Casual Friday Series

Troubleshooting Weight Loss Resolutions, Part III

A Biogenetix Clinical Presentation

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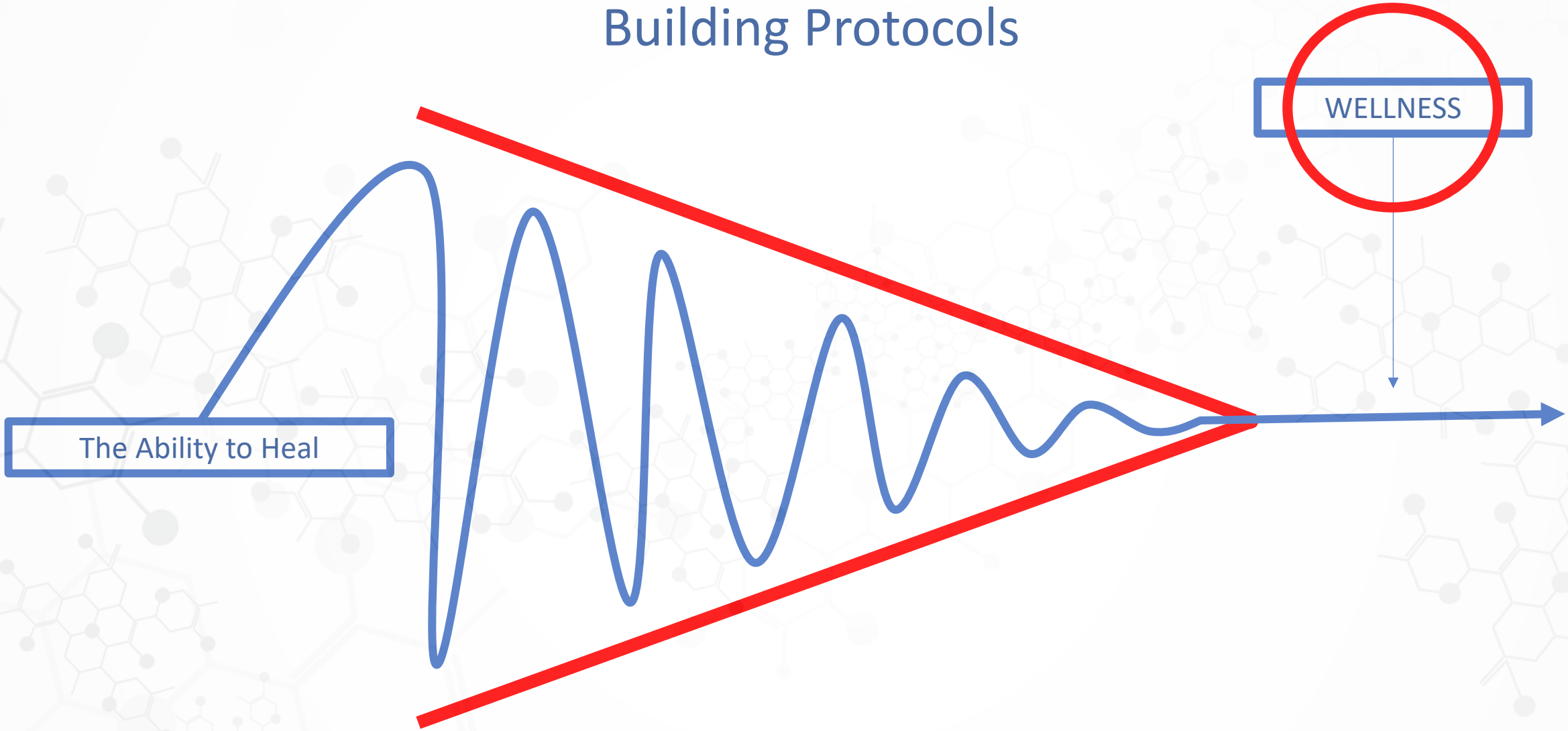
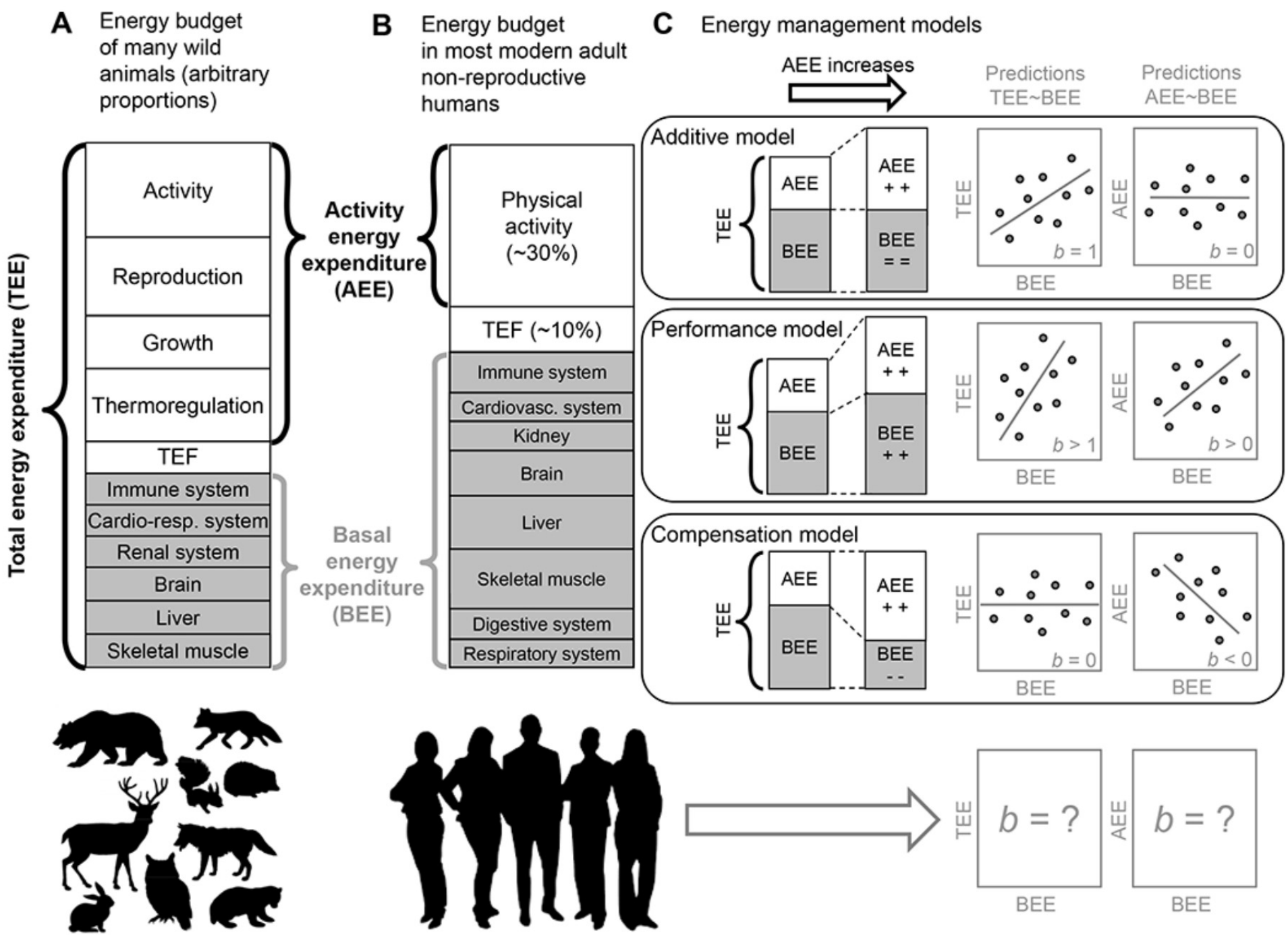
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Lifestyle + Genetics = Chronic Health IMPROVEMENT

Building Protocols



January Decisions…



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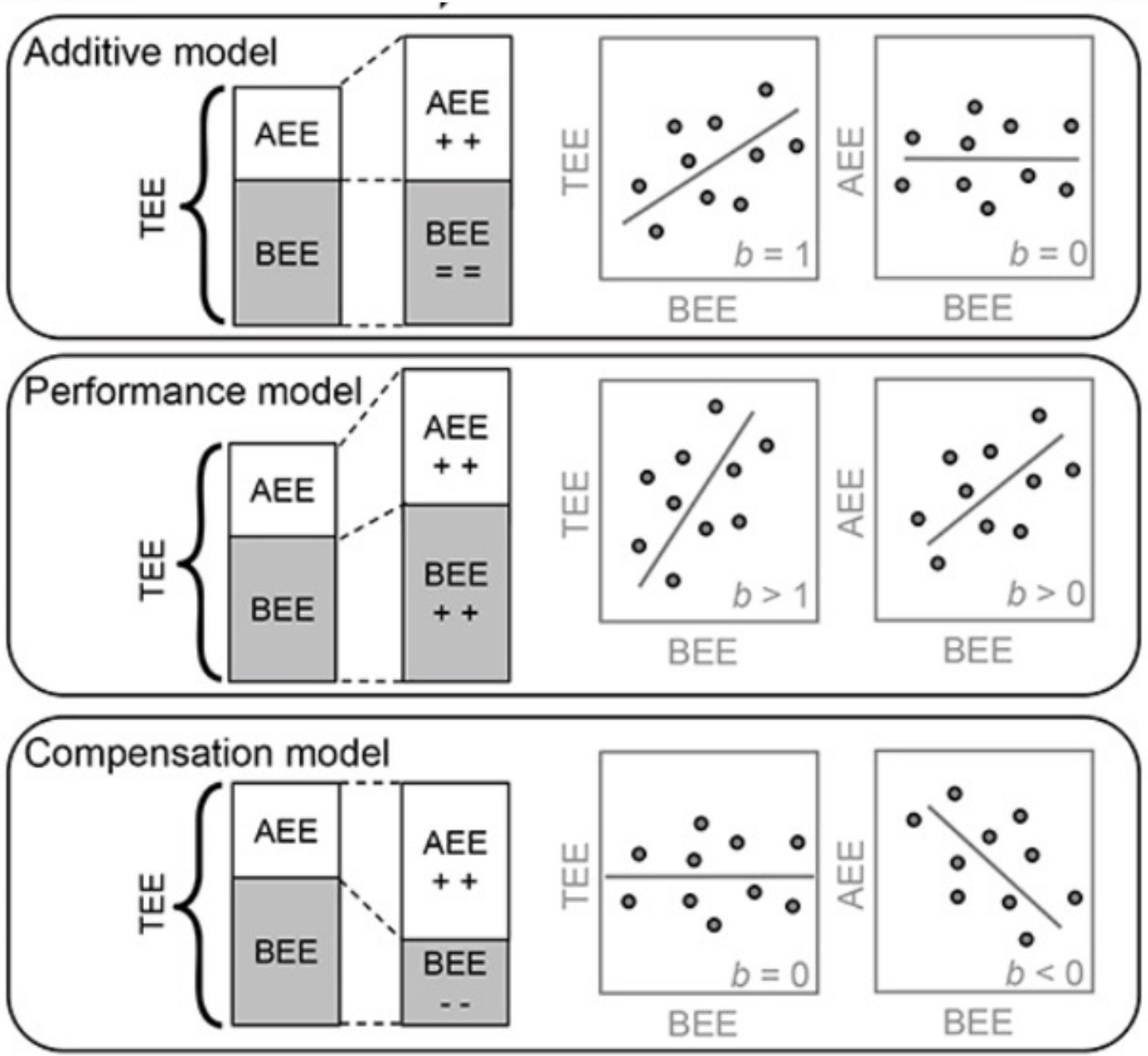

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High-Intensity Intermittent Exercise and Fat Loss

Stephen H. Boutcher*

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Most exercise protocols designed to induce fat loss have focused on regular steady state exercise such as walking and jogging at a moderate intensity. Disappointingly, these kinds of protocols have led to negligible weight loss [1, 2]. Thus, exercise protocols that can be carried out by overweight, inactive individuals that more effectively reduce body fat are required. Accumulating evidence suggests that high-intensity intermittent exercise (HIIE) has the potential to be an economical and effective exercise protocol for reducing fat of overweight individuals.

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What Is the Impact of Energy Expenditure on Energy Intake?

Anja Bosy-Westphal,* Franziska A. Hägele, and Manfred J. Müller

Paolo Piaggi, Academic Editor

maladaptive, leading to weight gain depending on the type of EE. This implies that not only EE per se, but also neuro-endocrine or metabolic effects that signal an increased energy demand under conditions of PA, cold exposure, sleep loss, a very low (or very high) protein diet, or growth and reproduction, are involved in the control of EI. Higher exercise, or PA and heat exposure favor weight loss, whereas an increase in EE due to cold exposure or sleep loss likely contributes to an overcompensation of EI, especially in vulnerable individuals with unfavorable energy partitioning (higher propensity for carbohydrates instead of fat oxidation), as well as under obesogenic conditions, such as an energy dense diet (for summary see Table 1). Irrespective of the type of EE, transient elevations in metabolic rate seem to be general risk factors for weight gain because a decrease in energy requirements is not compensated by an adequate adaptation of appetite and EI. The impact of day-to-day variance and timing (circadian variation) in energy demand due

EI: Energy Intake: Cellular Greediness vs Habit

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Dietary Energy Partition: The Central Role of Glucose

Xavier Remesar[1,2,3] and Marià Alemany[1,2,3,*]

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dietary excesses cease. Too often, very low-energy dieting and exercise combined (even using additional anti-obesity approaches) are unable to eliminate the excess fat and reverse the damages already induced by (or accompanying) this excess deposition. The size of fat depots is a critical point for the severity of the disorders and a barrier to a progressive and effective removal, often leaving only the alternative of surgical modification of nutrient intake [226]. However, none of these procedures can revert the metabolic homeostasis to the situation prior to depot engrossment. Nevertheless, drastic treatments may extend the

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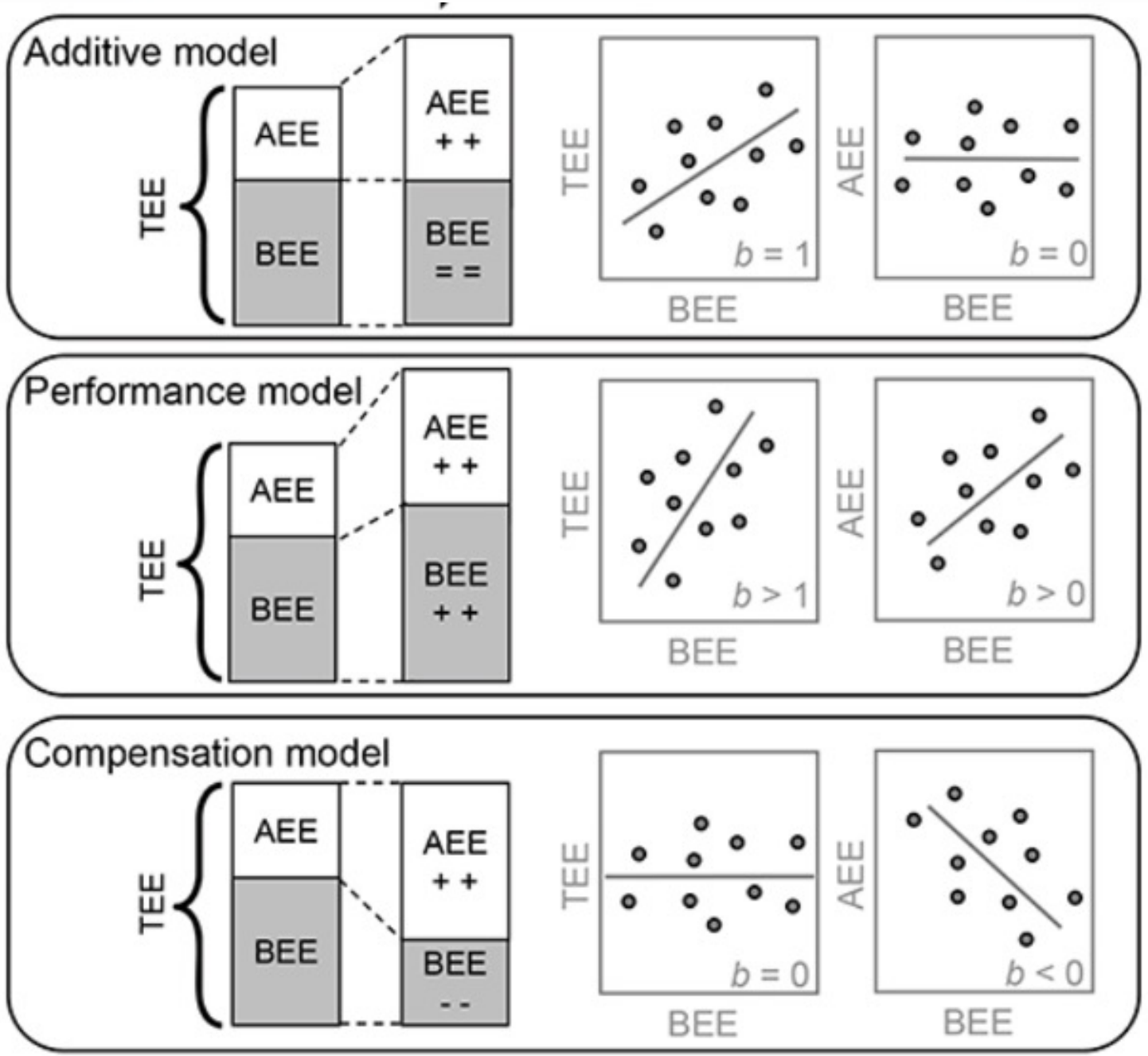
Under conditions of excess energy intake, the metabolic handling of substrates is parallel to modifications in the hormonal mechanisms that regulate energy metabolism [235], since the main system of regulation of glycaemia (insulin) has been severely damaged [236]. The other mechanisms complementary to insulin have a wider array of functions: the glucocorticoids, favor liver glucose output under conditions of stress or metabolic distress [237]. Glucocorticoids also affect the fate of the main gluconeogenic precursors, amino acids, and altering the excretion of N [238,239]. Testosterone pairs with insulin as a main anabolic hormone [240], but glucocorticoids tend to limit testosterone production and availability [241], also affecting the availability of estrogen [242], which plays a critical function favoring the oxidation of 2C (i.e., saving 3C) in females [243] via direct intervention in mitochondrial function [244,245], and also preventing liver steatosis [246]. An estrogen-derivative has been found to down-modulate the adjustment of the ponderostat, i.e., the oxidation/mobilization of lipids from adipose tissue [247,248], by decreasing food intake and maintaining thermogenesis [249]. Unfortunately, there is insufficient mechanistic information on the effect of estrogen derivatives because only recently these hormones are considered important metabolic regulators [250,251] and act not only in the sex-related way indicated by its etymology.



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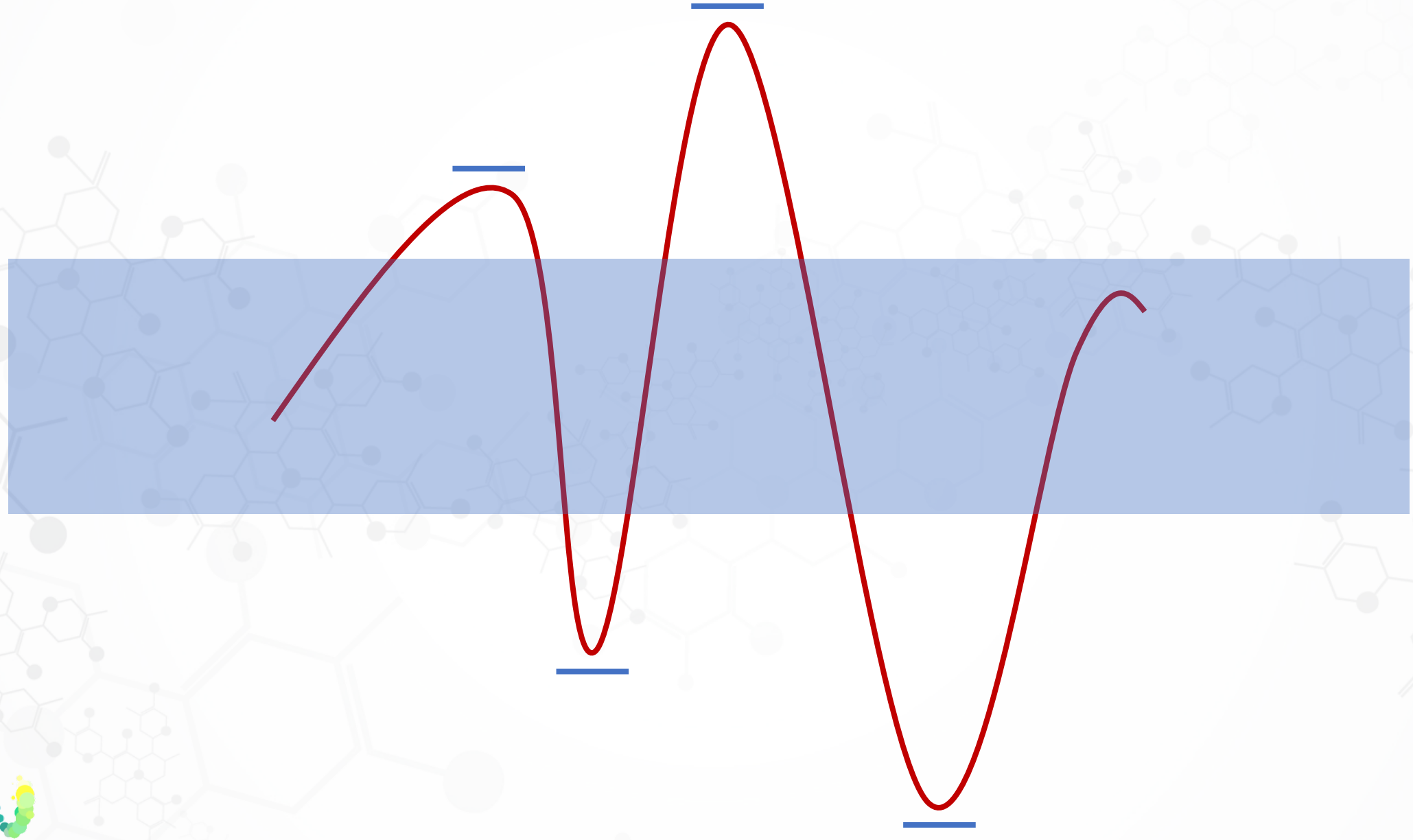

Wellness: Actual Stress vs Perceived Stress

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Does Modern Lifestyle Favor Neuroimmunometabolic Changes? A Path to Obesity

Camila Guazzelli Marques, [1] Marcus V. L. dos Santos Quaresma, [2] Fernanda Patti Nakamoto, [2]
Ana Carolina Oumatu Magalhães, [1,2] Glaice Aparecida Lucin, [2] and Ronaldo Vagner Thomatieli-Santos [1,3,*]

Although less debated and neglected by society in recent years, sleep duration and quality have also been negatively affected (23). Short sleep duration is associated with metabolic, immunological, and behavioral changes that compromise health status and predispose to weight gain (24–27). Systematic reviews and meta-analyses have shown an association between short sleep duration and obesity (25, 28, 29).

However, there is a segmented discussion about the influence of these three factors on body mass gain and obesity. We believe that common mechanisms are shared and overlapped, especially involving systemic inflammation and neuroinflammation, which may explain why isolated interventions against obesity are ineffective.

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Inflammation is characterized by a sophisticated process of pathogens elimination added to tissue repair and recovery, involving immune and other types of cells. According to the degree and extent of the inflammatory process (local or systemic), several metabolic and neuroendocrine changes can occur (48, 49). The inflammatory response is traditionally initiated by infections, resulting from the interaction between pattern-recognizing receptors expressed in cells of the innate immune system and pathogen-associated molecular patterns (PAMPs); likewise, damage-associated molecular patterns (DAMPs) trigger inflammatory response during conditions of physical stress, chemical stress, or through harmful metabolites such as biglycan, fibrinogen, uric acid, mtDNA, among others (50). The shift from a transient to a chronic inflammatory state affects immune tolerance, leading to modifications in the functions of tissues and organs, increasing chronic non-communicable risk for diseases (50). This chronic effect is known as LGCSI. In the absence of PAMPs, DAMPs play a role in triggering the inflammatory process. LGCSI is increased with aging, obesity, chronic infections (i.e., HIV), presence of metabolic diseases such as type 2 diabetes mellitus, non-alcoholic fatty liver, and pollutants (50). Moreover, LGCSI is directly regulated by factors such as diet, sleep, and sedentary lifestyle, in such a manner that this interaction is crucial for the development of several tissue modifications.

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Previous studies verified high levels of inflammatory biomarkers after sleep debt protocols (184). Interestingly, it is suggested that the increased inflammatory state induced by poor sleep also occurs by changes in gut microbiota and adipose tissue (185). The synergy between the immunological system and sleep is complex and it has several mechanisms to explain this bidirectional relationship (26). Sleep debt effects on neuroinflammation have been described previously (186). More recently, Ho et al. (187) verified that sleep fragmentation protocol (18 h of sleep fragmentation per every 24-h period) plus HFD promote microglial activation. Three days of exposure to sleep fragmentation or HFD increased Iba-1-ir (Ionized calcium binding adaptor molecule 1 immunoreactivity) in the arcuate nucleus and the ventromedial hypothalamus. Still, after 9 days, Iba-1-ir remained elevated in the arcuate nucleus in sleep fragmentation plus HFD group, suggesting an interactive effect of both factors.

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Chronic cortisol exposure as a result of sleep debt could increase the mesolimbic reward system, increasing palatable food intake (196). Cortisol also plays a pivotal role in leptin and ghrelin signaling, which may affect EB (197, 198). Also, cortisol reactivity is a crucial factor that modulates eating behavior. For instance, Herhaus et al. (199) observed that obese subjects with high cortisol reactivity demonstrated a significantly higher food intake than subjects with low cortisol reactivity. Interestingly, they did not verify this effect in lean subjects (199). Finally, cortisol increases blood glucose and adiposity, modulating metabolic pathways related to energy expenditure, energy intake, and body composition (197).

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Considering PE in the discussion of neuroinflammation is essential. In response to muscular contractions, myocytes produce and release several molecules, named myokines (200). IL-6 myokine upregulates the expressions of anti-inflammatory cytokine IL-10 and the levels of IL-1 receptor antagonist (IL-1Ra) (45). It has been shown that long-term moderate-intensity PE can increase the production and secretion of IL-10 in the skeletal muscles (201, 202). When IL-10 interacts with its receptor on microglia, it enhances the suppressor of cytokine signaling (SOCS) 3, an inhibitor of cytokine-induced signaling responses, resulting in inhibition of microglial activation, thus acting against the inflammatory state (203).

FIGURE 1 | Interconnections among sleep, diet, sedentarism, inflammation, and obesity. Created with BioRender.com.

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Although less elucidated to the present day, PE can also contribute to neuroinflammation control by the kynurenine (KYN) pathway. The inflammatory state can modify tryptophan metabolism, leading to the formation of KYN. KYN can take two paths, kynurenic acid (KYNA) or quinolinic acid (QUIN). While KYNA has positive effects, such as inflammation counterbalance, QUIN increases the oxidative process and neurotoxicity. The inflammatory environment appears to be an essential mediator for conversion of KYN to QUIN since high TNF-alpha levels increase QUIN production (206). On the other hand, PE promotes the conversion of KYN to KYNA, with neuroprotective-related effects.

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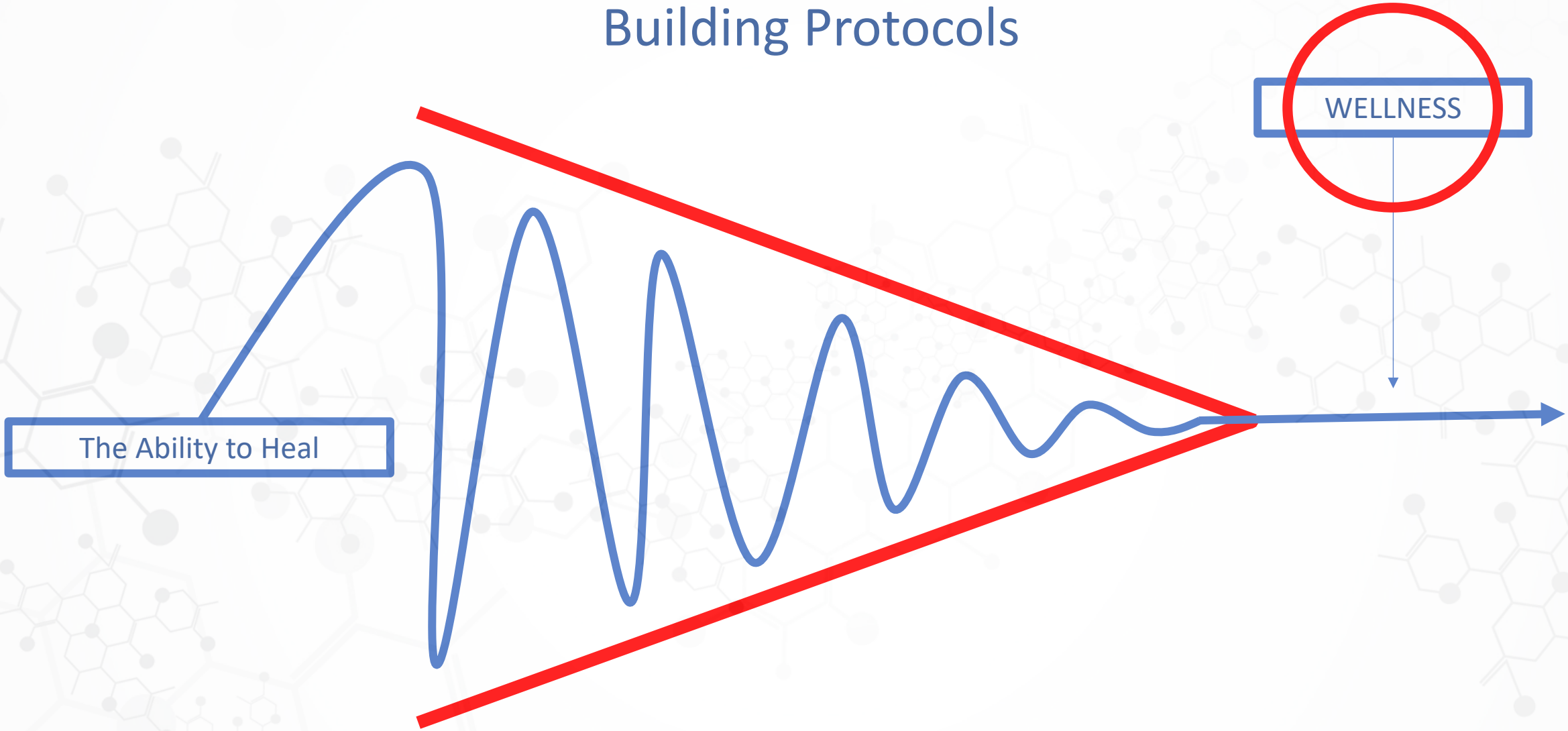
WD, sleep debt, and PE regulate the inflammatory state. WD and sleep debt similarly maximize the inflammatory mediators, energy intake, weight gain, and obesity. On the other hand, PE increases energy expenditure and metabolic efficiency and counterbalances inflammatory mediators, promoting weight gain and obesity resistance. Systemic inflammation and neuroinflammation in obesity are complex responses and share multifactorial features, hindering the establishment of just one mechanism. Future studies should consider that multi-interaction factors contribute to the inflammatory state, making way for further discussions on more strategies capable of regulating the inflammatory process.

Building Protocols



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