Casual Friday Series

Troubleshooting Weight Loss Resolutions, Part II

A Biogenetix Clinical Presentation

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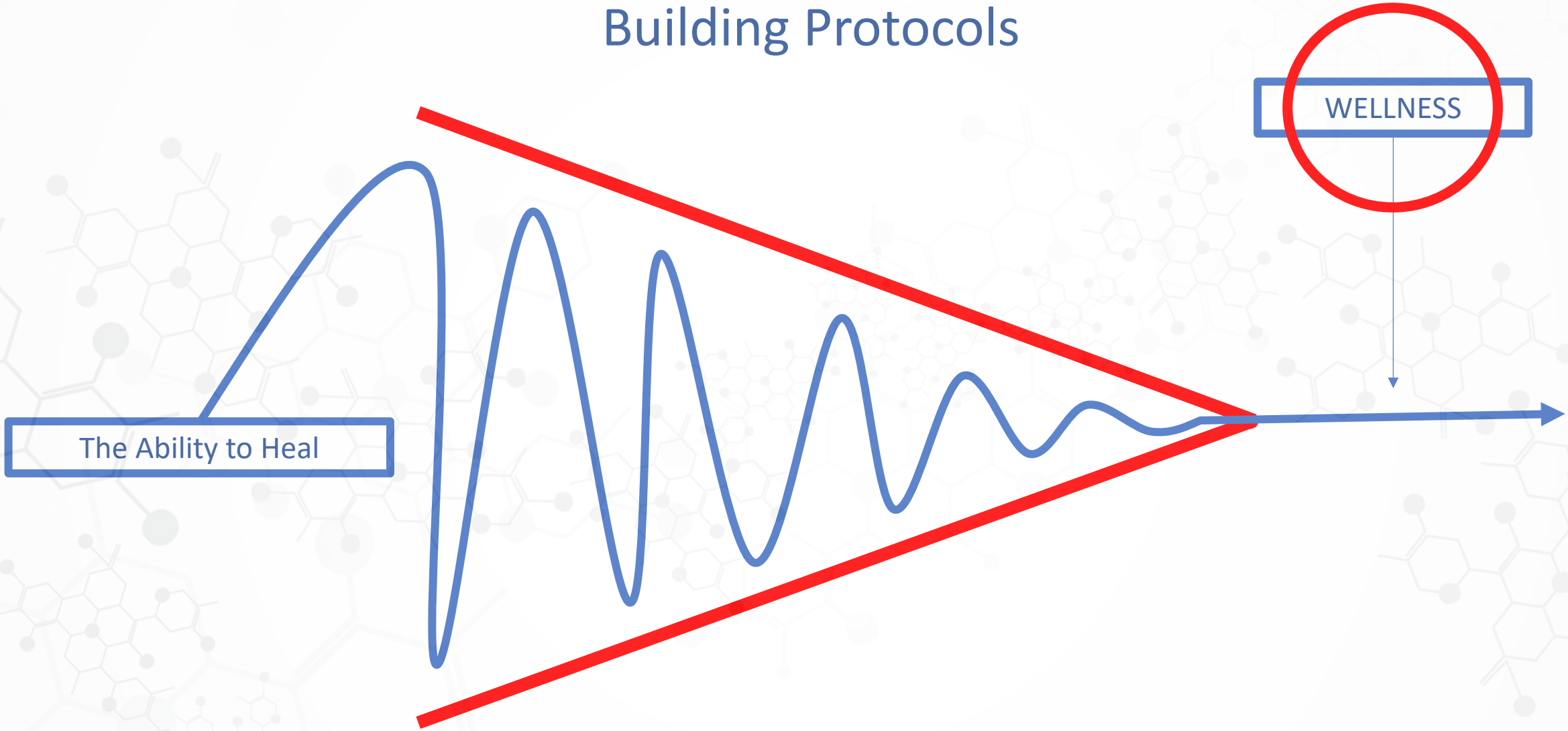
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Lifestyle + Genetics = Chronic Health IMPROVEMENT

Building Protocols



January Decisions…

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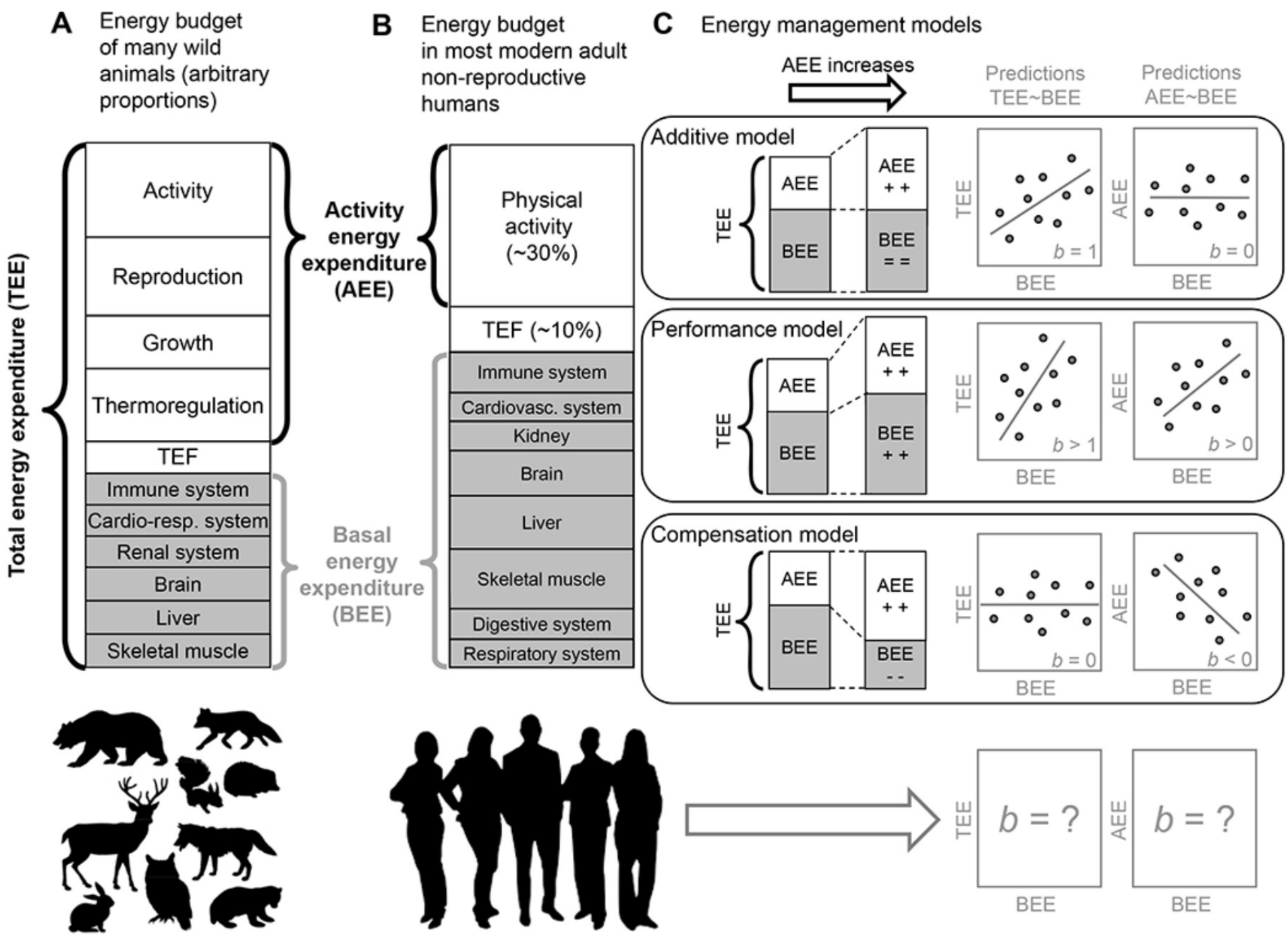
Energy compensation and adiposity in humans

Vincent Careau,[1,~#] Lewis G. Halsey,[2,~*] Herman Pontzer,[3,4,#] Philip N. Ainslie,[5] Lene F. Andersen,[6] Liam J. Anderson,[5,7] Lenore Arab,[8] Issad Baddou,[9] Kweku Bedu-Addo,[10] Ellen E. Blaak,[11] Stephane Blanc,[12,13]

Thus, humans living typical modern lives – not undertaking exceptional levels of activity or experiencing chronic food shortages – exhibit a fairly strong compensation between the energy they expend on activity and that expended on basal metabolic processes; over the long term more than a quarter of the extra calories burned by people during activity do not translate into extra calories expended that day. Presumably, such compensation would have been adaptive for our ancestors because it minimised food energy demands and hence reduced the time needed for foraging, the advantages of which may include reducing exposure to predation. However, it is potentially maladaptive for modern-living humans exercising to try to burn off excess food consumption, given the chain of association linking high-density foods to greater energy intake [14], obesity [15] and its related diseases [16].



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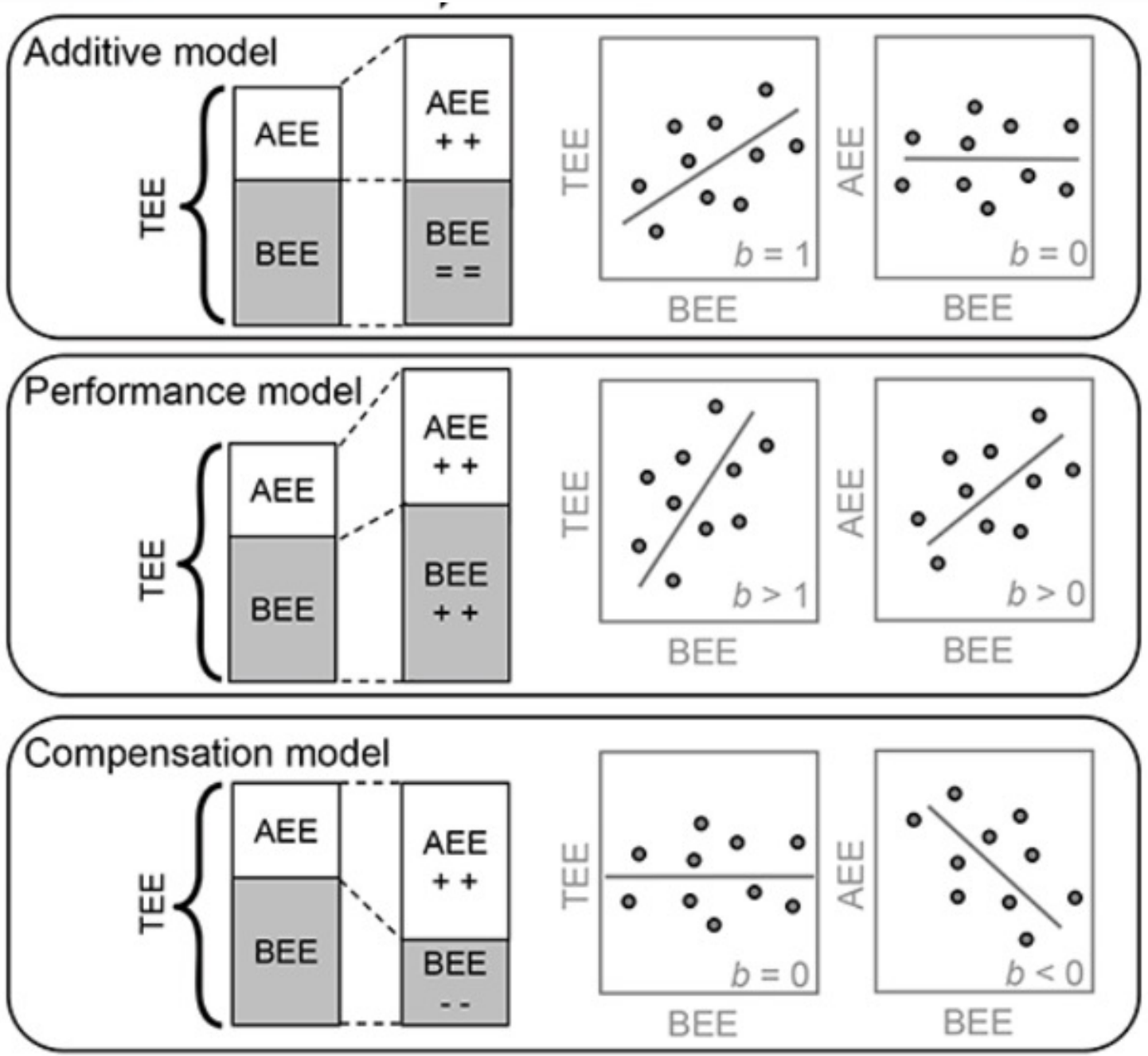

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J Obes. 2011; 2011: 868305.
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PMID: 21113312

High-Intensity Intermittent Exercise and Fat Loss

Stephen H. Boutcher*

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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Chronic responses to HIIE training include increased aerobic and anaerobic fitness, skeletal muscle adaptations, and decreased fasting insulin and insulin resistance (Table 1). Surprisingly, aerobic fitness has been shown to significantly increase following minimal bouts of HIIE training. For example, Whyte et al. [45] carried out a 2-week HIIE intervention with three HIIE sessions per week consisting of 4 to 6 Wingate tests with 4 min of recovery. Previously, untrained males increased their $\dot{V}O_{2\,max}$ by 7%. Increases in $\dot{V}O_{2\,max}$ of 13% for an HIIE program also lasting 2 weeks have been documented [42]. HIIE protocols lasting 6 to 8 weeks have produced increases in $\dot{V}O_{2\,max}$ of 4% [37] and 6–8% [39]. Longer Wingate-type HIIE programs lasting 12 to 24 weeks have recorded large increases in $\dot{V}O_{2\,max}$ of 41% [40] and 46% [6] in type 2 diabetic and older cardiac rehabilitation patients. The less intense protocols (8 s/12 s) coupled with longer duration conducted over 15 and 12 weeks resulted in a 24% [5] and 18% increase [46] in $\dot{V}O_{2\,max}$. Collectively, these results indicate that participation in differing forms of HIIE by healthy young adults and older patients, lasting from 2 to 15 weeks, results in significant increases in $\dot{V}O_{2\,max}$ from between 4% to 46% (Table 1). Mechanisms underlying the aerobic fitness response to HIIE are unclear

Table 1

Effect of high-intensity intermittent exercise on subcutaneous and abdominal fat, body mass, waist circumference, $\dot{V}O_{2\ max}$, and insulin sensitivity.

Study	Subcutaneous fat (kg)	Abdominal/ trunk fat (kg)	Body mass (kg)	Waist circumference (cm)	Type of HIIE	Length of intervention	$\dot{V}O_{2\ max}$ $ml \cdot kg \cdot min^{-1}$	Insulin sensitivity
Boudou et al. [8]	⇓18%	⇓44%	⇓1.9 kg (2%)	—	SSE + 5 × 2/3 min R	8 weeks	—	⇑58%
Burgomaster et al. [37]	—	—	⇔	—	4–6 Wingate/4.5 min R	6 weeks	⇑7%	—
Dunn [46]	⇓2.6 kg (8%)	⇓.12 kg (6%)	⇓1.9 kg (3%)	⇓3.5 cm (4%)	60 × 8 s/12 s R	12 weeks	⇑18%	⇑36%
Helgerud et al. [39]	—	—	⇓.8 kg (1%)	—	15 s/15 s R	8 weeks	⇑6%	—
Helgerud et al. [39]	—	—	⇓1.5 kg (2%)	—	4 × 4 min/4 min R	8 weeks	⇑7%	—
Mourier et al. [40]	⇓18%	⇓48%	⇓1.5 kg (2%)	⇓1.00 cm (1%)	SSE + 5 × 2/3 min R	8 weeks	⇑41%	⇑46%

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2991639/#!po=27.5000

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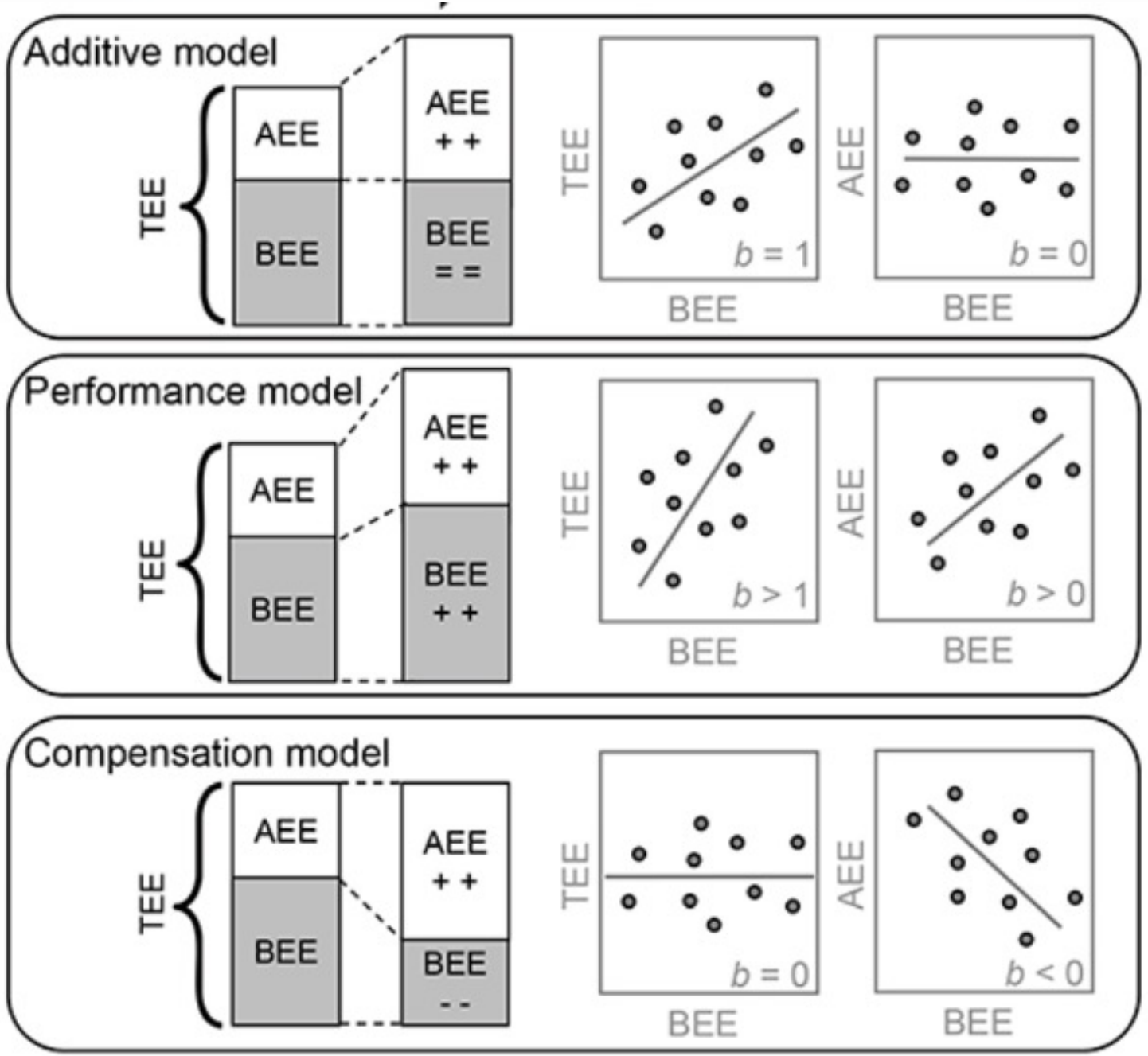
A summary of the results of studies examining the effects of HIIE on subcutaneous and abdominal fat, body mass, and waist circumference is illustrated in Table 1. As can be seen studies that carried out relatively brief HIIE interventions (2 to 6 weeks) only resulted in negligible weight loss. However, the majority of subjects in these short-term Wingate test studies have been young adults with normal BMI and body mass. Studies that used longer duration HIIE protocols with individuals possessing moderate elevations in fat mass [5] have resulted in greater weight/fat reduction. Interestingly, the greatest HIIE-induced fat loss was found in two studies that used overweight type 2 diabetic adults (BMI > 29 kg/m^2) as subjects [8, 40]. Given that greater fat loss to exercise interventions has been found for those individuals possessing larger initial fat mass [54], it is feasible that HIIE will have a greater fat reduction effect on the overweight or obese. Thus, more studies examining the effects of HIIE on obese or overweight individuals are needed.



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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,*]

Return to normalcy from the excess-driven picture of energy metabolism is difficult. The search for drugs is not a viable solution, because the problems observed have a known origin, and the severity of the damages is largely a consequence of the mechanisms established to ensure survival under scarcity. We cannot fight these mechanisms because a) we do not know them enough, b) we need them to sustain our "normal" homeostasis, and c) they have to be operative to achieve that normalcy. Right now, the main systems in use to fight obesity are—essentially, and despite their limited effect—diet and exercise; but, what type of diet? Hypocaloric?

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Under starvation, the stored fat is progressively shed to last as much as feasible; our bodies adapt to lower energy, lower carbohydrate and limited protein intake diets, including the absence of fats, following the blueprints for starvation [212]. The elimination of dietary carbohydrates [213,214] and lowering of energy intake share some characteristics, because of our adaptability to starvation, but there is a considerable discussion on the proposed benefits of ketogenic diets, which, in any case, could not be generalized from epilepsy to obesity [215,216,217]. Nevertheless, even after prolonged starvation or removal of dietary carbohydrates, a significant portion of body fat remains, even after prolonged exposure. Unfortunately, in the practice and for most obese people, dietary treatments remain not effective [218].

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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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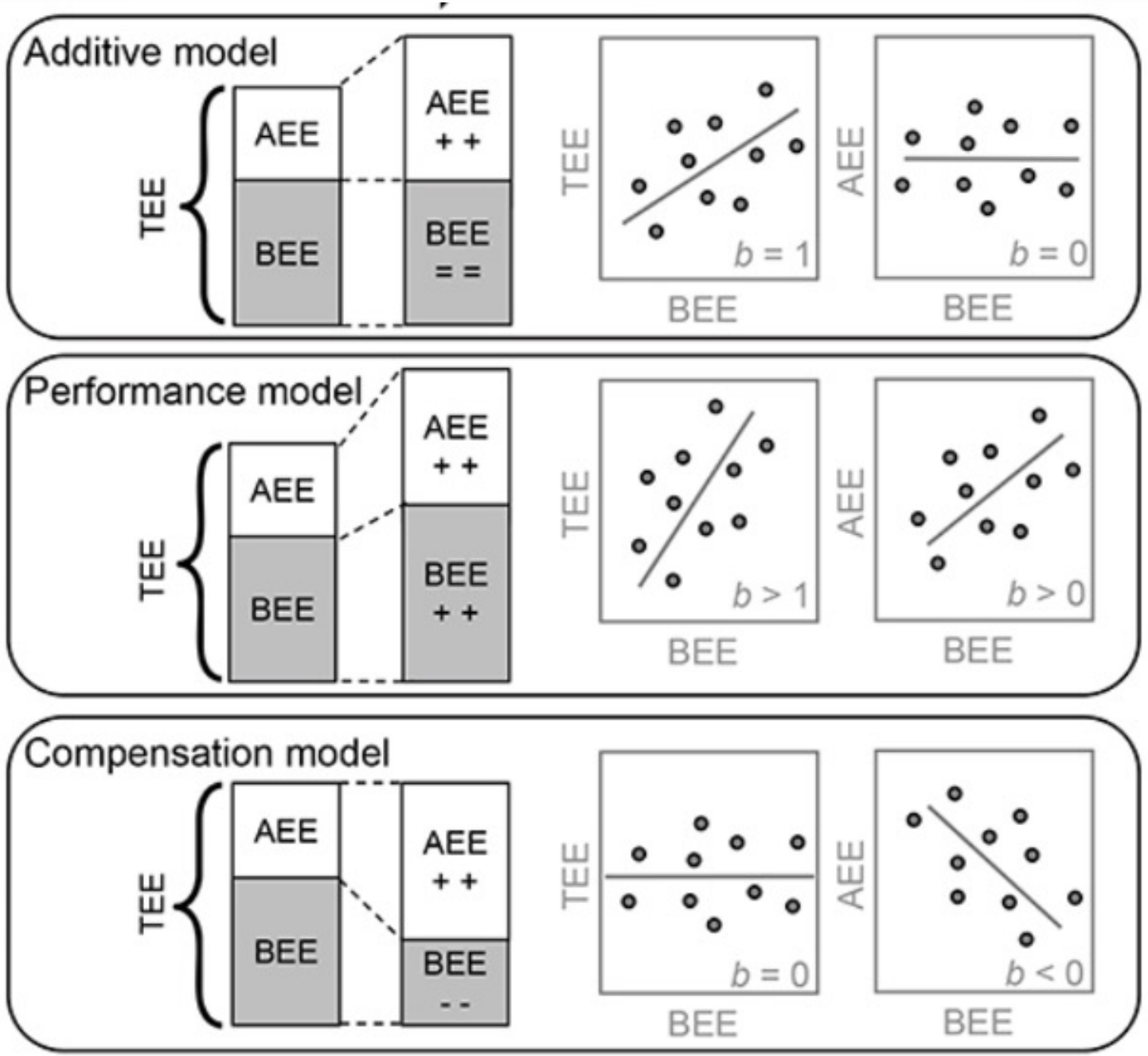
In the liver, glucocorticoids increase glucose output [237] and favor lipogenesis [259] and TAG deposition in most tissues [260]; testosterone induces the accrual of protein [261,262] and stabilizes the maintenance of glycaemia [252]. Estrogens favor 2C oxidation [263], increase oxidative metabolism in mitochondria [245], and limit lipogenesis and TAG deposition [264]. The role of these steroid hormones on the direct modulation of glucose is less clear, despite the large number of agents and effects uncovered. We already know a part of the puzzle, but there are not yet enough dots to draw a sufficiently clear line to understand their real function and help us fight the ravages of our own (effective) systems of protection of energy and protein.



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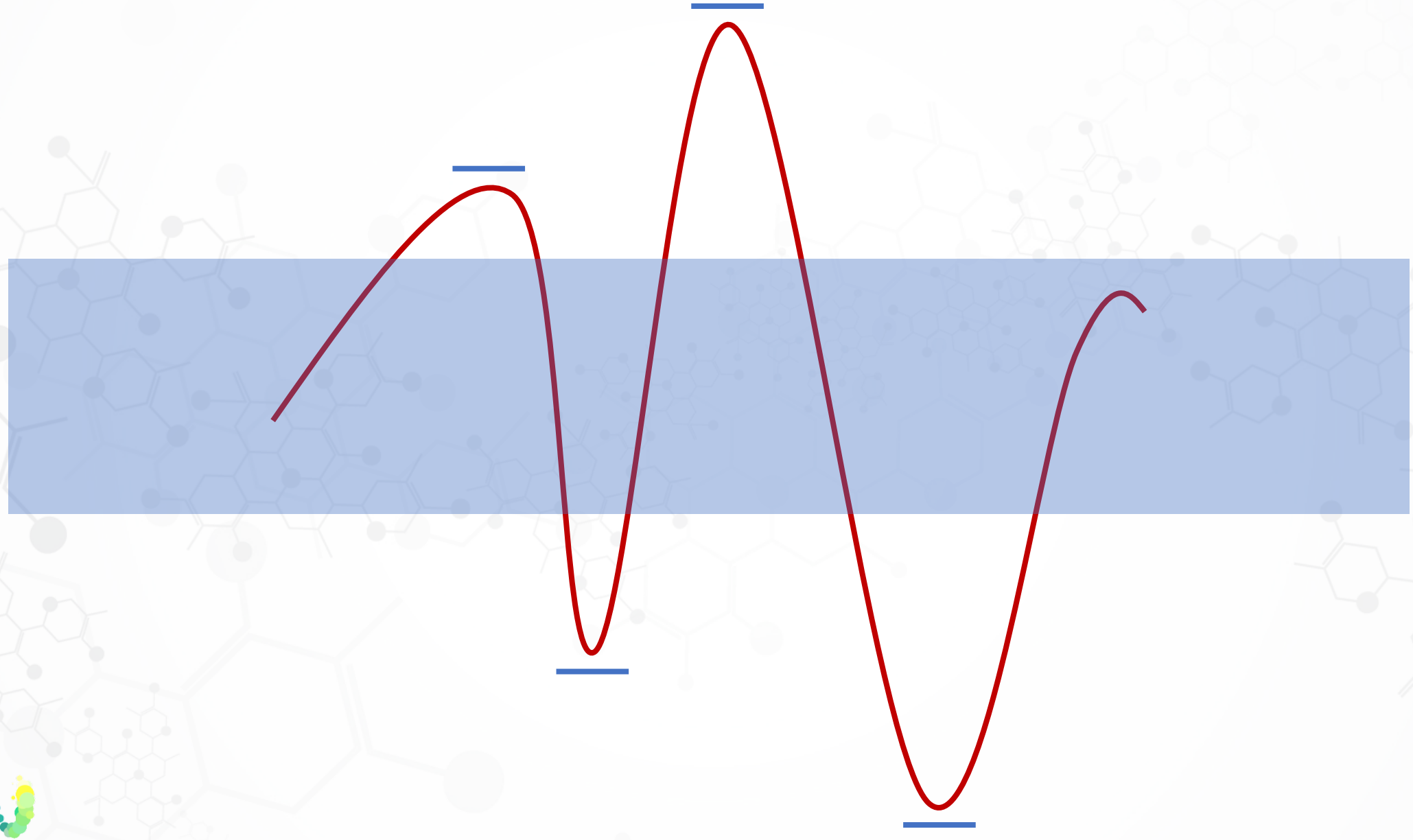
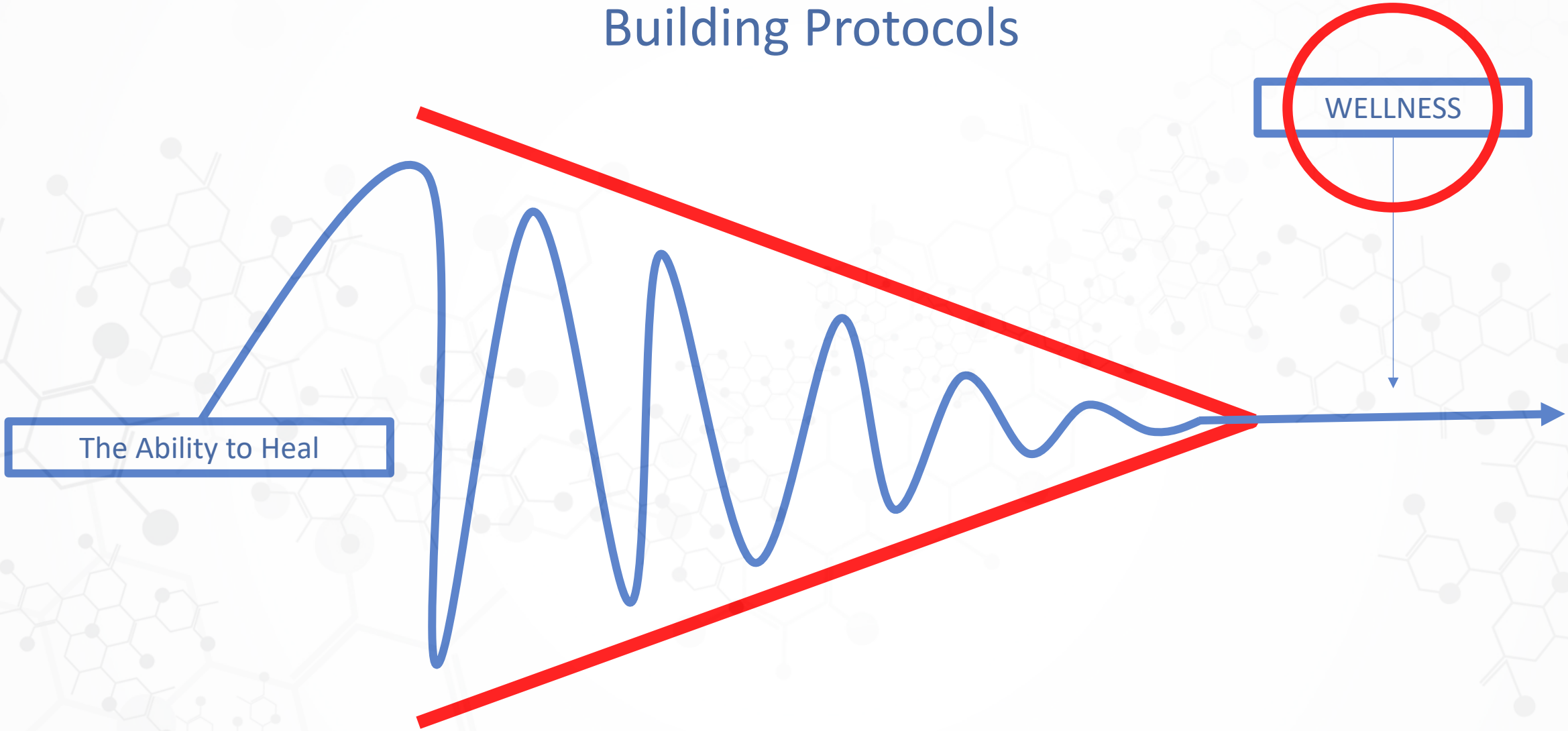


Wellness: Actual Stress vs Perceived Stress

Building Protocols



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