



## Hormonal Exercise Response Model (HERM): A Conceptual Framework of Endocrine Reactivity to the Physical Stress of Exercise

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### COMMENTARY

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#### Competing Interests

The authors declare that they have no conflict of interests.

#### Transparency

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study was reported; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained.

#### Ethical

This study follows all ethical practices during writing.

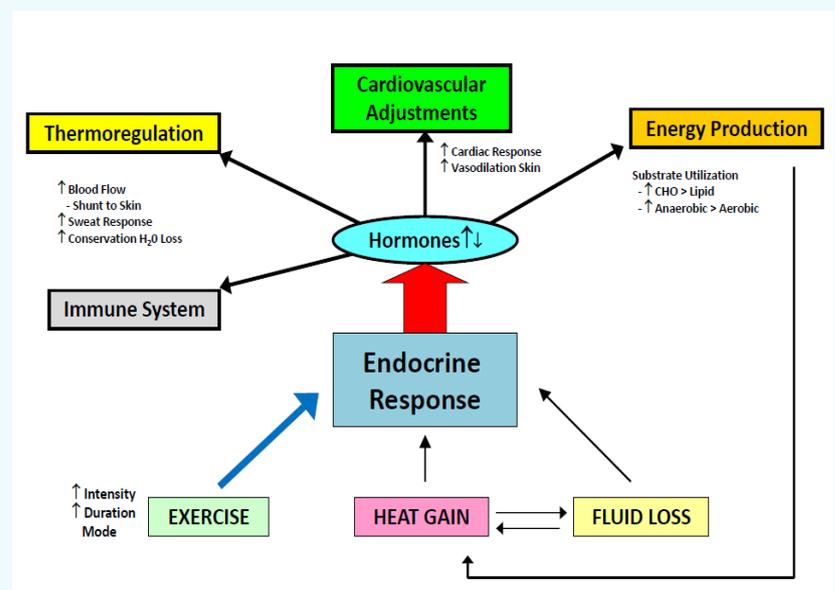
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### INTRODUCTION

Many exercise scientists are interested in the hormonal responses of the endocrine system to the physical stress of exercise. This is a logical pursue due to the critical roles hormones play in bringing about homeostatic adjustments in many bodily systems to allow coping with the demands of an exercise session (Johnson et al., 1992). The hormonal responses bring about or aid in inducing cardiovascular adjustments, energy metabolism, thermoregulations, and immunity changes during exercise (Hackney, 2006). Figure 1 provides a schematic illustration of the physiological consequences of these responses. Because of the wide-ranging effects of hormones, without their appropriate response (i.e., endocrine reactivity) the ability of an athlete-exerciser to perform physical exercise is severely compromised.



**Figure 1.** A schematic illustration of the biological systems invoked to allow for adjustments in the homeostasis of the body during the adjustment to physical exercise.

There is a multitude of hormones changes in a single exercise session, primarily increased circulating levels (N.B., although typically insulin becomes decreased). Many of these hormonal responses are not independent of one another but are highly interrelated and interdependent. To illustrate this point, an explanatory model is presented herein, i.e., the “Hormonal Exercise Response Model” (HERM). This model describes how the hormonal responses to an exercise session can be organized into a series of three interactive phases (Hackney and Lane, 2015).

The first phase of this model deals with the hormonal response immediately at the onset of exercise, with these responses taking just seconds to occur. These responses revolve around the increased sympathetic nervous system activation that occurs with the onset of bodily motion. This increased sympathetic nervous system activity can also be a result of anticipation of the ensuing exercise—which is most certainly the case in sport competition scenarios. This increased sympathetic nervous system activity results in catecholamine (norepinephrine) release at target tissues directly, as well as elevations in circulating catecholamine from so-called sympathetic “spillover” effects.

This effect is further amplified by the sympathetic connection to the adrenal medullary gland which in turn adds to the circulating catecholamine (epinephrine > norepinephrine) response. Simultaneous with these sympathetic-adrenal medullary actions, pancreatic insulin secretion begins to be inhibited, while glucagon secretion becomes stimulated. This entire process involves a feed-forward mechanism of the central nervous system to drive these initial responses, although the events are also modified by peripheral afferent neural input from sensory receptors, in particular, those of skeletal muscle once movement commences.

The intermediate or secondary phase takes slightly longer to develop but is still typically very fast beginning usually in much less than a minute from the onset of exercise. In this stage, the hypothalamus begins the process of releasing hormones such as thyrotropin-releasing factor, corticotrophin-releasing factor (CRF), and growth hormone-releasing factor in an attempt to provoke changes at the anterior pituitary gland to stimulate the release of specific hormones from this endocrine gland. As the pituitary begins to respond to the hypothalamic stimulus there is the release of the various “trophic hormones” from it into the circulation. These hormones in turn begin to affect their specific peripheral target endocrine glands to stimulate additional hormonal release. One of the most rapidly acting elements in this cascade of events is the hypothalamic-pituitary-adrenal cortical interaction where CRF brings about adrenocorticotrophic hormone release and that in turn ultimately brings about cortisol release from the adrenal cortex.

If the exercise session is continued (i.e., extending the duration) there is a transition beyond the intermediate phase into the third phase of response which is a more prolonged state of responsiveness. In this third phase, the responses of the sympathetic-adrenal axis are being augmented by other hormones from the anterior and posterior pituitary (e.g., growth hormone, prolactin, antidiuretic hormone) and the peripheral endocrine glands subordinated to pituitary regulation (such as testosterone, thyroxine, triiodothyronine, insulin-like growth factor-1 [i.e., hepatic release of IGF-1 into blood; muscle release of mechano-growth factor, a local IGF-1 variant]). As fluids shift from the vascular space and total body water stores are compromised due to sweating for heat dissipation and inter-compartmental fluid movement, the renin-angiotensin-aldosterone system (RAAS) is activated (inducing vasoconstrictive actions and water resorption action at the kidney). Additionally, during this phase, the skeletal muscle begins to release select cytokines (e.g., interleukin-6 [IL-6]), hormonal-like agents, into the circulation which affect other hormones to be released (e.g., cortisol) which can have actions to signal energy substrate mobilization and well as immune response.

Phases I and II of the model propose that neural factors are the primary stimuli regulating the hormonal responses to exercise; however, in the third phase of the model, 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 regulatory factors allows an increasing reliance upon feedback rather than a feed-forward control mechanism to determine the magnitude of the hormonal response. The influence of humoral and hormonal stimuli in modulating the hormonal levels is magnified as the exercise duration is extended and energy substrate availability issues cause shifts in energy fuel usage (i.e., decreased carbohydrate increased lipid), or hydration issues (i.e., hemoconcentration and, or dehydration) begin compromising the thermoregulatory ability and leading to greater heat storage within the body affecting hormonal responses (e.g., increased heat storage increased norepinephrine, epinephrine release). Interestingly, the core temperature changes with exercise (and also via ambient environment amplification) result in an exceedingly greater hormonal response for a number of key endocrine agents.

In summary, the proposed HERM model consists of; Phase I – an immediate phase primarily driven by neural mechanism, Phase II – the intermediate phase involves neural and pituitary mechanisms controlling responses, and Phase III – the humoral-based adjustment phase involving principally more slow-acting pituitary mechanisms. The model, while not perfectly inclusive of all hormonal responses, does provide an organized framework for the endocrine responses to exercise activities and helps to illustrate the highly interactive and complexity of these responses. Furthermore, the HERM model gives the context for the sequence of changes and the direction of change, But, it is important for scientists studying exercise hormonal responses to recognize that the intensity and duration of the exercise session are the critical factors in determining the magnitude of the hormonal change-response observed. In conclusion, it is hoped this model provides a conceptual framework to aid exercise scientist in understanding the endocrine reactivity to the physical stress of exercise.

**Keywords:** Exercise, Hormones, Physical Stress

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