

## Exercise as a stressor to the human neuroendocrine system

Anthony C. Hackney

Endocrine Section, Applied Physiology Laboratory, Department of Exercise and Sport Science,  
Department of Nutrition, School of Public Health and Medicine,  
University of North Carolina, Chapel Hill, North Carolina, USA

**Key words:** hormones; physical activity; stress.

**Summary.** This article is a brief review on the effects of exercise stress upon the major hormonal components that make up the human neuroendocrine system. The review is organized into four major topics, which are presented and dealt with in the form of questions. These questions are: 1) Is exercise a stressor to the neuroendocrine system? 2) Why would exercise be a stressor to the neuroendocrine system? 3) What are the effects of exercise as a stressor upon the neuroendocrine system? 4) Is exercise always a stressor to the neuroendocrine system? These questions are addressed and answered in the article in an attempt to provide fundamental background knowledge on neuroendocrine response to exercise.

### Introduction

Men and women who currently compete in sporting events are swimming, running, cycling, and skating faster than ever before. This is attested to by the fact that world records in many sporting events are being broken on nearly an annual basis. The factors that contribute to this improvement in human performance are many, but perhaps the most important, is the greater level of exercise training that athletes are performing in our modern era. This enhanced volume of exercise training results in an extreme amount of stress being placed on the human organism. Such stress induces many physiological changes and adaptations that are highly beneficial to the sporting persons and their ability to perform exercise (1). In humans, one physiological system that is extremely responsive to the stress of exercise and exercise training is the neuroendocrine system (2–4).

The purpose of this article is to present a concise synopsis of the effects of exercise (*i.e.*, physical activity) upon the major hormonal components that help comprise the human neuroendocrine system. The intent here is for this article to serve as a “primer” for those persons who may have a limited background in the area of “exercise physiology” and in particular the neuroendocrine responses to exercise. The discussion is meant as an introduction to the topic, and thus the level of depth is delimited to just major aspects and key points. The review is organized into four major topics, and these topics are presented in a didactic approach using questions which are proposed to the

reader and then answered in the course of the article.

### Question 1 – Is exercise a stressor to the neuroendocrine system?

In the title to this article, it is implied that exercise is stressful to human beings. Nonetheless, the first question (noted above) is proposed in order to address whether this is a valid assumption. To answer this question, it is perhaps best to begin by providing a definition of what is “stress.” The *Merriam-Webster’s English Dictionary* supplies 11 different definitions of the word “stress” (5). What appears to be the most appropriate to this article and in the physiological context is the following:

“Stress is a specific response by the body to a stimulus that disturbs or interferes with the ‘normal’ physiological equilibrium of an organism.”

A key word in this definition is “normal,” and it is important in the context of this article to recognize that stress responses would be considered those beyond the typical, daily (*i.e.*, natural) basal physiological responses and events, but not events that would be considered “distressful” in nature (*e.g.*, to observe a murder occurring). Furthermore, the above definition can be enhanced and expanded upon by the answering of the logical follow-on question, “What is a stressor?” The *Merriam-Webster’s Dictionary* provides this definition:

“Stressor is an activity, event, or stimulus that causes stress.”

A brief search of the research literature would

reveal that there are thousands of published research studies which have shown the “normal” neuroendocrine, resting equilibrium (*i.e.*, homeostasis) is disturbed by a bout of exercise (*i.e.*, a executed period of physical activity in which the metabolic rate is significantly elevated above rest for a period of time) (6). These research findings, when compared to the definition above, would support the answer that, “yes,” exercise is a “stressor” to the neuroendocrine system (*nota bene*, the most applicable type of exercise that the findings in this article applies to are dynamic, large muscle groups, so-called aerobic activities; *e.g.*, running and walking).

### Question 2 – Why would exercise be a stressor to the neuroendocrine system?

To answer to this question, one must first come back to what is the basic physiological role of the neuroendocrine system (NES; Table 1 contains a list of abbreviations used throughout this article). In the classic definition, the NES is referred to as a series of ductless glands (and neurons) that release chemical messengers called “hormones” into the circulatory system. These hormones in turn act to aid in the control and regulation of various physiological processes and organ system functions, such as metabolism, the cardiovascular-respiratory system, hydration status, thermoregulation, digestion, growth-maturation, and reproduction (7, 8).

Evidently, many of these physiological processes and organ systems just noted are essential to the body’s ability to perform exercise. Put another way, changes in many of these bodily processes are necessary in order to bring about the physiologic accommodation required to meet the demands of exercise (2). The NES is a primary regulator and modifier of many of these accommodations (9). Thus, as organisms we need the NES to respond to the stress of exercise in order to allow the human body to be able to accommodate physiologically and perform exercise.

### Question 3 – What are the effects of exercise as a stressor upon the neuroendocrine system?

It is necessary, before answering this question, to address several points in order to appreciate fully the complexity of the answer. First, the word “exercise” does not have a universal meaning for all researchers, nor is it translated uniformly in the research literature. For example, exercise can be thought of in its “acute” form when one is speaking about a single bout of exercise (*e.g.*, 30 minutes of brisk walking at a significantly elevated heart rate), or it can be thought of in its more

**Table 1. The following are abbreviations used throughout this article as well as ones typically found in the area of endocrinology**

Name	Abbreviations
Adrenocorticotrophic hormone	ACTH
Atrial natriuretic peptide	ANP
Arginine vasopressin	AVP
β-Endorphin	β-END
Central nervous system	CNS
Corticotropin releasing hormone	CRH
Decrease	↓
Delta ( <i>i.e.</i> , change)	Δ
Follicle-stimulating hormone	FSH
Growth hormone	GH
Growth hormone releasing hormone	GHRH
Increase	↑
Insulin-like growth factor-1	IGF <sub>1</sub>
Luteinizing hormone	LH
Metabolic clearance rate	MCR
Neuroendocrine	NE
Neuroendocrine system	NES
Peripheral nervous system	PNS
Prolactin	PRL
Sympathetic nervous system	SNS
Thyrotropin-releasing hormone	TRH
Thyroid-stimulating hormone	TSH
Thyroxine	T <sub>4</sub>
Triiodothyronine	T <sub>3</sub>

“chronic” form where one is dealing with the effects of a exercise training program (*e.g.*, several weeks of near daily exercise bouts, which have an accumulative physiological adaptation effect). These distinctions in terminology are important since the definition and categorization of responses of the NES to exercise can vary greatly depending upon which form of exercise you are focusing upon. Table 2 presents a summary and explanation of some of the common terminologies used to describe exercise forms and types.

Secondly, it is important to understand how researchers go about quantifying and assessing the “response of the NES” to exercise. The most typical and accepted way for clinicians and researchers to do this assessment is to measure circulating hormonal concentrations in the blood (or perhaps saliva and/or urine specimens) from the endocrine gland of interest. The findings noted in this article are principally based upon such assessment. However, the blood assessment technique has limitations in its ability to tell researchers what is exactly going on within a very dynamic

**Table 2. A categorization and definition of exercise intensity and exercise-type relative to exercise studies of physically fit individuals. In quantifying exercise intensity, it is typical to express it as a relative percentage of an individual's maximal oxygen uptake ( $VO_{2max}$ ; i.e., maximal aerobic capacity)**

Category term	Relative intensity (% $VO_{2max}$ )	Energy pathway predominating	Typical duration	Other terminology
Light or easy exercise	<35%	Aerobic	>30 min	Short-term, submaximal
Moderate exercise	35–70%	Aerobic	30–180 min	Submaximal prolonged
Heavy exercise	>70%	Aerobic – Anaerobic	<120 min	Submaximal prolonged, high intensity
Maximal exercise	100%	Aerobic – Anaerobic	<15 min	Maximal or max, high intensity
Supramaximal exercise	>100%	Anaerobic	<1 min	Sprints, power

Based upon work by C. Bouchard *et al.* in *Exercise, Fitness, and Health: A Consensus of Current Knowledge* (Champaign, IL. Human Kinetics, 1990) as reported by A. C. Hackney and J. Dobridge (reference 20).

system. For instance, changes in circulating hormonal concentrations are brought about not only by changes in glandular production and secretion, but also by changes in the metabolic clearance rate (MCR) for the hormone (8). During an exercise bout MCR can vary either through changes in target tissue uptake or hormonal degradation. Additionally, for some hormones the binding affinities of carrier protein change during exercise thereby making more or less of the hormonal free form available in the circulation (10). Thus, by measuring just blood hormonal concentration values, it is sometimes difficult to determine what mechanism is causing the NES response observed. Furthermore, in some situations the hormonal response observed is being influenced by hemoconcentration or hemodilution events associated with plasma fluids shifting in and out of the vascular bed during exercise (11). Consequently, when assessing the NES responses to exercise by examining circulating hormonal concentrations, it is important to try to take into account such factors as just mentioned above, if there is an attempt to discern “what” is really happening and “why” it is happening physiologically. To try and overcome of these limitations, some endocrinologists utilize area-under-the-curve analysis to get a more “dynamic” picture of hormonal events and endocrine function. This process involves taking multiple blood samples (or saliva in some cases) over a several hour period and then plotting the concentration responses and integrating the response curve, and the integrated response is evaluated (12). This technique has promise for providing some degree of further information; however, it is still influenced by some of the same factors

noted above and can be considered more invasive due to the multiple specimen collection. Another technique of assessment involves a pharmaceutical challenge to an endocrine gland. That is, a drug or compound that stimulates hormonal release is infused into an individual, and then multiple blood (or saliva) samples are collected afterwards to evaluate the endocrine glands responsive by looking at hormonal changes. This technique provides excellent information on the responsiveness (*e.g.*, production and secretions rates) and can serve as a good clinical tool but can have higher health risks and invasive aspects as well as also being influenced by some of the above factors.

Next, in order to answer the third question posed in the title to this section, initially it is convenient to address the NES responses to “acute exercise” (*i.e.*, a single bout) and later in the article to discuss “chronic exercise” (*i.e.*, exercise training). In a single, acute exercise bout, there are several components that dictate what will be the magnitude and direction of the NES response. The key components of “acute exercise” are the *intensity* at which the exercise is performed and the *duration* of the individual exercise bout (1, 13, 14).

Typically the greater the intensity of exercise, the greater the degree of stress placed upon the NES and the more exacerbated the hormonal response becomes, meaning there are greater disturbances in the circulating hormonal concentrations (1, 13, 15). The exact nature (*i.e.*, direction) of the responses one might see in the circulating hormonal concentrations is varied. Fig. 1 displays a generalized representation of such hormonal responses with an exercise bout of progres-

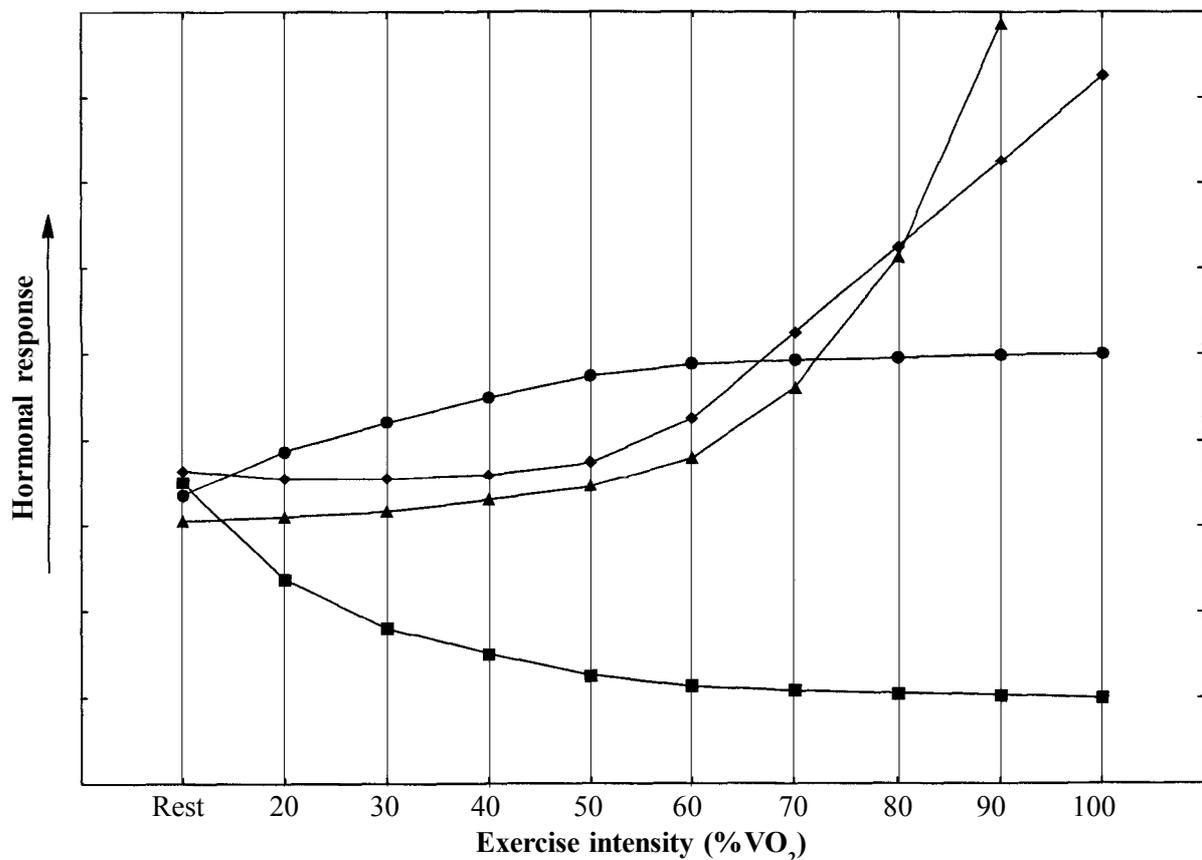
sively increasing intensity. As can be seen in the figure, hormonal response can take several forms. It could be linear or curve linear in nature or involve a threshold intensity needing to be reached before any response is observed (12). In addition, the response is not always necessarily increased circulating amounts of hormone, as some hormones can decrease in their levels in response to acute exercise (see Fig. 1).

Relative to duration, typically extending the length of time of an exercise bout at any given intensity tends to amplify an NES response; that is, as a person exercises longer and longer, one can see a gradual and further increase (or decrease; depending upon the specific hormone being examined) in the circulating hormonal levels (15). Furthermore, in some situations after the initial change in the hormonal concentration, there can be a plateau (*i.e.*, steady state) of the response even as exercise duration continues.

In addition to the intensity-duration components that can influence the NES response to an acute exercise bout, there are several other factors that can modify the response such as mode of activity, envi-

ronmental conditions, age, gender, nutrition, circadian rhythms, genetics, and exercise training status. Each of these factors is briefly addressed below.

Some excellent research work by the North American investigator, William Kraemer, and his associates over the years has helped researchers to recognize that the hormonal responses to various forms of resistance exercise can differ from that of an aerobic exercise sessions, such as running or bicycling for examples (16–18). While there is limited research in exercise endocrinology dealing with swimming, some evidence suggests it may produce some differing NES responses in some respects to other modalities of activities (2, 3, 19). This mode of activity differences can be due to a variety of features associated with the aerobic and anaerobic nature of the activities, such as muscle fiber recruitment patterns, activation of energy production pathways, and utilization of different substrates, postural differences, and hemodynamic disparities. For more explanation and details on these mode phenomena, the reader is directed to select research references (see 12, 17, 20).



**Fig. 1.** Differences in the type of hormonal responses to an exercise bout of increasing intensity

The Y-axis is an arbitrary scaling used only to indicate direction of hormonal changes. The X-axis represents exercise intensity in percentage of maximal oxygen uptake (%VO<sub>2</sub>).

The ambient environmental condition, a person is exposed to, can have great influences on the NES. For example, the more ambient temperatures deviate from what is typically normal for a person, or their heat storage status is some how altered, or they experience hypoxemia upon going to altitude, the more the hormonal response to exercise can become distorted from normal (21, 22). In some situations, environmental factors can dramatically amplify certain hormonal responses; in other situations, the ambient environment may provoke hormonal changes that are usually not seen with an exercise bout of a respective intensity or duration (9).

Age and maturation can also be influential factors. The very young and older mature individuals have certain NES responses to exercise that vary (*e.g.*, typically lessened) from those exhibited by the young to middle-aged adults (*i.e.*, college-aged) which usually are used in many exercise studies (12). These differing responses may represent an incomplete developmental process of the NES in children or apoptotic processes occurring in certain endocrine tissue of the elderly (23, 24).

The issue of gender differences in NES responses typically revolves around the influence of the menstrual cycle and the reproductive hormones in women upon other hormones and how that may in turn modify their NES response to exercise (25). However, certain nonreproductive hormones can also be vastly different between males and females too, for example the adipocyte hormone leptin. Regrettably, too few researchers seem to be mindful of such gender differences in hormonal concentrations when studying exercise physiology. For more information on this topic, the reader is directed to references 1, 12, and 25.

Nutrition status and dietary intakes that deviate greatly from normal have been shown to influence the NES responses to exercise, causing some hormonal responses to be augmented and others to be attenuated (2, 19).

Many hormones exhibit circadian rhythms and diurnal variations in their circulating concentrations. Work by several research groups has shown that the relative magnitude of the NES response to exercise for some hormones is variable based upon the time of day in which the exercise takes place – this is even when accounting for differences in resting-basal concentrations, circadian pattern effects of the different times of day, and standardizing the exercise amount (25–27). Thus, exercise of the same duration and intensity performed at two different times of the day

could provoke several magnitudes of difference in hormonal response. Interestingly, some hormones show a greater response to exercise in the morning than afternoon, and the responses of some hormones are opposite; however, this chronobiology issue has not been studied fully, and additional work is necessary before a complete summarization of hormone, exercise response, and time-of-day effects can be summarized adequately.

There is also genetic variation among people and this can result in some degree of NES responses that are different than expected. Atko Viru of Estonia has been a proponent of this factor influencing research outcomes in exercise endocrinology for many years (22, 24). Researchers may need to examine this factor much more closely in the future to better understand the hormonal response to the stress of exercise.

Lastly, it is important to recognize that exercise training itself can modify the subsequent exercise response to an acute exercise bout after training adaptations have occurred. In most cases, the NES responses to exercise become attenuated to some degree by prolonged exposure to appropriate levels of exercise training (as noted earlier, this point will be addressed later in the article) (2, 28).

This has been a long preamble to get to the answer for the third question, “What are the effects of exercise as a stressor upon the neuroendocrine system?” The discussion thus far should make the reader aware that multiple factors could be put together in different combinations to answer that specific question concisely. Therefore, for the sake of conciseness, only a single exercise example (*i.e.*, bout) will be discussed and explained in detail to illustrate the point of what can happen. This example involves looking at an exercise bout involving a dynamic, large muscle group, aerobic activity (*e.g.*, running) for roughly 30- to 45-minute duration, at a moderately hard intensity corresponding approximately to 70% of an individual’s maximal oxygen uptake (this intensity would correspond to about 80% of an individual’s heart rate maximum).

In addition, for organizational purposes, a concept from Henrik Galbo is used to divide the NES responses into phases (2, 3). The first phase is immediately at the onset of exercise, taking just seconds to occur. It consists primarily of increased sympathetic nervous system (SNS) activation with the onset of body motion. The increased SNS activity could also be a result of anticipation to the stress of the ensuing exercise. This increased SNS activity results in catecholamine release at target tissues directly, as well as elevations

in circulating catecholamine from so-called sympathetic “spill-over” effects. This effect is further amplified by the sympathetic connection to the adrenal medullary gland which in turn adds to the circulating catecholamine response (29). Concurrent with these sympathetic-adrenal actions, pancreatic insulin secretion begins to be inhibited while glucagon secretion becomes stimulated (Fig. 2, phase 1). The entire process to this point seems to involve feed-forward mechanisms of the central nervous system to drive these initial responses; although, the events are also modified by peripheral afferent neural input from sensory receptors of skeletal muscle (29, 30).

Next would be the intermediate or secondary phase which takes longer to develop, but is still typically very fast beginning usually in much less than a minute. In this stage, the hypothalamus begins the process of releasing its hormones such as CRH, GHRH, and TRH (see Table 1 for abbreviation definitions) in an attempt to provoke changes at the pituitary so this endocrine gland will in turn be stimulated to release its hormones (see Fig. 2, phase 2). As the pituitary begins to respond to the hypothalamic stimulus, its “trophic hormones” begin to be added to the circulation, and these hormones begin to act upon their specific peripheral target glands to stimulate additional hormonal release (7, 8). One of the fastest acting in this cascade of events is the hypothalamic-pituitary-adrenocortical interaction where CRH brings about ACTH release and that in turn brings about cortisol release (Fig. 2, phase 2). When several endocrine glands are linked together in such an interacting regulatory capacity, they are typically referred to as an “axis.” The above example is thus referred to as the “hypothalamic-pituitary-adrenocortical axis” (8, 31).

As exercise continues, there is a transition beyond the intermediate phase into a third phase of response which is a more prolonged state. In the phase 3 panel of Fig. 2, the responses of the sympathetic-adrenal axis are being augmented by other hormones from the anterior-posterior pituitary and the peripheral endocrine glands subordinated to pituitary regulation (2, 3, 8). Additionally, during this phase the skeletal and cardiac muscles themselves begin to release their select cytokines, hormonal-like agents, into the circulation (9, 32, 33).

Neural factors have been the primary stimuli regulating the NES response up to this point. However, now in the third phase, there is an ever-increasing influence of the humoral and hormonal factors that regulate the overall responses due to the changes in the

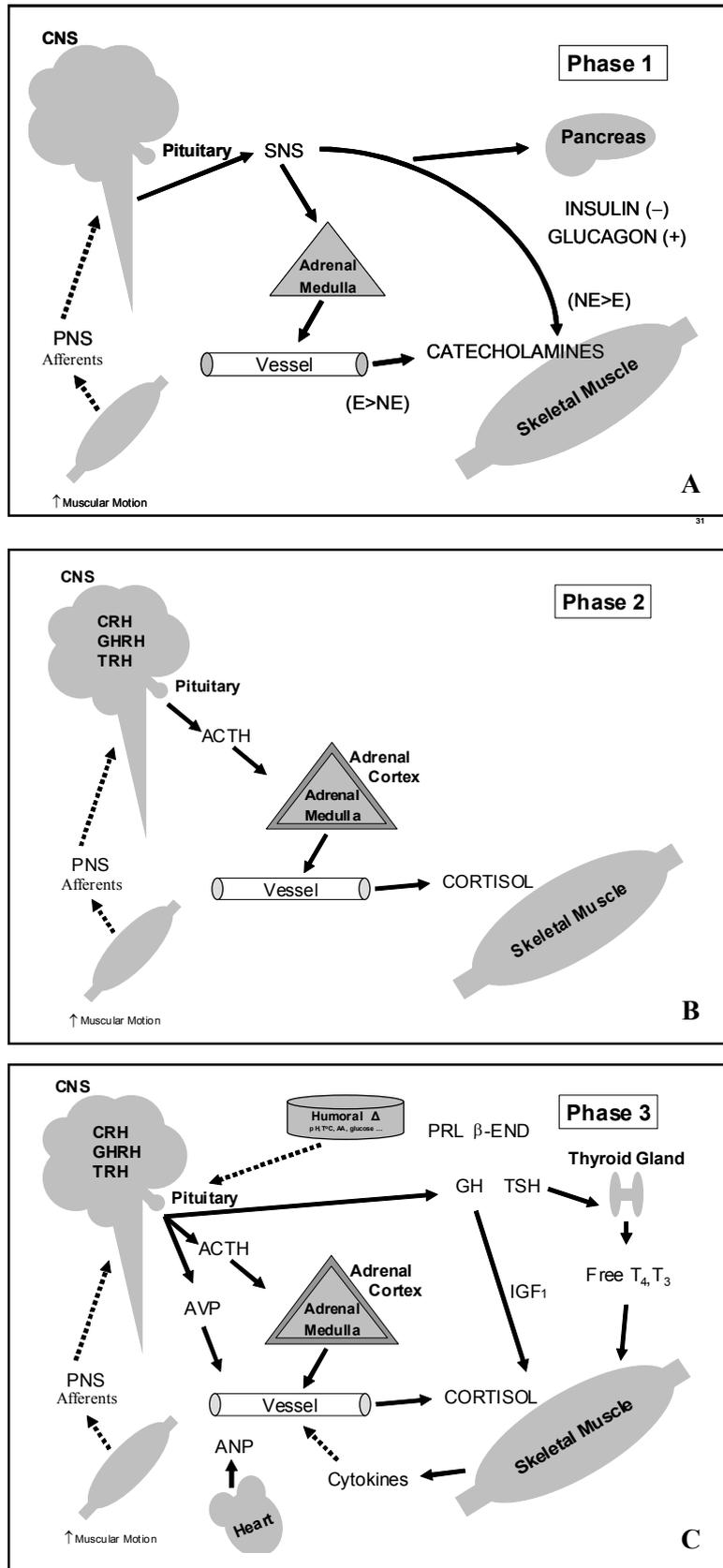
“internal milieu.” This shifting of primary regulators allows an increasing reliance upon feedback rather than feed-forward control mechanism. The influence of humoral and hormonal stimuli-types in modulating the hormonal levels is magnified as the exercise duration is extended, and energy substrate availability causes shifts in fuel usage (*i.e.*, ↓ carbohydrate ⇒ ↑ lipid), or hydration issues (*i.e.*, hemoconcentration and/or dehydration) begin compromising the thermoregulatory ability and heat storage (34, 35). By the completion of this example exercise bout, many of the hormones depicted in Fig. 2 would easily display between a 2- to 10-fold change in their circulating concentrations from those levels observed at rest before exercise.

As the exercise ends, we come to the fourth and last phase – recovery. In recovery, the NES attempts to return to a normal resting homeostasis, as are the other physiological systems of the body. The duration of the recovery period is totally dependent upon the severity of the exercise bout; that is, how much stress the NES has been subjected too (36, 37). Opinions exist that this phase actually deserves to be split into two distinct phases. That is, research from several investigators indicates that there is most certainly an early recovery that occurs within the first few minutes to hours after exercise, followed by a late recovery phase lasting many hours in length for certain hormones after exercise ends (26, 38)

The illustrative example just worked through is by no means complete and exhaustive, as already mentioned, the exact and specific NES responses would depend upon how the major components making up the exercise bout are manipulated (*i.e.*, intensity and duration). To provide a more complete summary of exercise NES events the reader is directed to Table 3. This table is modified from a book chapter reviewing exercise endocrinology developed by the article author and a colleague (12). The table indicates the direction of hormonal change and the approximate magnitude of hormonal change for different exercise activities at a variety of intensities.

#### **Question 4 – Is exercise always a stressor to the neuroendocrine system?**

This question is dealing with the topic of exercise training or “chronic exercise.” A review of the literature supports the idea that the answer to this question is most certainly, “yes” – exercise is always a stress to the NES. However, it appears that exercise can become less of a stressor over time as one is repetitively exposed to exercise. As mentioned earlier, the



**Fig. 2.** Regulation of hormonal secretion by SNS, pituitary gland (and subordinate glands), and muscle tissues at: (A) onset phase 1, (B) intermediate phase 2, and (C) prolonged phase 3. This example is for an exercise bout of moderate intensity and duration (reference 12).

**Table 3. Magnitude and direction of responses of select hormones to exercise bouts of different mode-type and duration (reference 12)**

Hormone	Exercise type – situation					
	anticipation	short-term submaximal	high intensity	prolonged exercise	resistance exercise	aerobic training
ACTH	+	+	++	++	++	–
Aldosterone	0	+	++	++	+	0
AVP	0	+	++	++	?	0
Catecholamine	+	+	++	++	++	–
Cortisol	+	+	++	++	++	–
β-Endorphin	0	+ or 0	++	++	+	0
Estrogens	0	+	+	?	?	– or 0
FSH	0	+	+ or 0	+, 0, –	++	–
Glucagon	0	+	+	++	?	–
Growth hormone	0	+	++	++	++	+
Insulin	0	–	–	–	–	–
Leptin	0	+ or 0	+ or 0	+ or 0	+ or 0	– or 0
LH	0	+	+, 0, –	+ or –	++	–
Progesterone	0	+	+	?	?	– or 0
Prolactin	+ or 0	+	++	+	+	– or +
T <sub>3</sub> , T <sub>4</sub>	0	0	0	+ or –	0	– or 0
Testosterone	0	+	+	+ or –	+	– or 0
TSH	0	+	+ or 0	+ or 0	?	0

“+” – increase; “++” – strong increase; “–” – decrease; 0 – no change; ? – unknow or unresolved.

general effect of exercise training is to attenuate the NES responses: the hormonal concentrations during exercise deviate less from resting homeostatic levels in physically trained *versus* untrained people (37). Thus, basically with more exercise training, an adaptation goes on and exercise almost becomes more of the “normal-like” state and less of a “stress-like” state (19, 35). This adaptation not only results in typically a smaller response in hormonal changes with exercise, but in many examples, resting basal hormonal levels are lower following exercise training. All of this is thought to come about due to improved regulatory capacity, increased receptor number in the target tissue, and improved receptor sensitivity (2, 20). Much further research work, however, is necessary in this aspect of exercise endocrinology dealing with the adaptation to exercise training. Such research work will allow investigators to more clearly elucidate exactly what is occurring not only within many of the endocrine regulatory axes, but also in response to different types of specific exercise training.

There are exceptions to this adaptation phenomenon. People who perform increasing amounts of exercise training may ultimately subject themselves to

excessive quantities of high intensity exercise or greater and greater volumes of exercise in an attempt to push themselves to do athletic-competitive levels of exercise training. When they do this type of “hard, disproportionate” training, the NES can be subjected to tremendous levels of stress and respond accordingly (in some cases greater than what had been observed prior to conducting a training program) (2, 35, 39). While there most certainly is an amazing adaptation and plasticity capacity within the NES to deal with exercise training, every system has its limits and can slip into inappropriate responses if pushed too far (30, 40, 41). There are numerous research examples of NES dysfunction and dysregulation developing in persons involved with demanding or excessive exercise training; such conditions as athletic amenorrhea, the overtraining syndrome, exercise-hypothyroidism, exercise osteopenia, and exercise-male hypogonadism have all been reported (and attributed or linked to NES abnormalities). There are excellent review articles dealing with each of these topics in the research literature, and the reader is directed to these works if they desire more information on the topic (see references 20, 25, 30, 41, 42).

## Conclusion

In conclusion, an attempt was made to present a brief, concise overview of the effects of exercise upon the hormonal components that make up the neuroendocrine system. Specifically, four major topics, in the form of questions, were addressed:

1) Is exercise a stressor to the neuroendocrine system?

– The answer is yes, regardless of the mode of exercise chosen. However, the degree of stress it creates is influenced greatly by the intensity and duration of the exercise, although there are many factors that can modify that level of stress.

2) Why would exercise be a stressor to the neuroendocrine system?

– Exercise needs to be a stressor to the neuroendocrine system, so that the body can make the physiological adaptations to be able to respond to the exercise.

3) What are the effects of exercise as a stressor upon the neuroendocrine system?

– The effects are profound and large, involving the sympathetic nervous system and a variety of hypothalamic-pituitary-peripheral gland axes. It shows a progression of phases in its responses and involves neural, humoral, and hormonal stimuli as regulators and

involves feed-forward and feedback regulatory loops.

4) Is exercise always a stress to the neuroendocrine system?

– The answer is yes, although there is most certainly an adaptation response to that stress, such that the stress becomes lessened with chronic exposure to exercise training. However, even this system has limits in its ability to adapt.

The answers to these questions are by no means exhaustive or to be considered entirely complete and definitive in nature. However, it is hoped the information provided could serve as a beginning point for those individuals interested in the fascinating aspects of exercise physiology that deals with the neuroendocrine system.

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## Fizinis krūvis kaip žmogaus neuroendokrininės sistemos stresą sukeliantis veiksnys

Anthony C. Hackney

*Šiaurės Karolinos universiteto Fizinio lavinimo ir sporto mokslo katedros*

*Taikomosios fiziologijos laboratorijos Endokrinologijos skyrius,*

*Visuomenės sveikatos ir medicinos fakulteto Mitybos katedra, Chapel Hill, Šiaurės Karolina, JAV*

**Raktažodžiai:** hormonai, fizinis aktyvumas, stresas.

**Santrauka.** Tai trumpa apžvalga apie fizinio krūvio sukulto streso poveikį pagrindinėms žmogaus neuroendokrininės sistemos sudedamosioms dalims. Apžvalga suskirstyta į keturias temas, kurios pateiktos bei pagrinėjamos klausimų forma. Šie klausimai yra: 1) ar fizinis krūvis yra stresą sukeliantis veiksnys neuroendokrininei sistemai? 2) kodėl fizinis krūvis galėtų būti stresą sukeliančiu veiksniumi neuroendokrininei sistemai? 3) koks yra fizinio krūvio kaip stresą sukeliančio veiksnio poveikis neuroendokrininei sistemai? 4) ar visada fizinis krūvis yra stresą sukeliantis veiksnys neuroendokrininei sistemai? Straipsnyje šie klausimai pateikiami ir į juos atsakoma siekiant suteikti esminių žinių apie neuroendokrininį atsaką į fizinį krūvį.

Adresas susirašinėti: Professor Dr. A. C. Hackney, Applied Physiology Laboratory, University of North Carolina, CB # 8700 Fetzer Building, Chapel Hill, NC 27599-8700, USA. El. paštas: ach@email.unc.edu

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