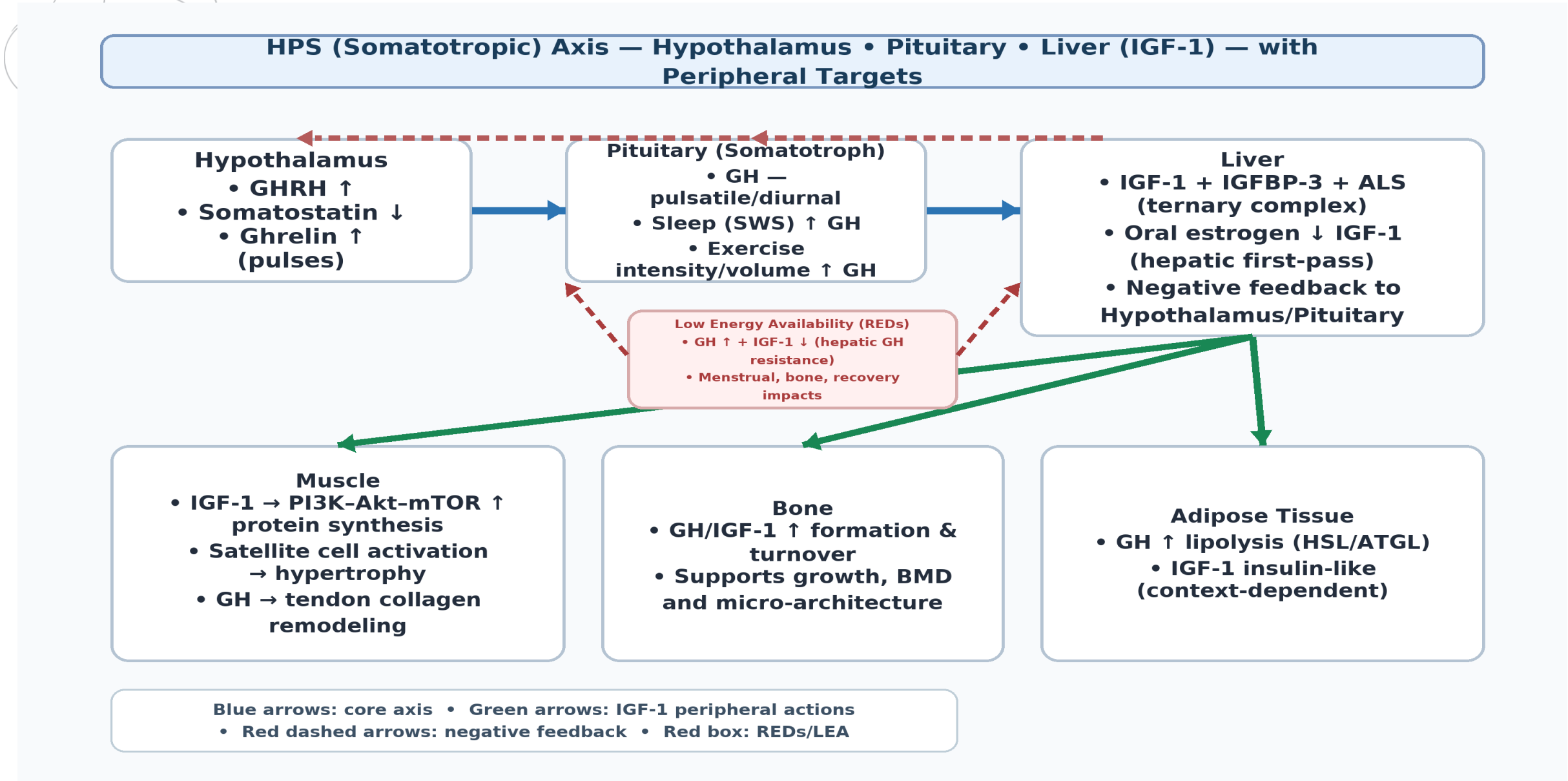
- Low-Impact Activities (Yoga, Pilates, Wal
- Breathing & Mindful Movement
- Avoid: Heat Exposure / HIIT / Overtraining
- Focus: Cooling, Parasympathetic Tone
- Caution: Heart Rate & Anxiety

Progressive resistance (Hypo) | HIIT avoidance (Hyper)

Hypothalamus – Pituitary – somatotropic



محور هیپوتالاموس–هیپوفیز–کبد



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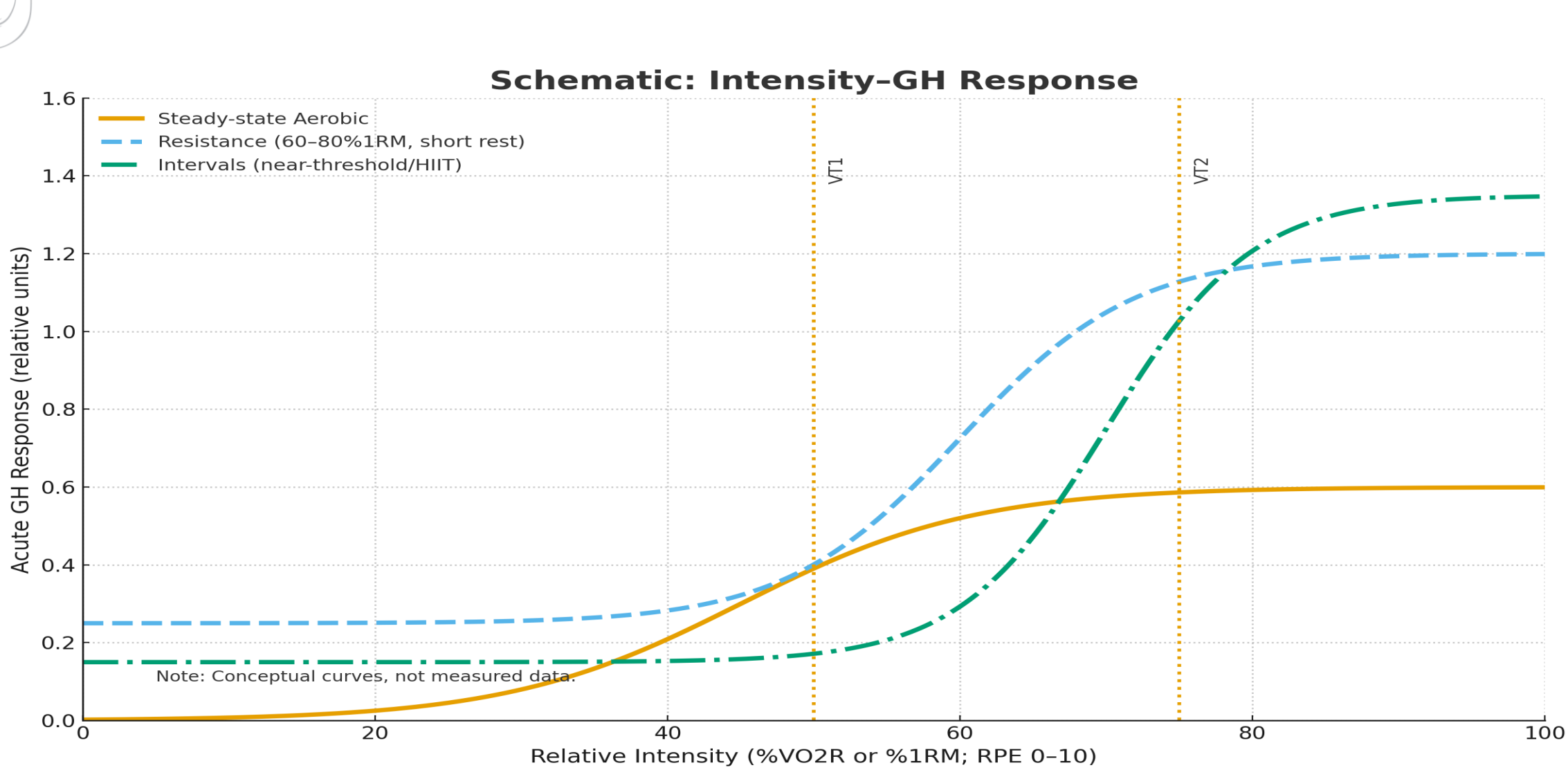
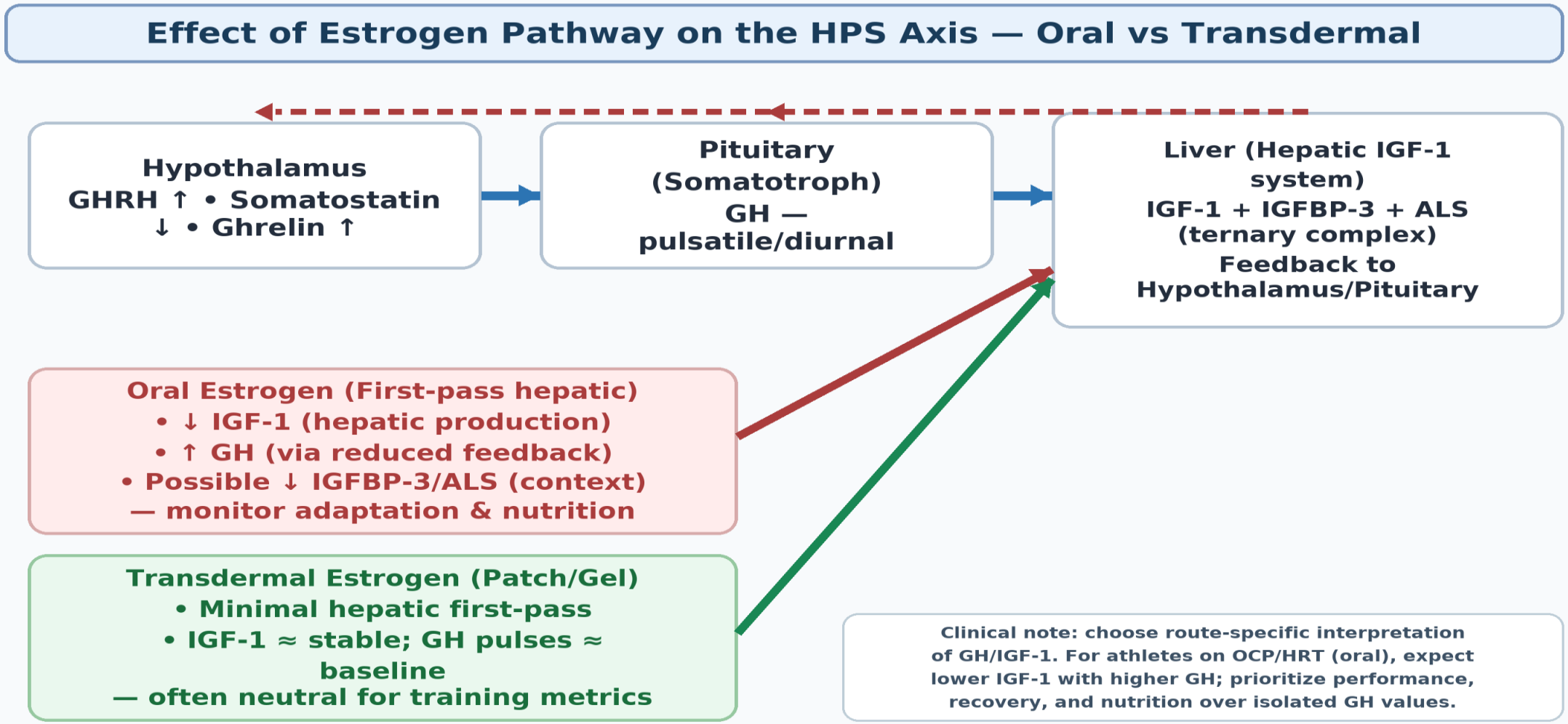
پاسخ هورمون رشد به ورزش



اثر استروژن خوراکی و ترنسدرمال بر محور HPS



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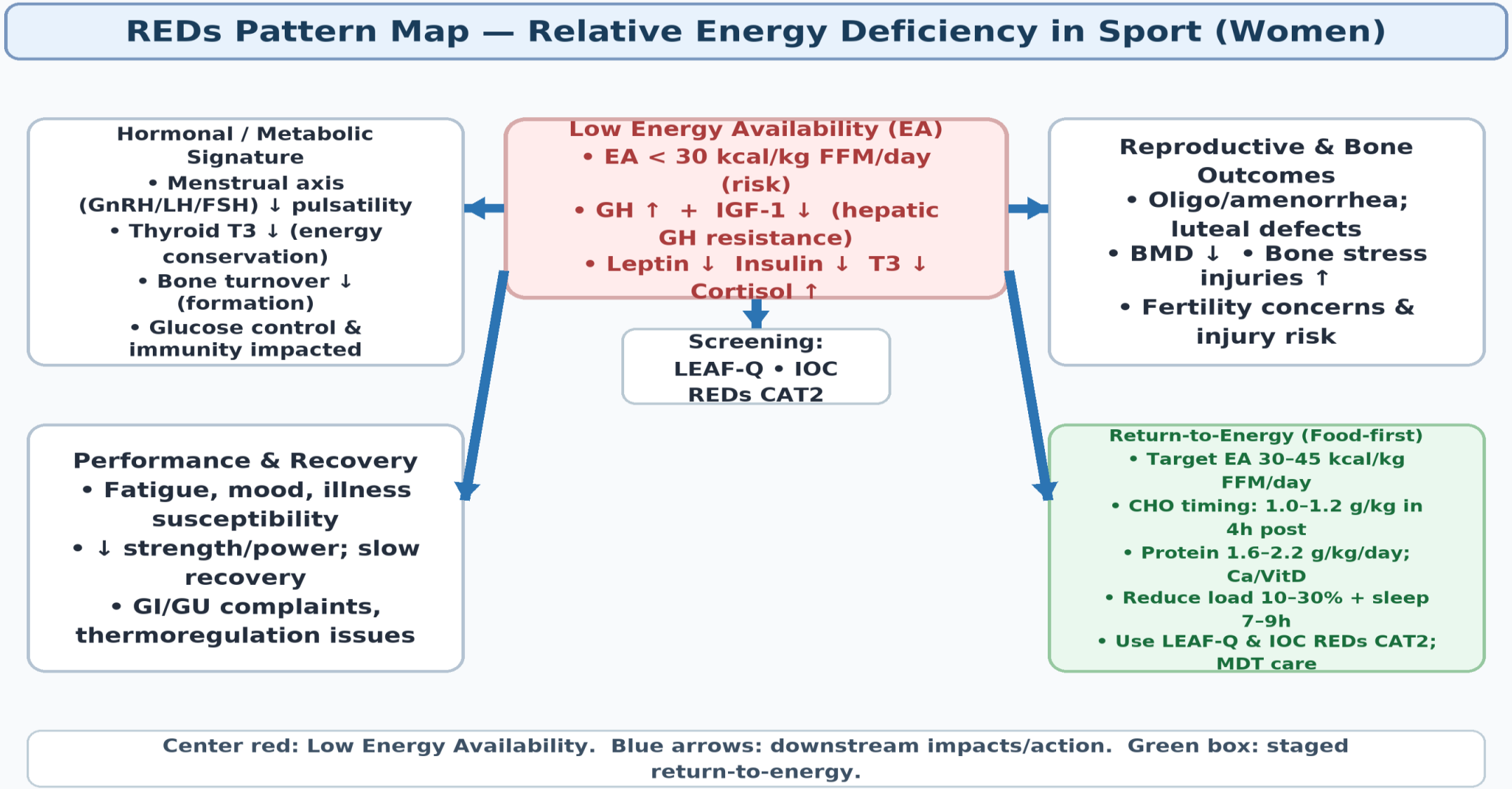
کمبود انرژی نسبی یا RED چیست؟



مقاله‌های برگزیده بسیار معتبر

biomolecules

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Review

How Does Physical Activity Modulate Hormone Responses?

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Abstract: Physical activity highly impacts the neuroendocrine system and hormonal secretion. Numerous variables, both those related to the individual, including genetics, age, sex, biological rhythms, nutritional status, level of training, intake of drugs or supplements, and previous or current pathologies, and those related to the physical activity in terms of type, intensity, and duration of exercise, or environmental conditions can shape the hormonal response to physical exercise. The aim of this review is to provide an overview of the effects of physical exercise on hormonal levels in the human body, focusing on changes in concentrations of hormones such as cortisol, testosterone, and insulin in response to different types and intensities of physical activity. Regular monitoring of hormonal responses in athletes could be a potential tool to design individual training programs and prevent overtraining syndrome.

Keywords: athletes; physical activity; cortisol; testosterone; growth hormone; thyroid; insulin; catecholamines

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1. Introduction

It is widely acknowledged that elite athletes exhibit changes in their hormonal composition due to conditioning; that is the adaptive response to training to become physically fit [1]. The influence of physical training on the hormonal system of athletes is intricate. Various factors, including the intensity and length of training, diet and energy levels, gender, sex, age, and stage of sexual development, all play a role in shaping the hypothalamus and pituitary gland responses to physical strain [2]. In particular, the intensity and duration of training stimulate the hypothalamic–pituitary–adrenal (HPA) axis, leading to increased cortisol levels, while an inadequate diet and low energy availability suppress reproductive hormones [3,4]. Gender and sex differences modulate the hormonal response, with variations related to testosterone and estrogen levels [5]. Age affects the reactivity of the hypothalamic–pituitary axis, with an attenuated response observed in older adults compared to younger individuals [6]. Additionally, stages of sexual development, such

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اثر بیش‌تمرینی بر عملکرد بافت‌ها

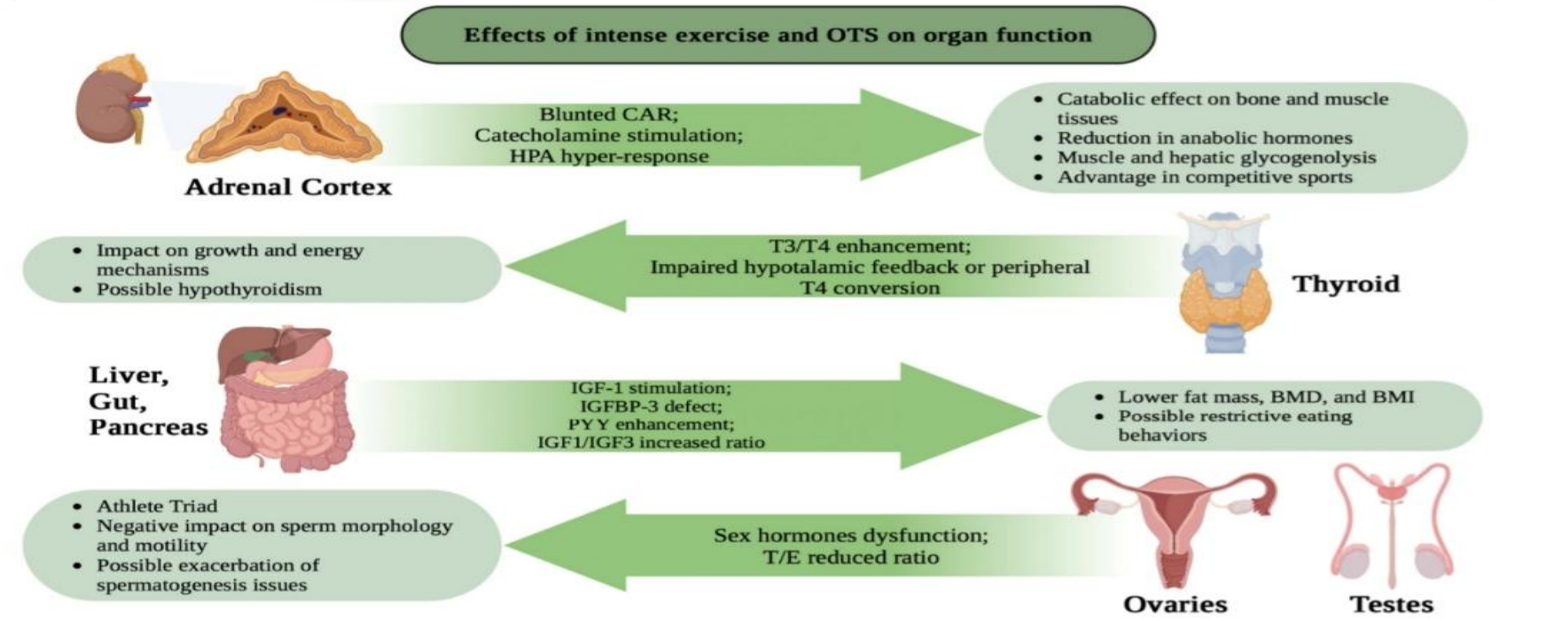
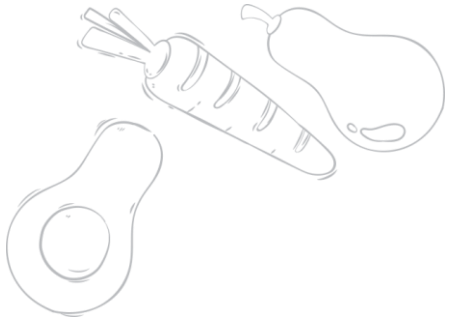


Figure 1. Effects of intense training/OTS on different organ function. The impact of OTS, due to too intense training or insufficient recovery, on some organ functions are summarized. Overtraining syndrome (OTS); cortisol awakening response (CAR); triiodothyronine (T3); thyroxine (T4); insulin-like growth factor-1 (IGF-1); insulin-like growth factor binding protein-3 (IGFBP-3); peptide YY (PYY); bone mineral density (BMD); body mass index (BMI).



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Epigenetics of functional hypothalamic amenorrhea

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Functional hypothalamic amenorrhea (FHA) is a temporary infertility characterized by the suppression of the hypothalamic–pituitary–gonadal (HPG) axis, induced by the inhibition of the hypothalamic pulsatile secretion of the gonadotropin-releasing hormone (GnRH), in the presence of stressors, including eating disorders, excessive exercise, and psychological distress. Although the stressful factors that may lead to FHA are well-established, little is known about the inter-individual variability in response to stress and the consequent inhibition of the HPG axis. Not all women, indeed, manifest FHA in presence of stressful conditions. Recent studies highlighted a genetic contribution to FHA. Rare or polymorphic variants in genes that control the development and/or function of GnRH neurons may contribute, indeed, to the adaptability of the reproductive axis to stress factors. Also epigenetic changes have been associated with different pathways involved in the HPG axis and therefore, take part in FHA and confer a personal predisposition to anovulation consequent to a stressful event, or represent biological markers of response to stress. This review summarizes recent advances in the identification of the contribution of (epi)genetics to FHA and to long-term complications of functional amenorrhea, and reports insights into the involvement of additional genetic loci in FHA development on the bases of the clinical and molecular overlap with other gynecological and/or psychological conditions. Finally, we describe the promising application of induced pluripotent stem cells (iPSCs) as a new approach to investigate the molecular pathways involved in FHA.

KEYWORDS

functional hypothalamic amenorrhea (FHA), epigenetics, susceptibility genes, anorexia nervosa, delayed puberty

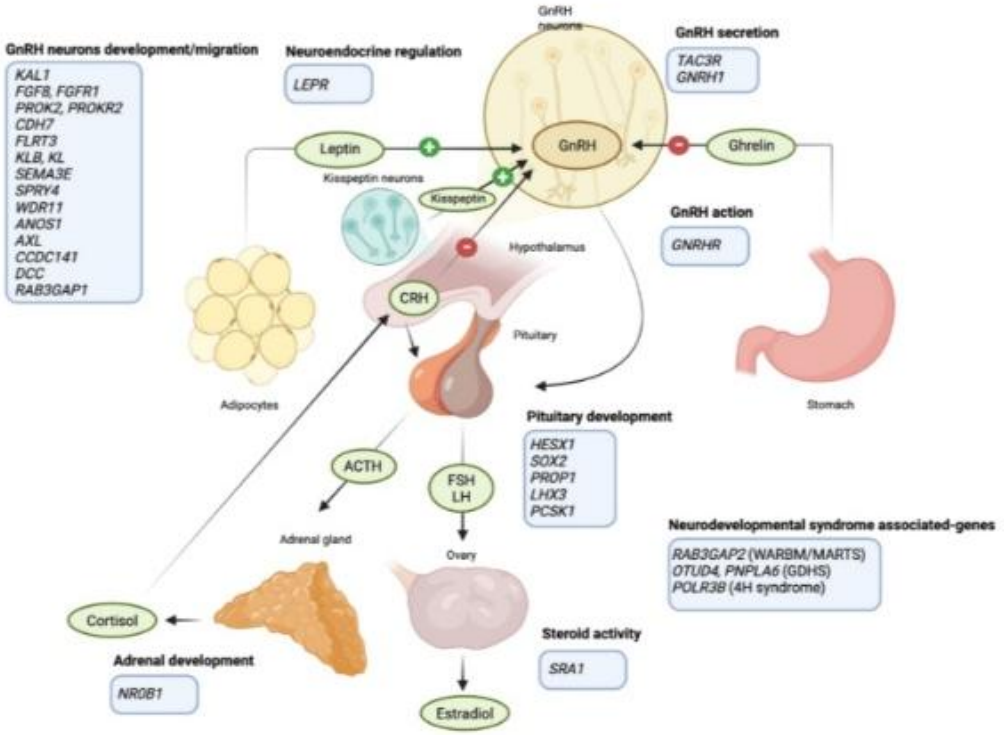

FIGURE 1
Schematic representation of the HPG axis regulation and the FHA predisposing genes. GnRH neurons in the hypothalamus release GnRH upon different stimuli: kisspeptin, produced by a specific group of hypothalamic neurons, is a major player in the neuroendocrine control of GnRH and gonadotrophins secretion; ghrelin and leptin allow the regulation of GnRH secretion according to energy balance (link between HPG axis and food intake); cortisol inhibits GnRH secretion (link between HPG axis and anxiety). FHA-predisposing genes are listed in the light blue boxes (created with BioRender.com).

TABLE 2 Predisposing genes to gynecological and psychological conditions showing overlapping features with FHA, and to long-term consequences of FHA.

Disorder	Condition	Overlapping with FHA	Affected pathway	Genes involved
Gynecological disorders	Anorexia nervosa	AN is a chronic energy deficiency that leads to the suppression of the HPG axis because of the reduced secretion of GnRH	Neurotrophin signaling pathway	*BDNF* *NTRK2* *NTRK3*
			Serotoninergic and leptin pathways	*OPRD1* *HTRD1* *EBF1* *SLC6A4*
	Delayed puberty	Delayed puberty may occur in patients with FHA and can be considered an early clinical sign of this condition	IHH development	*LEPR* *GNRH1* *TACR2* *HS6ST1* *FGFR1* *KLB*
			GnRH neuron migration	*IGSF10*
Psychological disorders	Anxiety	The neuroendocrine response to stress and stress-related neuronal plasticity involves the HPG axis	Energy balance and anxiogenic effect of CRH	*NPY*
	Mood disorders	Altered neuroplasticity related to stress	Neuroplasticity, neurogenesis, neuronal survival, and differentiation	*BDNF*
Long-term consequences	Osteopenia and osteoporosis	Prolonged hypoestrogenism in FHA leads to osteopenia and osteoporosis	Estrogen receptor	*ESR1-XbaI*
			Vitamin D receptor	*VDRBsmI site* *VDRFokI site*

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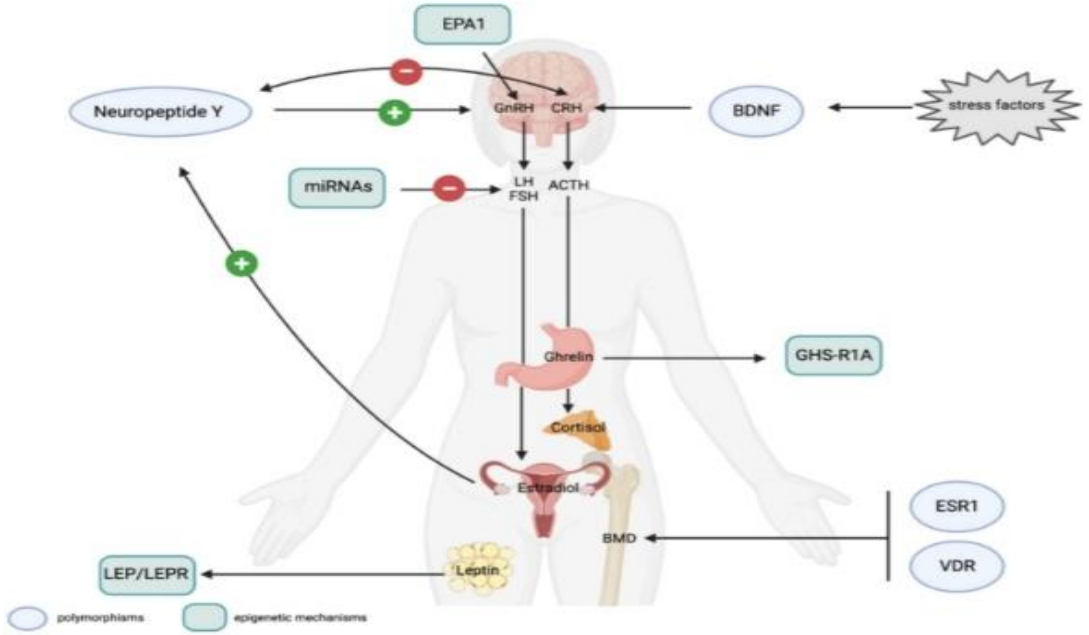

FIGURE 2
(Epi)genetic mechanisms possibly involved in FHA development and FHA-related long-term consequences. **Genetic mechanisms**. Polymorphisms in the *Neuropeptide Y* (*NPY*) and *BDNF* genes affect stress response. The NPY positively controls GnRH secretion in presence of adequate levels of estrogen and has an anxiolytic effect by counteracting CRH activity. CRH, itself, downregulates the expression of the NPY. NPY polymorphisms have been associated to resilience or stress-sensitive phenotypes. BDNF polymorphisms are suggested to affect neuroplasticity and stress responses. Polymorphisms in the estrogen receptor (ESR1) and in the vitamin D receptor (VDR) genes influence bone mineral density (BMD) and may be associated to osteopenia and osteoporosis, consequent to prolonged hypoestrogenism. **Epigenetic mechanisms**. The EPA1 transcription factor controls GnRH expression and a 5'-UTR polymorphism has been associated with a higher risk of amenorrhea in animal models. Altered methylation levels of the *LEP* and *LEPR* genes have been associated with the effect of leptin, produced by adipocytes on the HPG axis and on the personal response to psychotherapeutic treatment in AN patients. Methylation of the ghrelin receptor gene (*GHS-R1A*) are thought to be involved in ghrelin resistance affecting GnRH secretion. Specific miRNAs have been reported to control the post-transcriptional expression of LH and FSH, and to be a promising peripheral biomarkers to control the effect of hormonal therapy in FHA women. Light blue circles indicate polymorphic variants in genes possibly associated to response to stress or long-term consequences in FHA women; light green rectangles indicate the epigenetic mechanisms (including transcription factors, miRNA and methylation) that can play a role in the regulation of the HPG axis and in FHA development (created with BioRender.com).

مدیریت آمنوره عملکردی با تغذیه و ورزش

nutrients

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Review

Dietary and Lifestyle Management of Functional Hypothalamic Amenorrhea: A Comprehensive Review

Katarzyna Dobranowska [1,†], Stanisława Plińska [2] and Agnieszka Dobosz [1,*]

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Abstract: Functional Hypothalamic Amenorrhea (FHA) is a condition characterized by the absence of menstruation, which is increasingly affecting young women. However, specific recommendations for treating and preventing this condition are lacking. Based on a review of the available literature, this article provides practical and feasible dietary management recommendations for healthcare professionals and researchers in women's health and nutrition. It answers the question of what interventions and nutritional recommendations are necessary to restore menstrual function in women struggling with FHA. Physicians recommend an energy availability threshold of 30 kcal/kg FFM/day to prevent FHA. Also, energy availability below and above this threshold can inhibit LH pulsation and cause menstrual disorders. In addition, the risk of menstrual disorders increases with a decrease in the caloric content of the diet and the duration of the energy deficit, and women with FHA have significantly lower energy availability than healthy women. It is essential to ensure that adequate kilocalories are provided throughout the day (regular meals that are a source of proper glucose) to avoid a negative energy balance, as glucose has been proven to affect LH pulses and T3 and cortisol concentrations in the body. Dietary intervention should focus on increasing the caloric content of the diet, thus increasing energy availability and restoring energy balance in the body. Treatment and diagnosis should also focus on body composition, not just body weight. An increase in body fat percentage above 22% may be required to restore menstrual function. In women with FHA, even an increase in body fat mass of one kilogram (kg) increases the likelihood of menstruation by 8%. It is advisable to reduce the intensity of physical activity or training volume, while it is not advisable to give up physical activity altogether. It is also important to ensure adequate intake of micronutrients, reduce stress, and incorporate cognitive–behavioral therapy.

Keywords: functional hypothalamic amenorrhea (FHA); dietary intervention; energy availability; energy deficit; nonpharmacological treatment of FHA

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1. Introduction

Functional hypothalamic amenorrhea (FHA) is a common problem among physically active women, especially in sports related to body shape and endurance. However, the problem is also increasingly prevalent in women playing sports recreationally and aiming for weight loss [1,2]. Long-term persistent FHA increases the risk of osteoporosis, cardiovascular disease, depression, and infertility [3,4]. It is, therefore, essential to raise awareness of the FHA problem and identify appropriate treatments.

Functional hypothalamic amenorrhea is defined as the absence of menstruation for a period of three or six months in a previously menstruating woman. It is associated with the inhibition of the hypothalamic–pituitary–ovarian (HPO) axis [2–5]. Three main factors are assumed to cause functional hypothalamic amenorrhea: psychological stress, caloric restriction, and excessive physical activity. Unlike other causes of secondary amenorrhea,

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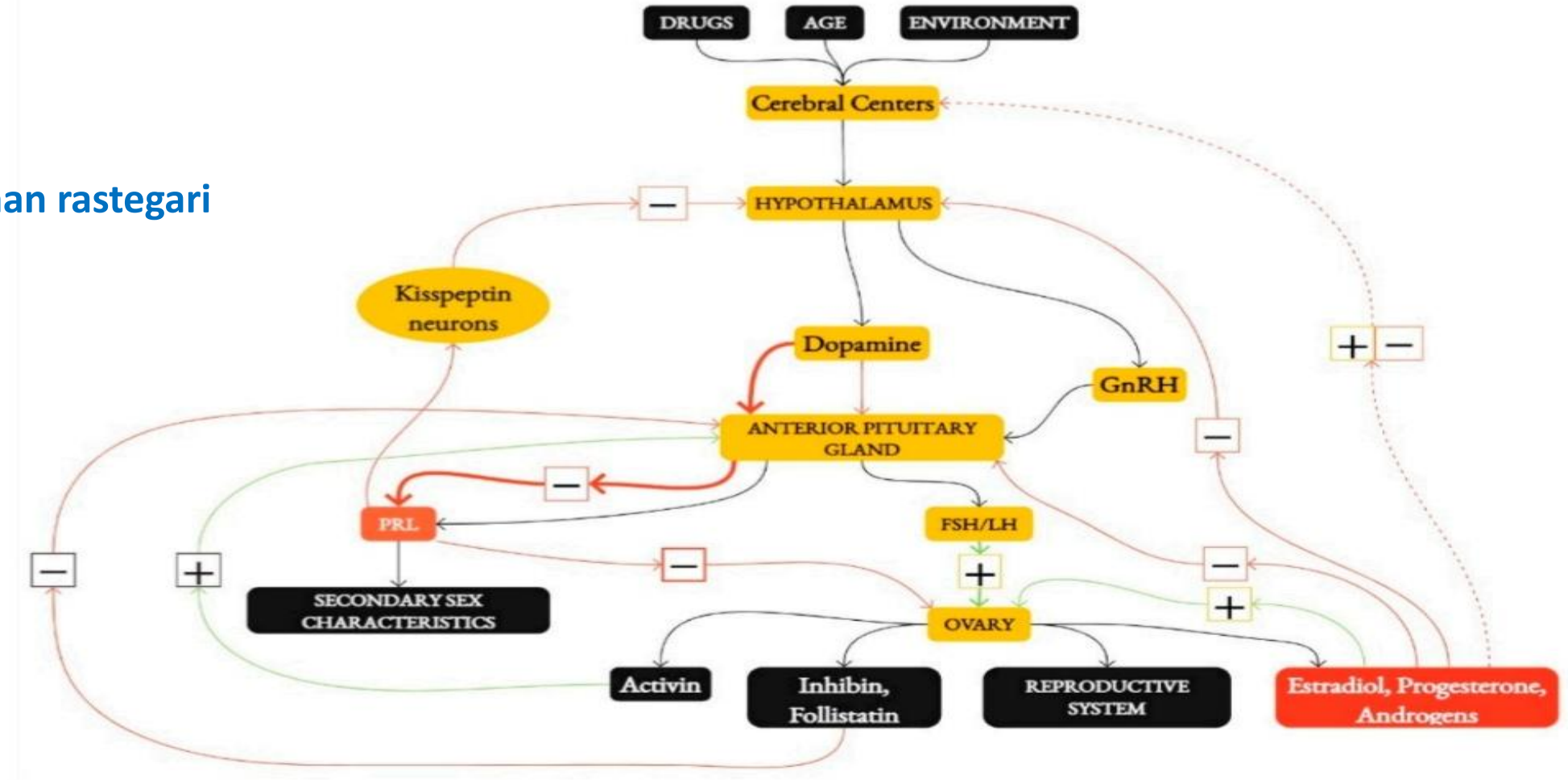

Figure 1. Regulation of the Reproductive System. Abbreviations: GnRH—Gonadotropin-Releasing Hormone, LH—Luteinizing Hormone, FSH—Follicle-Stimulating Hormone, PRL—Prolactin.

اثر کالری دریافتی بر سیکل ماهیانه

Table 1. Summary of Studies on the Impact of Energy Availability on the Menstrual Cycle.

Study	Energy Availability (EA) Levels	Findings on LH Pulses	Findings on Menstrual Disorders	Notes
Loucks et al. [1]	30 kcal/kg FFM/day 20 kcal/kg FFM/day 10 kcal/kg FFM/day	−20 kcal/kg FFM/day, 16% decrease in LH pulse frequency, a 21% increase in amplitude, −10 kcal/kg FFM/day, 39% decrease in LH pulse frequency, 109% increase in amplitude	EA below 30 kcal/kg FFM/day is linked to a higher likelihood of menstrual disorders, such as oligo/amenorrhea	Establishes a threshold for EA below which LH pulsatility and menstrual function are impaired
Koltun et al. [14]	No specific threshold identified. Reduction from 38 to 28 kcal/kg FFM/day	Decrease in LH pulse frequency by 0.017 pulses/hour for each unit decrease in EA. Lower EA also significantly reduces LH secretion frequency	Increased risk of luteal phase defects with lower EA. Significant EA reductions heighten the likelihood of menstrual disorders	No clear threshold for EA, but findings suggest more severe impacts with greater EA reduction
Liberman et al. [2]	EA < 30 kcal/kg FFM/day	LH pulse frequency decreases and amplitude increases with reduced EA	Menstrual disorders (luteal phase defects, anovulation, oligomenorrhea) become more likely as EA decreases but can occur even above 30 kcal/kg FFM/day	Highlights that menstrual disorders can occur even above 30 kcal/kg FFM/day, challenging the strict threshold concept
Reed et al. [10]	FHA group: 30.9 ± 2.4 kcal/kg FFM/day vs. 36.9 ± 1.7 kcal/kg FFM/day in control	No specific findings on LH pulses were provided	Women with functional hypothalamic amenorrhea (FHA)had significantly lower EA compared to regularly menstruating women	EA of 30 kcal/kg FFM/day does not clearly differentiate between regular menstruation and menstrual disorders

Abbreviations: EA—Energy Availability, FFM—Fat-Free Mass, LH—Luteinizing Hormone, FHA—Functional Hypothalamic Amenorrhea.

اثر افزایش کالری دریافتی بر ریکاوری منس

Table 2. Summary of Studies on the Impact of Increased Caloric Intake on Menstrual Recovery.

Study	Population	Intervention	Results	Conclusion
De Souza et al. [20]	Thirty-three women (age 18–35) with secondary amenorrhea or oligomenorrhea, BMI 16–25 kg/m^2, exercising >2 h/week	Increased caloric intake by 330 ± 65 kcal/day (20–40%) over 12 months	Weight gain: 2.6 ± 0.4 kg, Fat mass gain: 2.0 ± 0.3 kg, Increase in T3 concentration by 9 ± 4 ng/dL	A modest caloric surplus (~300–350 kcal/day) is sufficient for restoring menstrual cycles. Improved energy balance leads to menstrual recovery
Łagowska et al. [21]	Fifty-two athletes and ballet dancers with menstrual disorders, training >4 times/week	Increased caloric intake by 20–30%, energy availability increased by >30 kcal/kg FFM/day over 9 months	Weight gain: 1.3 kg (ballet dancers), no significant weight changes (athletes), Increased LH and LH/FSH ratio, Menstrual recovery in 3 dancers and 7 athletes	Increased caloric intake is critical for hormonal improvement and menstrual recovery. Menstrual function can be restored when body fat mass reaches 22%
Mallinson et al. [23]	Two women with FHA of different durations	A 12-month nutritional intervention with individualized caloric increases	Weight gain: 4.3 kg (long-term FHA) and 2.8 kg (short-term FHA), Improvements in leptin and T3 concentrations	Weight gain and improved hormone levels are crucial for menstrual recovery, with individual variations of response
Cominato et al. [24]	Adolescents with eating disorders	A 20-week nutritional intervention	Recovery of menstrual function linked to increases in BMI, LH, IGF-1, and estradiol	IGF-1 may serve as a potential marker for menstrual recovery. Nutritional rehabilitation is a key to restoring menstrual function
Deampfle et al. [25]	One hundred and fifty-two girls (age 11–18) with eating disorders and underweight	Observational study followed participants over 12 months	Forty-seven percent regained menstrual function, Strong correlation between %EBW and resumption of menstruation	Achieving expected body weight is strongly associated with menstrual recovery. BMI is not a reliable predictor of menstrual function

Abbreviations: BMI—Body Mass Index, T3—Triiodothyronine, LH—Luteinizing Hormone, FSH—Follicle-Stimulating Hormone, FFM—Fat-Free Mass, FHA—Functional Hypothalamic Amenorrhea, IGF-1—Insulin-Like Growth Factor 1, %EBW—Percentage of Expected Body Weight.

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Research Article

Reproductive and metabolic adaptation to multistressor training in women

Robert M. Gifford, Thomas J. O'Leary, Sophie L. Wardle, Rebecca L. Double, Natalie Z. M. Homer, A. Forbes Howie, Julie P. Greeves, Richard A. Anderson, David R. Woods, and Rebecca M. Reynolds

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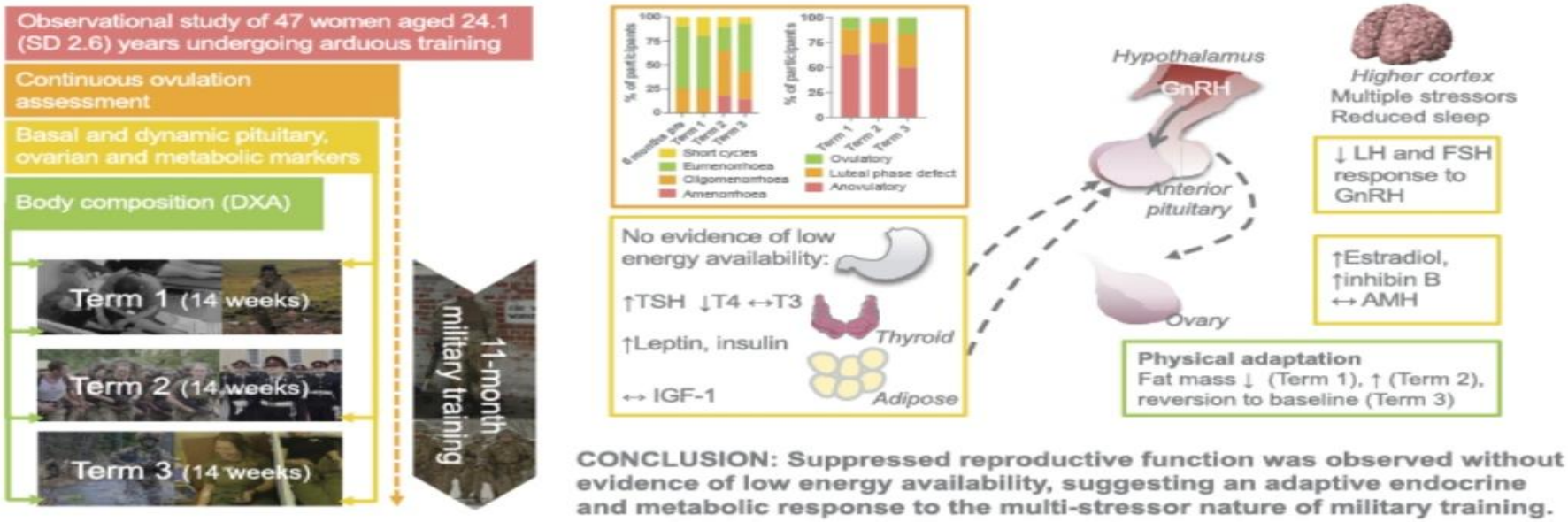
سازگاری های تولید مثل و متابولیک به انواع استرسورهای تمرینات ورزشی

مدیریت حجم تمرین ورزشی برای سرکوب نشدن تولید مثل

Reproductive and metabolic adaptation to multi-stressor training in women



CONCLUSION: Suppressed reproductive function was observed without evidence of low energy availability, suggesting an adaptive endocrine and metabolic response to the multi-stressor nature of military training.

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Harnessing the power of nutritional antioxidants against adrenal hormone imbalance-associated oxidative stress

Anil Patani[1], Deepak Balram[2], Virendra Kumar Yadav[3*], Kuang-Yow Lian[2*], Ashish Patel[3*] and Dipak Kumar Sahoo[4*]

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Oxidative stress, resulting from dysregulation in the secretion of adrenal hormones, represents a major concern in human health. The present review comprehensively examines various categories of endocrine dysregulation within the adrenal glands, encompassing glucocorticoids, mineralocorticoids, and androgens. Additionally, a comprehensive account of adrenal hormone disorders, including adrenal insufficiency, Cushing's syndrome, and adrenal tumors, is presented, with particular emphasis on their intricate association with oxidative stress. The review also delves into an examination of various nutritional antioxidants, namely vitamin C, vitamin E, carotenoids, selenium, zinc, polyphenols, coenzyme Q10, and probiotics, and elucidates their role in mitigating the adverse effects of oxidative stress arising from imbalances in adrenal hormone levels. In conclusion, harnessing the power of nutritional antioxidants has the potential to help with oxidative stress caused by an imbalance in adrenal hormones. This could lead to new research and therapeutic interventions.

KEYWORDS

adrenal hormone imbalance, oxidative stress, nutritional antioxidants, reactive oxygen species, HPT axis

قدرت آنتی اکسیدان خوراکی برای بالانس کردن هورمونهای استرسی

ساختار غده فوق کلیوی و تولید هورمون

Patani et al.

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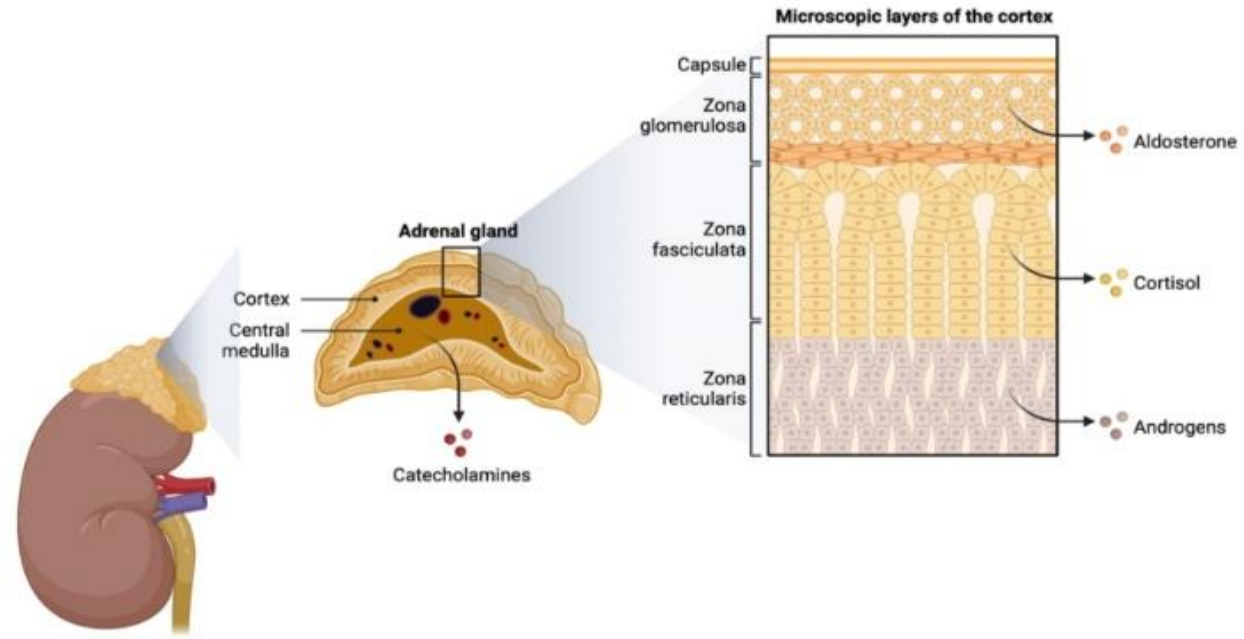

FIGURE 1
Adrenal gland structure and hormone production. The figure was produced with BioRender (Biorender.com; accessed on 29th July 2023).

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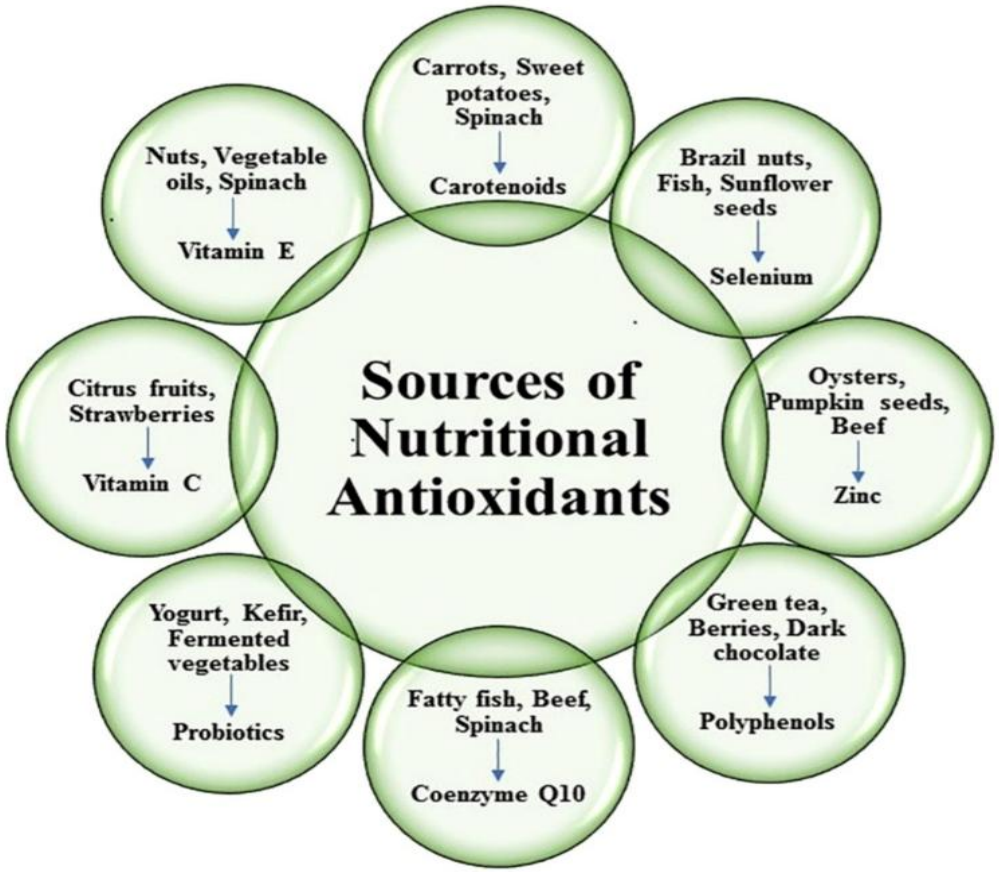

FIGURE 2
Sources of different types of nutritional antioxidants.

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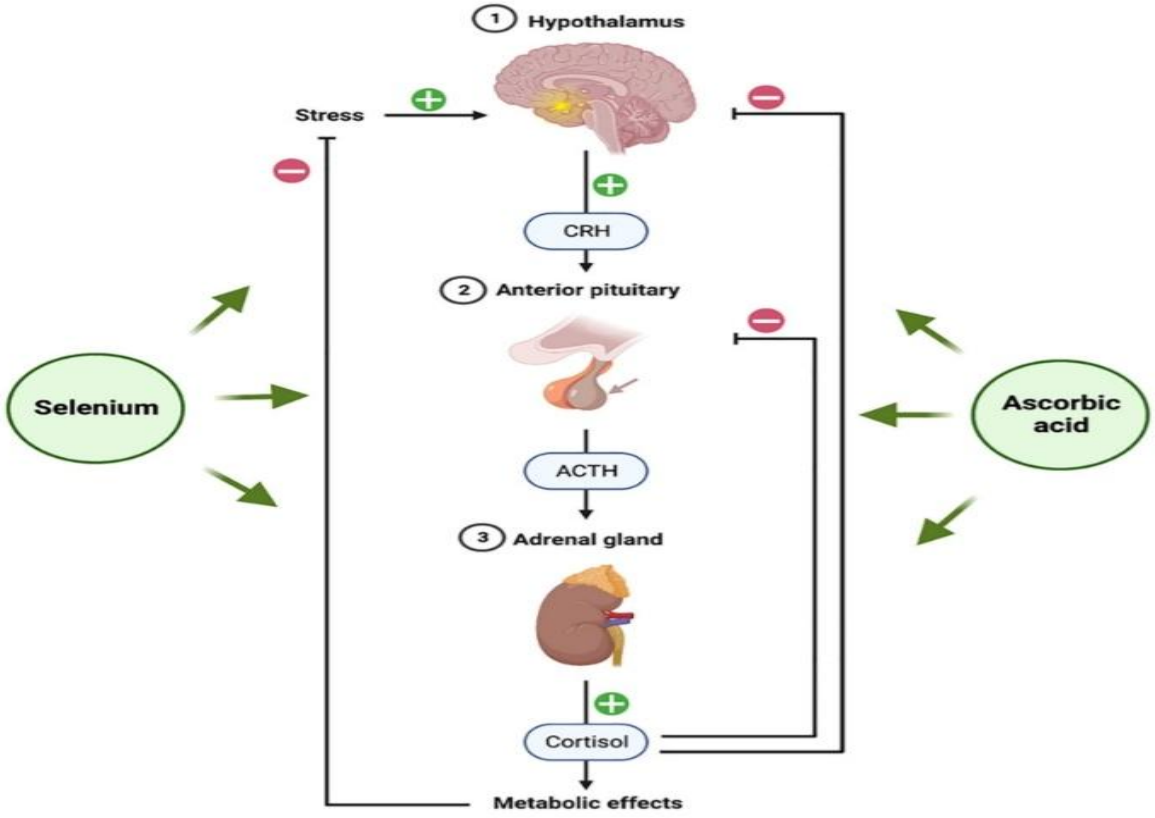

FIGURE 4
Hypothalamic-Pituitary-Adrenal (HPA) axis and role of selenium and ascorbic acid supplements. The figure was produced with BioRender (Biorender.com; accessed on 29th July 2023).

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چرا مصرف میوه, سبزیجات, حبوبات, غلات کامل و فیبرها برای سلامت همه جانبه‌ی یک زن ضروری است؟

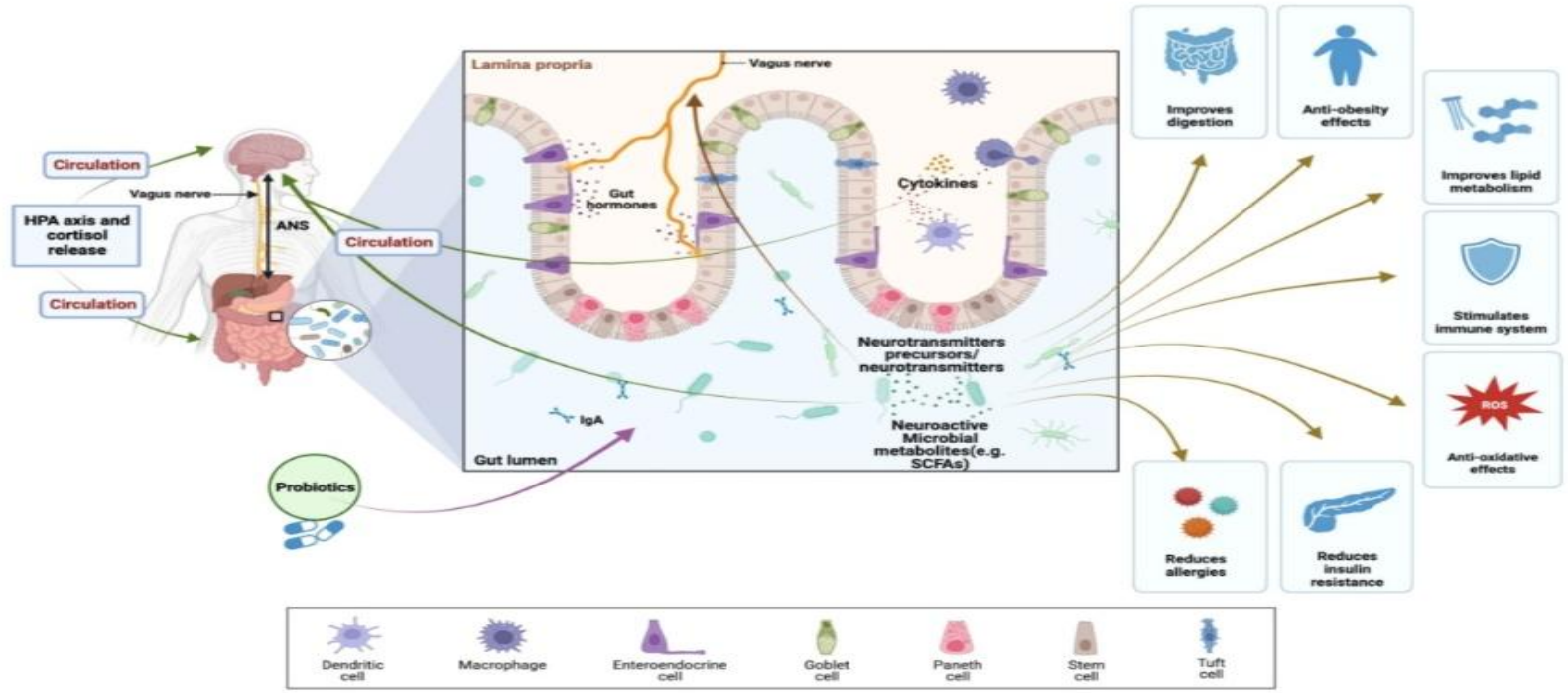

FIGURE 5
Health benefits of probiotics and their effects on brain, gut, and microbiome (BGM) axis modulating of HPA axis and cortisol release. The BGM axis network of routes that facilitate the exchange of information and signals encompasses neuronal elements (vagus nerve, neurotransmitters, and enteric nervous system), the HPA axis, and stress hormones like cortisol. Furthermore, immune mechanisms, specifically cytokines, contribute to this complex interplay. (SCFAs), Short-chain fatty acids; (ANS), autonomic nervous system; (ROS), reactive oxygen species; (HPA axis), Hypothalamic–pituitary–adrenal axis. The figure was produced with BioRender (Biorender.com; accessed on 30th Oct 2023).

چرا مصرف بیش از اندازه آنتی اکسیدان به صورت مکمل مضر است؟

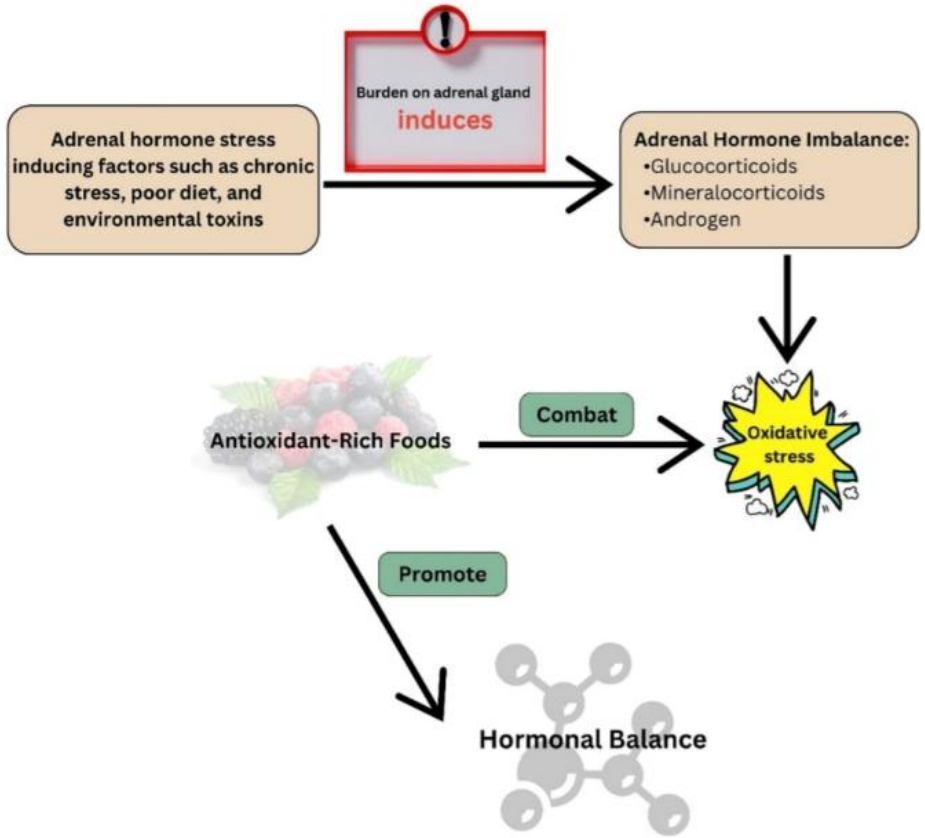

FIGURE 6
Role of nutritional antioxidants in alleviating adrenal hormone imbalance.

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نقش آنتی اکسیدان خوراکی در عملکرد هورمونی غده فوق کلیوی

TABLE 2 The role of nutritional antioxidants in Adrenal hormone function.

Nutritional Antioxidant	Role in Adrenal Hormone Function	Reference
Vitamin C	- Supports the production of androgens, glucocorticoids, and mineralocorticoids - Serves as a cofactor in the process by which cholesterol is transformed into pregnenolone	(103)
Vitamin E	- Protects adrenal cells from oxidative stress - Possibly plays a function in regulating cortisol levels	(197)
Carotenoids	- Carotenoids contained in several foods, beta-carotene and lycopene, act as antioxidants - Aiding in the reduction of oxidative stress in the adrenal glands	(10)
Selenium	Important in the synthesis of selenoproteins such as glutathione peroxidase, which protects adrenal cells from oxidative damage.	(144)
Zinc	- Zinc is required for the synthesis, release, and general function of adrenal hormones - As an antioxidant, it protects cells from oxidative stress.	(198)
Polyphenols	Reduce oxidative damage and inflammation in the adrenal glands to help with adrenal hormone balance.	(161)
Coenzyme Q10	- Plays a critical function in the cellular energy production process - Supports the overall function of the adrenal glands and may lessen oxidative stress.	(179)
Probiotics	- Indirectly altering adrenal hormone balance and encouraging optimal function by mitigating oxidative stress and inflammation.	(199)

محور هورمون رشد و IGF1 در سلامتی و بیماری

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Growth hormone/insulin-like growth factor I axis in health and disease states: an update on the role of intra-portal insulin

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Growth hormone (GH) is the key regulator of insulin-like growth factor I (IGF-I) generation in healthy states. However, portal insulin delivery is also an essential co-player in the regulation of the GH/IGF-I axis by affecting and regulating hepatic GH receptor synthesis, and subsequently altering hepatic GH sensitivity and IGF-I generation. Disease states of GH excess (e.g., acromegaly) and GH deficiency (e.g., congenital isolated GH deficiency) are characterized by increased and decreased GH, IGF-I and insulin levels, respectively, where the GH/IGF-I relationship is reflected by a "primary association". When intra-portal insulin levels are increased (e.g., obesity, Cushing's syndrome, or due to treatment with glucocorticoids and glucagon-like peptide 1 receptor agonists) or decreased (e.g., malnutrition, anorexia nervosa and type 1 diabetes mellitus), these changes secondarily alter hepatic GH sensitivity resulting in a "secondary association" with discordant GH and IGF-I levels (e.g., high GH/low IGF-I levels or low GH/high IGF-I levels, respectively). Additionally, intra-portal insulin regulates hepatic secretion of IGFBP-1, an inhibitor of IGF-I action. Through its effects on IGFBP-1 and subsequently free IGF-I, intra-portal insulin exerts its effects to influence endogenous GH secretion via the negative feedback loop. Therefore, it is important to understand the effects of changes in intra-portal insulin when interpreting the GH/IGF-I axis in disease states. This review summarizes our current understanding of how changes in intra-portal insulin delivery to the liver in health, disease states and drug therapy use and misuse that leads to alterations in GH/IGF-I secretion that may dictate management decisions in afflicted patients.

Dr. Arman rastegari

تغییرات انسولین ورید پورتال در سلامتی و انواع بیماری‌ها

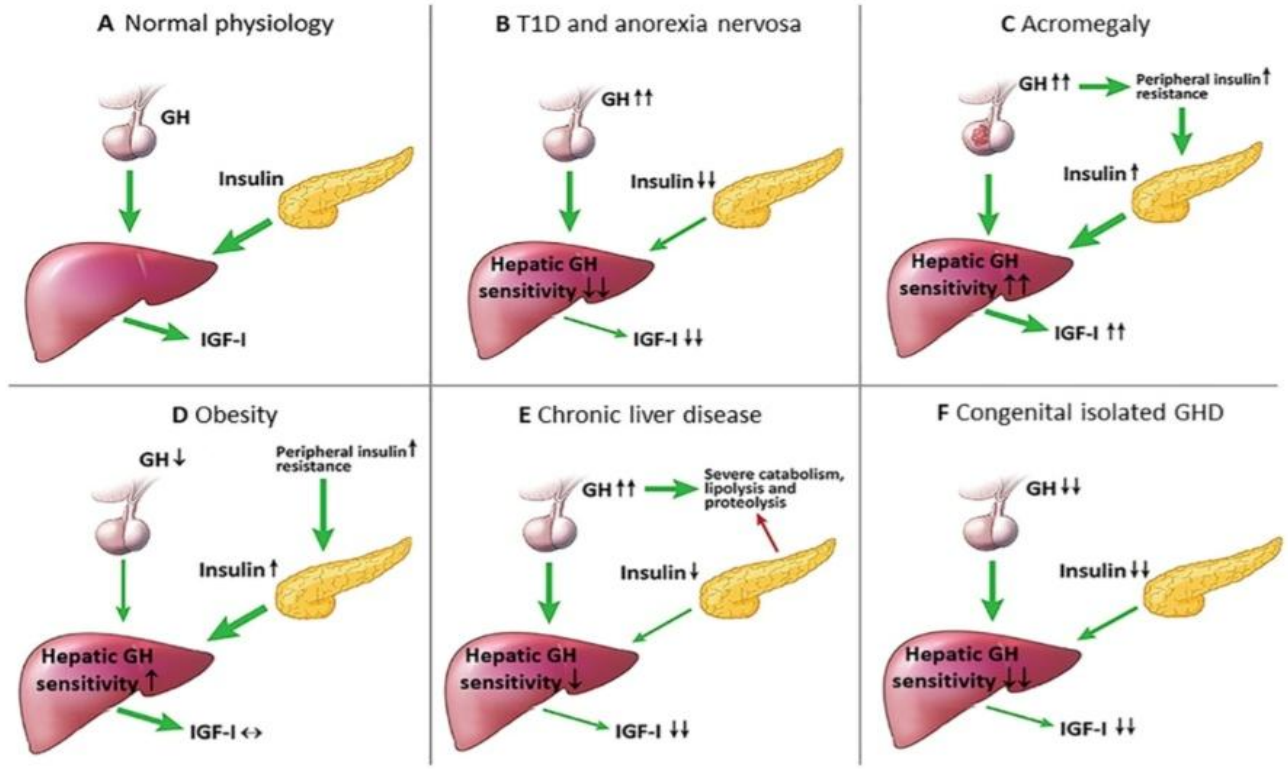

FIGURE 1
Intra-portal insulin changes in health and several disease states. **(A)** Normal physiological state: intra-portal insulin regulates hepatic GH sensitivity in the generation of IGF-I; **(B)** T1D and anorexia nervosa: intra-portal hypoinsulinemia decreases hepatic GH sensitivity resulting in low IGF-I levels, and due to the lack of negative feedback by IGF-I on the hypothalamus, GH levels increase; **(C)** Acromegaly: GH excess increases insulin resistance causing intra-portal hyperinsulinemia that leads to increased hepatic GH sensitivity, and the combination of increased GH and hepatic GH sensitivity leads to further increase in IGF-I levels; **(D)** Obesity: peripheral insulin resistance causes compensatory intra-portal hyperinsulinemia that increases hepatic GH sensitivity resulting in high normal IGF-I levels and low GH levels; **(E)** Chronic liver disease: the liver fails to produce sufficient IGF-I resulting in high GH levels due to the lack of negative feedback by IGF-I on the hypothalamus that causes a state of catabolism, lipolysis and proteolysis, lipolysis and decreased β-cell insulin secretion; **(F)** Congenital isolated GHD: severe lifelong GHD results in decreased β-cell mass and insulin secretion, and the combination of decreased GH and intra-portal hypoinsulinemia leads to markedly low IGF-I levels.

توانایی انسولین اینترا پورتال به عنوان تقویت کننده یا ترمز هورمون رشد

Dr. Arman rastegari

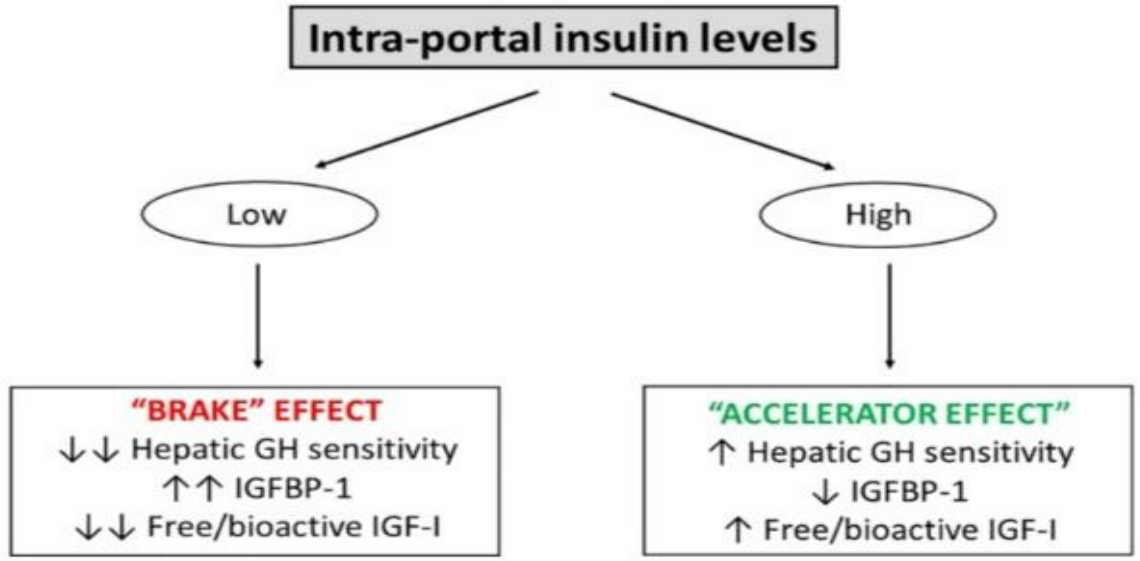

FIGURE 2
The ability of intra-portal insulin to serve as an "accelerator" and "brake" on hepatic GH sensitivity and free/bioactive IGF-I. In the setting of low intra-portal insulin levels (e.g., overnight fasting and T1D), there is a "brake" effect leading to reduced hepatic GH sensitivity, increased IGFBP-1 and reduced free/bioactive IGF-I. In the setting of high intra-portal insulin levels (e.g., feeding and obesity), an "accelerator" effect takes place leading to increased hepatic GH sensitivity. However, with regards to the "accelerator" and "brake" effects of insulin on serum free/bioactive IGF-I, it appears that the ability of high intra-portal insulin levels to increase ("accelerate") serum free/bioactive IGF-I activity is less pronounced compared to the ability of low intra-portal insulin levels to decrease ("brake") serum free/bioactive IGF-I activity. Two arrows indicate a marked effect, one arrow indicates a milder effect.

اثرات بیماری و کاهش وزن بر محور هورمون رشد و IGF1

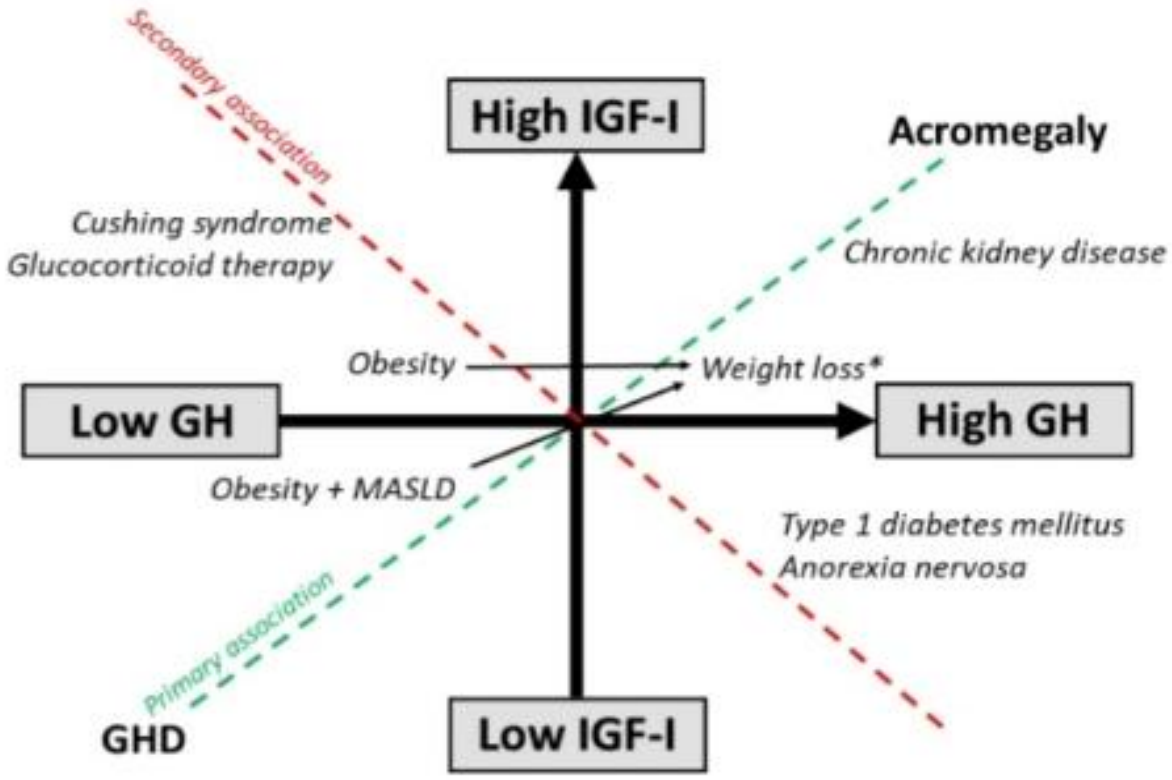

FIGURE 3
Effects of disease states and weight loss on the GH/IGF-I axis. *Weight loss induced by diet, glucagon-like peptide 1 receptor agonist therapy and surgery.

هورمون رشد به عنوان یک تعدیل کننده‌ی بالقوه‌ی کاهش بافت چربی احشایی به وسیله فعالیت ورزشی هوازی

Dr. Arman rastegari

Growth Hormone as a Potential Mediator of Aerobic Exercise-Induced Reductions in Visceral Adipose Tissue

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Keywords: growth hormone, cardiometabolic health, abdominal adipose tissue, cardiorespiratory fitness, obesity, aging

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INTRODUCTION

Obesity remains one of the leading causes of death worldwide and is a well-known risk factor for a myriad of non-communicable diseases including diabetes, cardiovascular disease, and a variety of cancers (Wolf and Colditz, 1998; Frühbeck et al., 2013). While the relationship between obesity and cardiometabolic risk is well-established, the location of adipose tissue, particularly in the abdominal region, is considered a greater predictor of metabolic dysfunction than total fat mass (Kahn et al., 2006). Central obesity, characterized by the excess accumulation of adipose tissue in the abdominal region, is strongly and independently correlated with metabolic syndrome and is assessed clinically through the measurement of waist circumference (Shen et al., 2006). Central adiposity can be further subcategorized into abdominal subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) (Snel et al., 2012). While the relationship between SAT and cardiometabolic risk remains equivocal, VAT has been established as a unique pathogenic fat depot. VAT acts as an endocrine organ by secreting adipocytokines and other vasoactive substances (Kanaya et al., 2004) and is associated with cardiometabolic risk independent of body mass index (BMI) or total body adiposity (Fox et al., 2007; Pak et al., 2016). Consequently, it is important to identify new, as well as further develop existing therapies to improve the management of obesity.

A landmark study in 1990 showed that exogenous growth hormone (GH) administered to older healthy males led to significant improvements in total body adiposity and lean body mass (Rudman et al., 1990). Since then, the results from further studies have shown that GH therapy can improve VAT, circulating lipid levels, and insulin resistance in adults with obesity and/or diabetes (Johannsson et al., 1997; Nam et al., 2001). Although studies like these highlighted the potential utility of GH therapy for the amelioration of age-related declines in metabolic function and body composition, further studies identified various side effects of GH therapy such as an increased likelihood of soft tissue edema, joint pain, carpal tunnel syndrome, gynecomastia, and diabetes (Liu et al., 2007). Consequently, exogenous GH therapy became typically reserved for individuals with GH deficiencies resulting from hypothalamic/pituitary disease (Clemmons et al., 2014). Despite this, there has since been increasing interest in identifying therapies, including lifestyle interventions, that increase physiologic GH release and action.

Exercise and diet modification are cornerstone therapies for the management of obesity-related disease. Interestingly, pooled data from clinical trials show that while exercise is less effective than diet modification for body weight loss, it appears to elicit superior reductions in VAT (Verheggen et al., 2016). This finding may partly be explained by exercise-induced changes in lipolytic hormones, such as GH, during and after exercise, which seem to target VAT (Berryman and List, 2017). Acute exercise has been shown to temporarily increase GH release in an intensity-dependent

مسیرهای مکانیستی تغییرات در هورمون رشد به وسیله فعالیت ورزشی هوازی

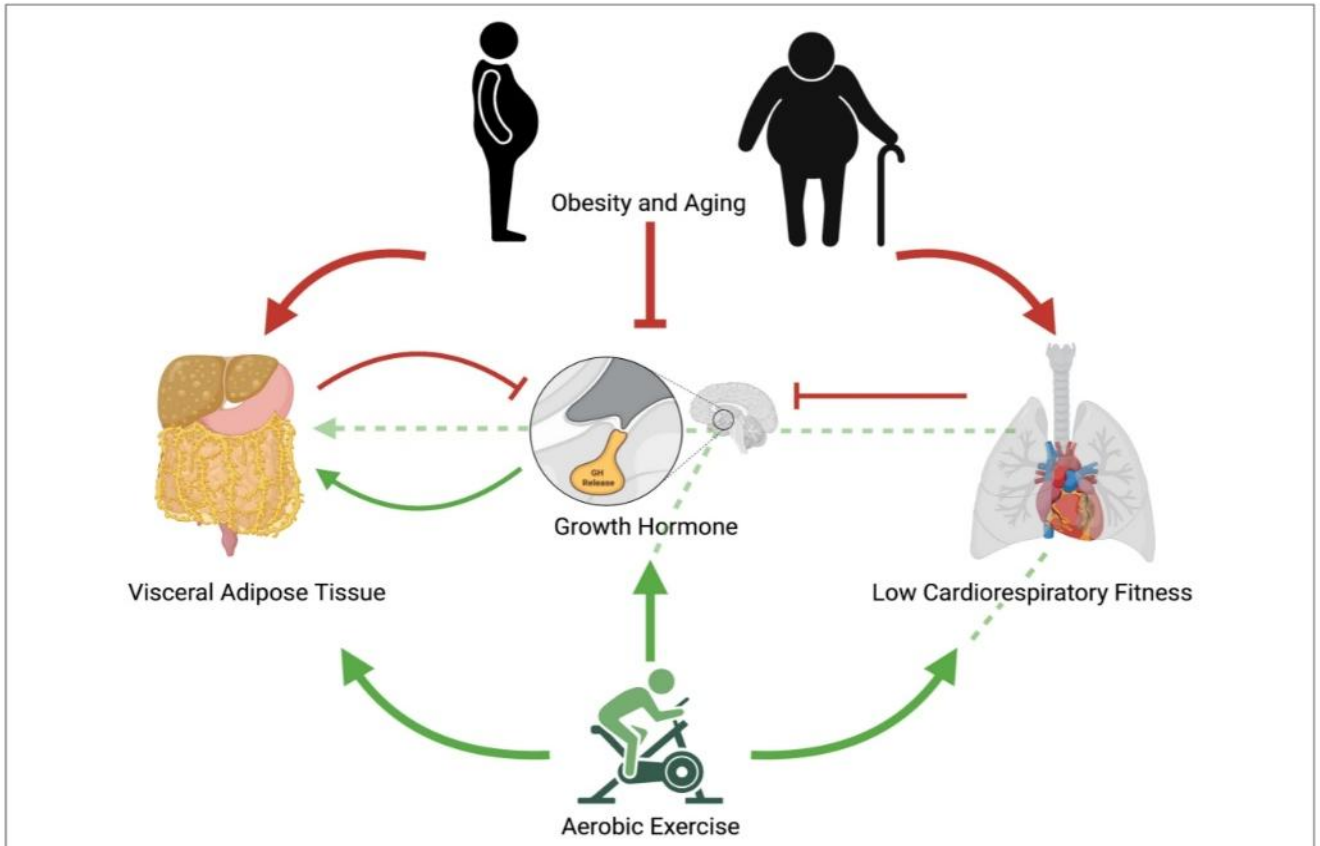

FIGURE 1 | Proposed mechanistic pathway for aerobic exercise induced changes in growth hormone release and ensuing reductions in visceral adipose tissue. Red arrows represent negative effects; green arrows represent positive effects; unbroken red lines represent inhibitory effects; broken green lines represent potential positive pathways of exercise-induced improvements in cardiometabolic health. Obesity and aging contribute to increased visceral adipose tissue, reduced cardiorespiratory fitness, and impaired growth hormone release. Low cardiorespiratory fitness further contributes to impaired growth hormone release as does visceral adipose tissue. Regular aerobic exercise reduces visceral adipose tissue, increases growth hormone release, and increases cardiorespiratory fitness. Regular aerobic exercise may also indirectly improve visceral adipose tissue through increases in cardiorespiratory fitness, which lead to improved growth hormone response and ensuing interactions in visceral adipocytes. Acute aerobic exercise elicits growth hormone release, which increases lipolysis of visceral adipose tissue.

اثر فعالیت ورزشی بر فاکتورهای رشدی در زنان بعد از یائسگی

RESEARCH Open Access

The impact of exercise on growth factors in postmenopausal women: a systematic review and meta-analysis

Dr. Arman rastegari

Yasaman Nasir[1], Mohammad Reza Hoseinipouya[1], Hesam Eshaghi[1,2] and Mohammad Hossein Rahimi[1,3*]

Abstract

Background Aging results in many changes in health status, body composition, muscle strength, and, ultimately, functional capacity. These changes coincide with significant alterations in the endocrine system, such as insulin-like growth factor-1 (IGF-1) and IGF-binding proteins (IGFBPs), and may be associated with many symptoms of aging. The objectives of this study is to investigate the potential influence of different types of exercise, such as resistance training and aerobic training, on IGF-1 and IGFBP-3 levels in postmenopausal women.

Methods Medline, Scopus, and Google Scholar databases were systematically searched up to November 2023. The Cochrane Collaboration tool was used to assess the risk of bias and the quality of the studies. The random-effects model, weighted mean difference (WMD), and 95% confidence interval (CI) were used to estimate the overall effect. Between-study heterogeneity was assessed using the chi-squared and I^2 tests.

Results Seventeen studies were included in the present systematic review and 16 studies were included in the meta-analysis. The pooled results from 16 studies (21 trials) with 1170 participants examining the impact of exercise on IGF-1 concentration showed a significant increase in IGF-1, and the pooled results among six studies (trials) showed a significant decrease in IGFBP-3 concentration (730 participants). In addition, resistance training and aerobic training had a significant effect on increasing IGF-1 concentration post-exercise compared with placebo.

Conclusion Based on this meta-analysis, Women who have completed menopause and followed an exercise routine showed changes in IGF-1 and IGFBP-3 levels that can indirectly be associated with risk of chronic age-related conditions.

Keywords IGF-1, Insulin-like growth factor-1, Insulin-like growth factor binding proteins, IGFBP3, Postmenopause, Exercise, Meta-analysis

Consensus statement

OPEN ACCESS

Female athlete health domains: a supplement to the International Olympic Committee consensus statement on methods for recording and reporting epidemiological data on injury and illness in sport

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ABSTRACT

The IOC made recommendations for recording and reporting epidemiological data on injuries and illness in sports in 2020, but with little, if any, focus on female athletes. Therefore, the aims of this supplement to the IOC consensus statement are to (i) propose a taxonomy for categorisation of female athlete health problems across the lifespan; (ii) make recommendations for data capture to inform consistent recording and reporting of symptoms, injuries, illnesses and other health outcomes in sports injury epidemiology and (iii) make recommendations for specifications when applying the Strengthening the Reporting of Observational Studies in Epidemiology-Sport Injury and Illness Surveillance (STROBE-SIIS) to female athlete health data.

In May 2021, five researchers and clinicians with expertise in sports medicine, epidemiology and female athlete health convened to form a consensus working group, which identified key themes. Twenty additional experts were invited and an iterative process involving all authors was then used to extend the IOC consensus statement, to include issues which affect female athletes.

Ten domains of female health for categorising health problems according to biological, life stage or environmental factors that affect females in sport were identified: menstrual and gynaecological health; preconception and assisted reproduction; pregnancy; postpartum; menopause; breast health; pelvic floor health; breast feeding, parenting and caregiving; mental health and sport environments.

This paper extends the IOC consensus statement to include 10 domains of female health, which may affect female athletes across the lifespan, from adolescence through young adulthood, to mid-age and older age. Our recommendations for data capture relating to female athlete population characteristics, and injuries, illnesses and other health consequences, will improve the quality of epidemiological studies, to inform better injury and illness prevention strategies.

INTRODUCTION

Injury and illness surveillance is a fundamental element in our efforts to protect the health of athletes. Hence, in 2020 the IOC published a consensus statement that describes standards to monitor and report health problems in sports.[1] This consensus aims to ensure consistency in the definitions and methods used, and to guide the collection of comparable epidemiological data across studies. Since then, several sports-specific[2–4] and population-specific[5] extensions have been produced, further supporting the appropriate and consistent application of the IOC recommendations across different settings.

Consensus statements were traditionally developed and applied to record injuries without focused consideration of the female athlete.[6] Indeed, the 2020 IOC consensus statement[1] does not mention the female athlete in its recommendations. Historically, injury and illness data that inform the development of injury surveillance systems and consensus statements are typically from male athletes, with such systems then being more frequently used in men's sport.[6] However, female athletes have additional specific biological, sociocultural and environmental considerations that could impact sports exposure or health outcomes. For example, circulating concentrations of both endogenous and exogenous oestrogen and progesterone influence several health conditions,[7] which vary with events (eg, puberty, pregnancy, menopause) and across life stages (eg, adolescence, young adulthood, mid-age). Postpuberty population characteristics are rarely reported but may influence injury and illness onset and recovery.[8–10] Breast health issues likely go unreported as, like other body regions, the breast does not have a specific diagnosis category in commonly used coding systems[11] and, until 2020, did not appear in these coding systems at all. Therefore, female-specific health risks across the lifespan remain largely undocumented, with limited quality data on female athlete health.

قلمرو سلامت زنان ورزشکار

Table 1 Female health domains and their definitions

Health domain	Domain abbreviation	Definition
Menstrual and other gynaecological health	D-MG	The health of the menstrual cycle and female reproductive organs and tract.
Preconception/Assisted reproduction	D-AR	Undergoing treatments to assist in becoming pregnant without sexual intercourse.[33]
Pregnancy	D-PR	The condition of being pregnant.[34]
Postpartum	D-PO	Immediately follows childbirth until 2 years* postchildbirth.
Menopause	D-ME	The transitional time between perimenopause and postmenopause, when menstruation surceases.[7]
Breast health	D-BH	The health of the mammary glands.[34]
Pelvic floor health	D-PF	The physical and functional integrity of the pelvic floor unit through the life stages of an individual (male or female).[35]
Breast feeding, parenting and caregiving	D-BP	Providing direct care for another individual who needs help taking care of themselves (eg, a baby, child, the elderly, chronically ill), including suckling milk from a mother's breast.
Mental health†	D-MH	The psychological, emotional and social well-being[36] of an athlete.
Sport environment†	D-SE	The physical and social context within which athletes train and compete.

*Based on WHO breastfeeding recommendation[37] and mental health outcomes.[38]
†This health domain is particularly prevalent but not unique to female athletes only. Consideration should be given to all athletes.

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REVIEW ARTICLE

Recommendations and Nutritional Considerations for Female Athletes: Health and Performance

Bryan Holtzman[1,2] · Kathryn E. Ackerman[2,3,4]

Accepted: 16 June 2021 / Published online: 13 September 2021

Abstract
Optimal nutrition is an important aspect of an athlete's preparation to achieve optimal health and performance. While general concepts about micro- and macronutrients and timing of food and fluids are addressed in sports science, rarely are the specific effects of women's physiology on energy and fluid needs highly considered in research or clinical practice. Women differ from men not only in size, but in body composition and hormonal milieu, and also differ from one another. Their monthly hormonal cycles, with fluctuations in estrogen and progesterone, have varying effects on metabolism and fluid retention. Such cycles can change from month to month, can be suppressed with exogenous hormones, and may even be manipulated to capitalize on ideal timing for performance. But before such physiology can be manipulated, its relationship with nutrition and performance must be understood. This review will address general concepts regarding substrate metabolism in women versus men, common menstrual patterns of female athletes, nutrient and hydration needs during different phases of the menstrual cycle, and health and performance issues related to menstrual cycle disruption. We will discuss up-to-date recommendations for fueling female athletes, describe areas that require further exploration, and address methodological considerations to inform future work in this important area.

Key Points

Female athletes should aim for energy availability (EA) of 45 kcal·kg^{-1} fat-free mass·day^{-1} for optimal health and performance; optimizing nutrient composition based on menstrual cycle phase is ineffective without the requisite energy for basic functioning.

Micronutrient deficiencies are common in female athletes, particularly in iron, vitamin D, and calcium; nutritional strategies should be used to prevent these deficiencies, including increasing consumption of diverse foods and potential supplementation.

Micro- and macronutrient requirements, as well as hydration needs, may change during various phases of the menstrual cycle as a result of hormonal fluctuations.

1 Introduction

Female athletes make up nearly 50% of sports participants. Unfortunately, research into optimizing nutrition for health and performance specific to female physiology is lacking. In this review, we will describe the challenges of studying women, the potential pitfalls of applying research from males to females, provide recommendations for adequate caloric intake, describe sequelae of insufficient caloric intake, propose a simple framework for designing nutrition plans for female athletes, and outline basic recommendations for nutrition plans for female athletes with resources for further reading.

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توصیه ها و ملاحظات تغذیه‌ای زنان ورزشکار برای سلامت و عملکرد

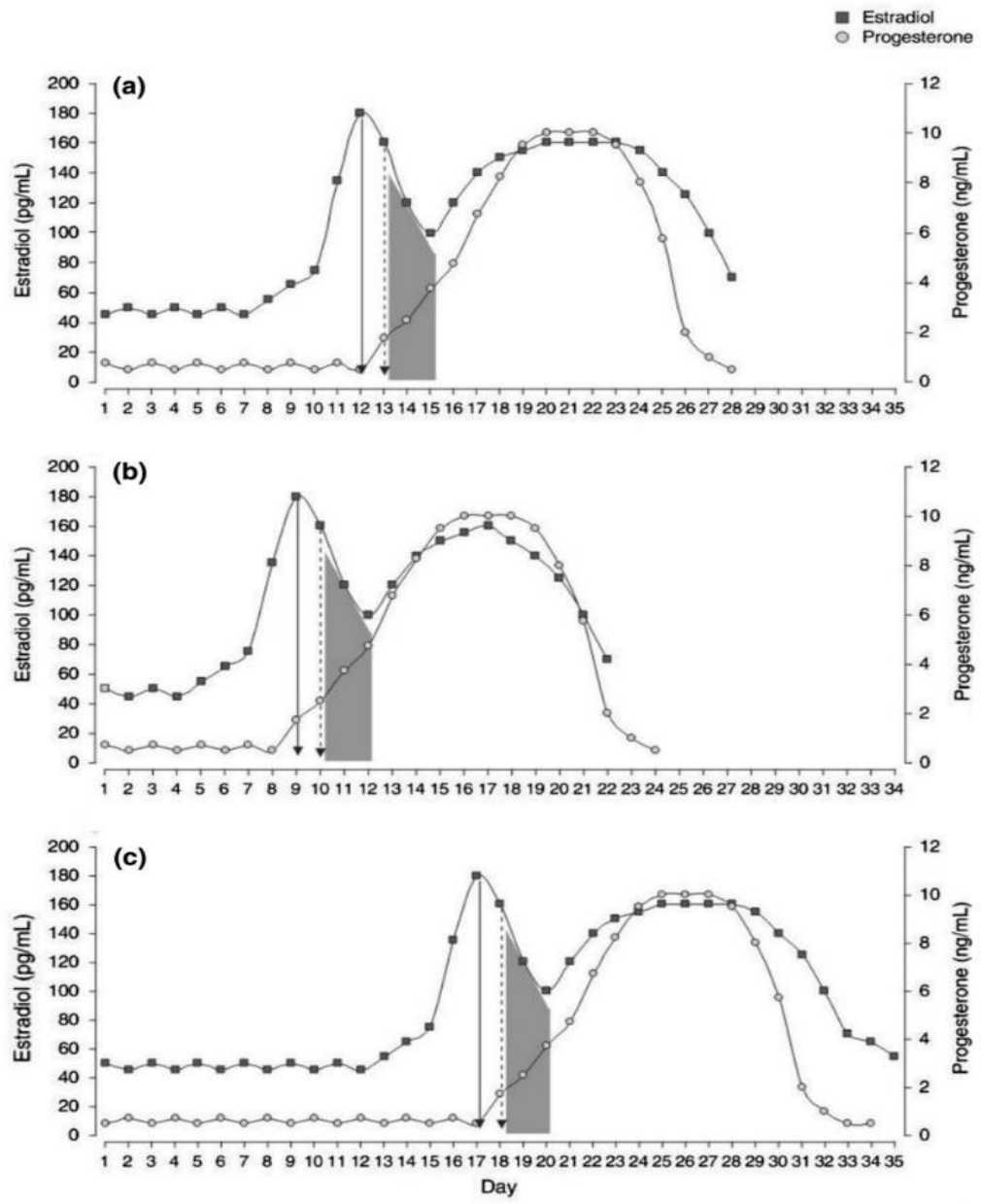

Fig. 1 Hypothetical examples of the hormonal profiles of three eumenorrheic women with different cycle lengths. **a** 28-day cycle; **b** 22-day cycle; **c** 35-day cycle. Solid arrow indicates estradiol peak; dashed arrow indicates luteinizing hormone peak; shaded area indicates ovulation. Reproduced from Vescovi with permission [3]

پروفایل هورمونی ۳ طیف زن یومنوره با طول دوره قاعدگی ۲۸ روزه ۲۲ روزه و ۳۵ روزه

Dr. Arman rastegari

عواقب سلامتی کاهش انرژی در دسترس در فعالیت ورزشی در زنان

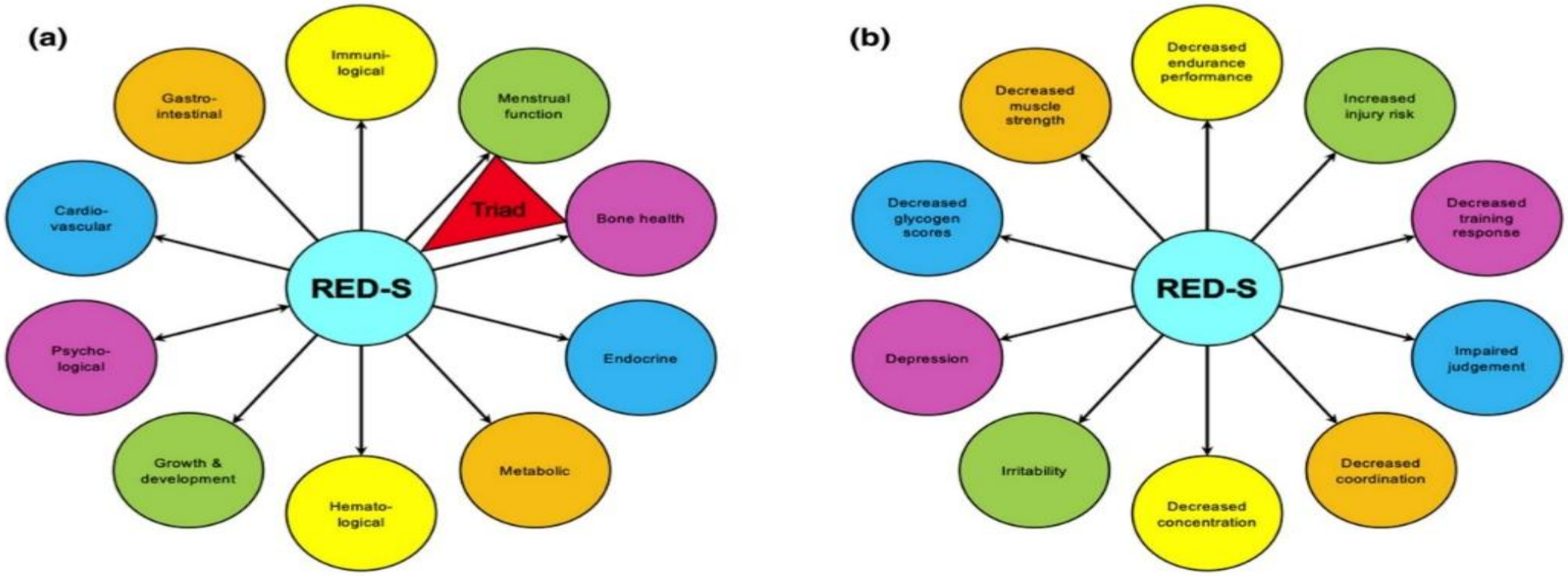

Fig. 2 **a** Health consequences of relative energy deficiency in sport (RED-S); **b** performance effects of RED-S. Adapted from Constantini (with permission) [25]

کمبود آهن در زنان ورزشکار

Table 2 Proposed guidelines by Peeling and colleagues for iron deficiency severity in athletes [112]

Stage 1: Iron deficiency	Iron stores in bone marrow, liver, spleen depleted Ferritin < 35 ng mL^{-1} Hemoglobin > 11.5 ng dL^{-1} Transferrin saturation > 16%
Stage 2: Iron-deficient non-anemia	Erythropoiesis diminishes as the iron supply to the erythroid marrow is reduced Ferritin < 20 ng mL^{-1} Hemoglobin > 11.5 ng dL^{-1} Transferrin saturation < 16%
Stage 3: Iron-deficient anemia	Hemoglobin production falls, resulting in anemia Ferritin < 12 ng mL^{-1} Hemoglobin < 11.5 ng dL^{-1} Transferrin saturation < 16%

Dr. Arman rastegari

میزان بهینه ویتامین D در زنان ورزشکار

Table 3 Institute of Medicine levels of vitamin D concentrations [130]

25-OH-vitamin D concentration (nM)	Vitamin D status
< 12.5	Very deficient
12.5– < 30	Deficient
30–50	Inadequate
> 50	Adequate
> 180	Toxic

Dr. Arman rastegari

بایومارکرهای بررسی میزان هیدراتاسیون

Table 4 Biomarkers of hydration status. Adapted from ACSM guidelines with permission [138]

Measure	Practicality	Validity (acute vs. chronic changes)	EUH cut-off
TBW	Low	Acute and chronic	$<2\%$
Plasma osmolality	Medium	Acute and chronic	<290 mOsm
Urine specific gravity	High	Chronic	<1.020 g mL^{-1}
Urine osmolality	High	Chronic	<700 mOsm
Body weight	High	Acute and chronic[a]	$<1\%$

EUH euhydration, *TBW* total body water

[a]Potentially confounded by changes in body composition during very prolonged assessment periods

پایان بخش اول

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