A Computational Framework of Mind-Body Unity: Formalizing Embodied Models of Health

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Health is not merely a passive read-out of biology but an active, ongoing prediction made by the brain. Here, we formalize this intuition within the Embodied Models of Health (EMH) framework, which integrates three well-established yet often disconnected forces—beliefs, expectations and attention—into a unified attention-weighted Bayesian update. In this model, context-evoked mindsets serve as priors over latent health states, while an attention parameter modulates the extent to which incoming sensory information updates those priors. The resulting posterior belief then loops back to physiology through autonomic, endocrine, immune and behavioral pathways, capturing placebo, nocebo and stress phenomena within one tractable equation. We validate the EMH framework through three complementary studies. (i) A controlled laboratory bruise-healing experiment, where participants received identical mild bruises under conditions manipulat-

ing perceived elapsed time (slow, normal, or fast desk timer). A causal-forest analysis shows that the fast-timer cue accelerates objective healing most among individuals who both expected to heal quickly and paid close attention to subtle changes—precisely the joint prior-attention pathway predicted by EMH. (ii) A large-scale "synergistic mindsets" field trial ($N \approx 2,500$ students), where re-estimating the original data with a parsimonious interaction model reveals that a 30-minute lesson reframing intelligence and stress yields the greatest improvements in stress mindset and academic indicators for students who began with the strongest fixed-ability beliefs. This finding illustrates how shifting priors alone can redirect downstream physiology and behavior at scale. (iii) An open-label placebo study, where participants received the same placebo rationale but differed in their pre-existing beliefs about placebo effectiveness. Consistent with EMH, the intervention reduced allergic responses in those with strong placebo beliefs but increased them among participants with especially low beliefs, illustrating how identical inputs can produce divergent physiological outcomes depending on prior expectations and attentional weighting. Because each component of the EMH framework is measurable and manipulable, it offers a quantitative blueprint for precision mind-body interventions that harness beneficial cognitive loops while disrupting harmful ones.

The Embodied Models of Health (EMH) framework, introduced in (1), formalizes how beliefs, expectations, and attention interact to shape health-related outcomes. In our previous psychological review, we outlined how placebo effects and health mindsets emerge from this continuous interplay. Here, we extend these qualitative insights by presenting a mathematical formulation that quantifies how contextually activated mindsets influence the integration of bodily evidence and the updating of health beliefs. We further test the core components of this model empirically across three complementary studies, spanning laboratory, field, and clinical contexts.

Bayesian Model of Beliefs, Expectations, and Attention in Health

Mathematical Formulation of the EMH Model: Mind-Body Economics

Within the Embodied Models of Health (EMH) framework, an individual's general health beliefs constitute a latent knowledge base-broad assumptions about how health works and what signals mean (e.g., "healing takes time," or "pain implies damage").

Beliefs correspond to probabilistic estimates over hidden or latent states of the world (2; 3), including internal bodily states (4). These beliefs emerge from the brain's generative models, which encode probabilistic mappings between hidden causes and sensory consequences. Within this framework, beliefs are continuously updated through the integration of prior knowledge and incoming sensory evidence via Bayesian inference. From these beliefs, the brain derives expectations, understood as predictions about forthcoming sensory outcomes (5). Expectations guide perception and action by minimizing the mismatch between predicted and actual sensory signals (prediction errors) through recursive inferential processes. While both beliefs and expectations are subject to change, beliefs—particularly higher-order priors—may exhibit relative stability depending on their assigned precision and the volatility of the environment. Persistent or significant prediction errors can trigger updates to higher-level beliefs or revisions to the generative model itself, a process central to hierarchical learning and model optimization (2; 3). Thus, rather than operating as static mental representations, beliefs and expectations interact dynamically within a unified inferential architecture that underpins perception, action, and physiological regulation.

When a particular context activates a subset of these beliefs, the person adopts a mindset M. A mindset is a contextually activated belief structure that *guides both attention allocation and expectation formation* (1; 6). It defines what information is considered relevant and what outcomes are anticipated. From this mindset, the individual generates a prior belief about their latent health state H (e.g., how healed they are). Incoming bodily evidence I-such as pain, swelling, or wound appearance-is then interpreted via a likelihood function and integrated with the prior to form a posterior about health. The influence of this evidence is modulated by an attention weight (7; 8) $w(M) \in [0, 1]$ that depends on the mindset itself: some mindsets prioritize internal cues (high w), while others discount them (low w).

Formally, the posterior $P_{\text{posterior}}(h) \equiv P(H = h \mid I, M)$ is given by an attention-weighted Bayesian

update:

$$P(H = h \mid I, M) \propto [P(I \mid H = h)]^{w(M)} \, P_{\text{prior}}(h \mid M) \tag{1}$$

Here:

- $P_{\text{prior}}(h \mid M) \equiv P(H = h \mid M)$ is the prior distribution over health states implied by the currently active mindset M;
- $P(I \mid H = h)$ is the likelihood of observing bodily cues I given latent health state $H = h$;
- $w(M)$ is an *attention weight function*-the precision given to bodily evidence depends on the current mindset M:
  - $w(M) \approx 1$ - mindset prioritizes internal cues (likelihood-dominated);
  - $w(M) \approx 0$ - mindset discounts symptoms (prior-dominated);
  - $0 < w(M) < 1$ - a graded compromise reflecting partial updating.

The resulting posterior $P(H = h \mid I, M)$ represents the updated belief about one's current health state. This posterior can influence health outcomes through multiple channels: behavioral, affective, and physiological pathways (1), including neural, autonomic, endocrine, and immune mechanisms (8; 9; 10; 11; 12). Equation (1) therefore formalizes how contextually activated mindsets shape both expectations and attentional weighting, determining which signals are integrated and the magnitude of their influence on health-related inference.

Glossary of Terms in the Model

Term	**Mathematical Notation**	**Description**
Health State	H	The latent state of health or healing of the individual (e.g. degree of pain, speed of wound healing) $h \in \mathcal{H}$. This is the latent variable the mind attempts to infer.

Term	Mathematical Notation	Description
Mindset (Contextually Activated Beliefs)	M	A subset of general beliefs cued by the current context. The mindset selects which beliefs are operative, thereby shaping the prior distribution over H.
Prior (Contextual Prior)	$P_{\text{prior}}(h \mid M)$	Distribution over possible health states h implied by the active mindset M *before* new bodily evidence is considered. Encodes what outcomes are deemed plausible or likely.
Bodily Evidence (Internal Cues)	I, $P(I \mid h)$	Sensory signals from the body-pain, heart rate, wound sensation, etc. Modeled as a likelihood: the probability of observing I if the true state were h.
Attention Weight	$w(M)$ $(0 \leq w \leq 1)$	Precision-like function indicating how strongly internal cues are weighted relative to the prior. The value of w is determined by the active mindset M. High w = close monitoring of bodily signals (enhancing their impact on beliefs); low w = reliance on prior expectations / external cues.
Posterior (Updated Prior)	$P_{\text{posterior}}(h)$	Updated distribution over H after integrating prior and evidence via Eq. (1). Serves as the *new* prior for the next inference cycle.

Term	Mathematical Notation	Description
Expectation (Point Estimate)	$\hat{h} = \mathbb{E}[H \mid I, M]$	Subjective best guess or anticipated outcome derived from the posterior-e.g. "I am 70% healed." This is the person's new expectation about their health or healing, given what they've felt and focused on. It is the actionable expectation likely to drive behavior and physiology (e.g., triggering relief or stress responses).

How the Model Reflects EMH Interactions

Equation (1) combines the three pillars of the EMH framework- **beliefs, expectations, and attention**-in a single probabilistic mechanism. The mapping from psychological constructs to model terms, and the resulting dynamics, are outlined below.

- **Beliefs as prior and posterior.**
  A contextually activated mindset M supplies the prior distribution $P_{\text{prior}}(h \mid M)$. Strongly positive priors (e.g., the conviction that "this treatment works") place high probability on favorable health states. After bodily evidence I is integrated, the prior becomes the posterior $P_{\text{posterior}}(h)$. Thus an *initial belief-driven expectation* is updated into a *new expectation*, mirroring how mindsets bias perception and can steer physiology. Empirically, people who expect rapid healing often do heal faster; in the model their priors are skewed toward "healed," so even moderate evidence is read as confirmation, creating a self-fulfilling loop.

- *Expectations as dynamic states.*
  The posterior $P_{\text{posterior}}(h)$ is the updated belief after bodily signals I are filtered through the mindset. Its point estimate $\hat{h} = \mathbb{E}_{\text{posterior}}[H]$ is the individual's actionable expectation-e.g., "I am about 70 % healed." Even modest objective change can shift $\hat{h}$ if the mindset directs

attention to positive cues, and that new expectation in turn guides emotion, behavior, and physiology.

- **Attention as selective gate & precision modulator.**
  The weight $w(M)$ acts as a precision parameter on the likelihood $P(I \mid h)$ and therefore determines how strongly I can move expectations:

  High $w(M)$**:** bodily signals are *amplified*. This corresponds to interoceptive focus or symptom hyper-vigilance; small deviations from the prior pull the posterior toward the evidence.

  Low $w(M)$**:** sensations are *down-weighted*. Strong priors or external cues dominate, so even large internal signals may be ignored or re-interpreted. This "turning down the volume" explains why a reassuring context can sustain pain relief despite ongoing nociception.

  Hence $w(M)$ tunes the precision of prediction errors, in line with predictive-processing accounts (8).

- **Balancing internal and external information.**
  $P_{\text{prior}}(h \mid M)$ carries contextual or external knowledge-clinician reassurance, diagnostic labels, cultural scripts-whereas $P(I \mid h)$ embodies internal bodily data. Attention $w(M)$ tilts the balance between these two streams, so health outcomes emerge from an on-line negotiation between what the mind expects and what the body signals.

- **Physiological pathways.**
  The posterior expectation is not merely a cognitive inference; within the EMH framework, it participates in an active inference process (13; 9). Organisms do not passively perceive bodily states but act to minimize discrepancies between expected and actual internal conditions, aligning physiology with prior beliefs. In this context, the posterior — the updated belief about one's latent health state — feeds back to the body via autonomic, endocrine, immune, and behavioral pathways. Positive posteriors (e.g., "I am healing") are hypothesized to up-regulate parasympathetic tone, lower cortisol, and promote tissue repair and growth factors, thereby reducing prediction errors between expected and perceived bodily states.

Conversely, negative posteriors can elicit stress physiology and inflammatory cascades. Empirical research illustrates these dynamics: expecting lower fatigue decreases fatigue (14; 15); expecting to catch a cold predicts higher infection rates (16); reappraising physiological stress responses alters cardiovascular and endocrine reactivity (17; 18); and in a field study, hotel housekeepers informed that their work "counts as exercise" subsequently exhibited improved cardiometabolic profiles compared to those who were not informed (19). Thus Eq. (1) supplies the cognitive trigger that-via the affective and behavioral routes detailed in the *Extensions* section-translates belief updates into measurable biological change.

Extensions: Priming, Affective/Behavioral Pathways, and Bidirectional Dynamics

Conceptual priming and diagnostic labels. Subtle contextual cues-diagnostic labels, clinical rituals, cultural scripts transiently *activate* a particular *mindset* M (17; 18; 20; 21). This mindset modulates the prior distribution over latent health states, $P_{\text{prior}}(h \mid M)$. Thus conceptual priming is modeled as a *shift of mindset*, not a change in the belief database itself. For example, being told one is "*prediabetic*" can prime a *risk mindset* that places higher prior probability on ill health (22). Empirical studies show that individuals labeled as prediabetic subsequently exhibit higher rates than matched controls; in the model the label moves mass in P_{prior} toward poorer health expectations, which-after Bayesian updating-raises posterior beliefs in disease risk and can influence both behavior and physiological regulation.

Affective and behavioral pathways. Equation (1) formalizes the *cognitive* component of health-related inference — the construction of updated expectations about one's latent health state based on bodily evidence and mindset. However, within the Embodied Models of Health (EMH) framework, these posterior expectations are not confined to abstract beliefs; they actively participate in regulating physiology, emotion, and behavior. This process aligns with principles of *active inference* (9; 13), whereby organisms strive to minimize prediction errors not only by updating beliefs but by adjusting bodily states and actions to fulfill prior expectations. EMH holds that posterior expectations act through emotional and behavioral channels. We can represent this with two auxiliary variables:

- *Affective pathway:* Let A_t denote a mindset-induced emotional state (e.g., stress vs. calm). Posterior expectations modulate A_t, and A_t feeds back into the latent health state H_t:

$$\frac{dH_t}{dt} = f\big(H_t,\ A_t\big), \qquad A_t = g\big(P_{\text{posterior},t}\big).$$

  Positive expectations are associated with reductions in cortisol and inflammation, speeding recovery; negative expectations can engage pathways that raise sympathetic tone and slow healing (23; 24; 25; 26; 27).

- *Behavioral pathway:* Let B_t be health-relevant behavior (exercise, adherence, help-seeking) chosen in light of the actionable expectation $\hat{h}_t$:

$$B_t = h\big(\hat{h}_t\big), \qquad H_{t+1} = H_t + \beta\, B_t.$$

  Optimistic expectations ($\hat{h}_t$ high) promote constructive actions, while pessimistic expectations suppress them (23; 26; 27).

Bidirectional dynamics and iterative updating. The EMH mechanism can be naturally extended to recursive time-dependent inference. Let I_t denote bodily evidence at time t, and M_t the mindset at time t. Then the posterior at time t becomes the prior at $t + 1$:

$$P^{(t+1)}(H = h \mid I_{1:t+1}, M_{1:t+1}) \propto [P(I_{t+1} \mid H = h)]^{w(M_{t+1})}\, P^{(t)}(H = h \mid I_{1:t}, M_{1:t}) \tag{2}$$

The EMH mechanism is recursive: the posterior formed at time t immediately becomes the prior at t+1, while affect A_t and behavior B_t feed back into the latent health state H_t. This loop can spiral upward (placebo) or downward (nocebo).

1. **Prior.** At time t the agent holds a mindset-tuned prior $P^{(t)}_{\text{prior}}(h \mid M_t)$.

2. **Evidence.** Bodily cues I_t are observed.

3. **Update.** Attention-weighted Bayes gives

$$P^{(t)}_{\text{post}}(h) \ \propto\ \big[P(I_t \mid h)\big]^{w(M_t)} P^{(t)}_{\text{prior}}(h \mid M_t),$$

with actionable expectation $\hat{h}_t = \mathbb{E}^{(t)}_{\text{post}}[H]$.

4. **Feedback.** The expectation $\hat{h}_t$ shapes emotion A_t and behavior B_t, which jointly influence physiology-e.g. $\frac{dH_t}{dt} = f(H_t,\, A_t,\, B_t)$.

5. **Propagation.** Set $P^{(t+1)}_{\text{prior}} = P^{(t)}_{\text{post}}$. The attentional policy may also adapt: $w_{t+1} = g\big(P^{(t)}_{\text{post}}, \text{beliefs}\big)$; this bidirectional dynamic can lead to *cognitive traps* (28), systematic mistakes in how people perceive, interpret, or attend to information that lead to persistent suboptimal behaviors - a misallocation of attention and mental models that can lock individuals into maladaptive patterns.

If $\hat{h}_t$ is optimistic, lower stress and constructive actions improve H_{t+1}, so new evidence reinforces the positive prior-a *virtuous placebo cycle*. Conversely, a pessimistic $\hat{h}_t$ heightens vigilance and stress, worsening H_{t+1} and confirming the negative prior-a *vicious nocebo cycle*. Interventions can break harmful loops by re-priming the mindset (shifting the prior) or by retraining the attentional policy $w(M)$ (e.g., mindfulness or cognitive reframing) to steer the trajectory toward a healthier equilibrium.

Linking Internal and External Attention to the Three Pathways

Mindset-driven inference in the EMH framework operates on *two attentional channels*. When $w(M) \to 1$, the update in Eq. (1) privileges internal (interoceptive) evidence-body sensations such as pain, heart rate, or wound appearance. When $w(M) \to 0$, the mind relies more heavily on external cues: contextual information, social signals, and culturally shared scripts that shape $P_{\text{prior}}(h \mid M)$. Whichever channel dominates, the resulting posterior expectation $\hat{h}$ propagates through three downstream pathways:

1. **Affective pathway** (A_t): optimistic posteriors reduce threat appraisal and cortisol, whereas pessimistic posteriors heighten stress responses.

2. **Behavioral pathway** (B_t): expectations guide adherence, physical activity, and help-seeking-behaviors that feed back into the latent health state H_t.

3. **Direct physiological pathway**: beliefs can modulate autonomic, endocrine, and immune function *even without* changes in affect or behavior.

Social context factors-clinician warmth, treatment rituals, diagnostic labels-enter the model in two places: (i) by priming a mindset M and thus shifting the prior; (ii) by steering attention toward or away from bodily signals (e.g., a calming ritual lowers w, a symptom-monitoring app raises w). Together, these mechanisms explain how the same pharmacological treatment can yield divergent outcomes across settings (29): context alters both what is *expected* and what is *noticed*, thereby reshaping the full affective-behavioral-physiological cascade.

Neural appraisal mechanisms Recent meta-analytic work on placebo effects (30) shows that context cues engage a default–mode appraisal network (vmPFC (ventromedial prefrontal cortex)–PCC (posterior cingulate cortex)–TPJ (temporoparietal junction) $\leftrightarrow$ ventral striatum), which (i) constructs value-laden expectations about future well-being, (ii) modulates learning in sensory and autonomic pathways (periaqueductal gray, rostral ventromedulla, spinal cord), and (iii) drives endocrine, immune, and dopaminergic responses. This neurobiological circuit provides a concrete substrate for two core EMH operations: generating the prior $P_{\text{prior}}(h \mid M)$ and gating symptom precision via the attention weight $w(M)$. For example, vmPFC$\rightarrow$PAG coupling predicts the magnitude of placebo analgesia, while placebo-induced μ-opioid release in ventral striatum tracks improved mood and motor function in depression and Parkinson's disease. Further studies highlight the key role of the PFC in representing the prior $P_{\text{prior}}(h \mid M)$, which exerts a descending influence on physiological pathways (31), hence also supporting the EMH. Thus, the appraisal account supplies mechanistic evidence that mindsets alter health by re-valuing bodily evidence and by recruiting descending control systems-exactly the pathways formalised in Eq. (1).

Empirical Application of the Model

Each element of the proposed model corresponds to something measurable or manipulable in research and practice, making the model empirically testable:

- **Expectations (Prior and Posterior):** These can be measured via questionnaires (e.g. asking patients how much relief they expect, or their general mindset about healing), via behavioral choices or performance, or inferred from physiological signals such as autonomic responses or brain activity.. Experimentally, they can be manipulated through suggestions, informa-

tion, conditioned associations, immersive or contextual manipulations, feedback, or social influences (for example, giving different expectations to different groups about a therapy's effectiveness). In a wound-healing study (32), participants' *perception of time* was manipulated to set different expectations (33) , which led to measurable differences in healing speed. Such paradigms help quantify how changes in prior expectations affect outcomes.

- **Bodily Evidence (Internal Signals):** This corresponds to physiological and subjective data - e.g. heart rate, levels of pain, biomarkers of stress or healing (like cortisol, inflammatory markers), etc. These are routinely measurable. In Bayesian terms, one can estimate how consistent the sensations are with various health states. For instance, is the patient's pain level low (consistent with "getting better") or high (consistent with "still in pain")? Modern studies in placebo analgesia use fine-grained measures of pain reports and brain activity to model such likelihood functions (e.g. (34)). The information conveyed by these internal signals can also be experimentally manipulated using biased biofeedback paradigms. For instance, a recent study demonstrated that modifying heart rate feedback altered perceived effort (35).

- **Attention Weight:** Attention allocation can be tracked via self-report (asking what the person is focusing on), behavioral tests (like dot-probe or attention tasks to see if health-related stimuli are prioritized), or physiological and neural proxies (e.g., pupil dilation, EEG signals, or fMRI activity) (36; 37; 38; 39; 40; 41)). It can also be experimentally manipulated: instruct some participants to focus on their sensations intently, others to distract themselves or focus on external cues. By varying w, one can test the model's prediction that the same prior and evidence yield different outcomes. For example, does a strong positive expectation yield more pain relief when attention is directed away from symptoms (low w) than when attention is absorbed by symptoms? Studies show that both positive expectations (placebo analgesia) and distraction independently reduce pain, and their effects are largely additive (42; 43). (42) found that expectancy-driven pain relief persisted even when participants were distracted by a demanding task, suggesting that belief and attention work through separate mechanisms. Neuroimaging confirms this: expectation recruits prefrontal regions to engage descending inhibitory pathways, while distraction dampens pain signals via attention and executive

networks, even at the spinal level (43). Clinical findings similarly suggest that patients who hold strong positive expectations and can shift attention away from symptoms report greater relief, highlighting the complementary roles of belief and attentional focus in pain modulation (44; 45; 46). Experiments like these directly validate the attention-modulation effect predicted by the model.

- **Outcomes (Physiological or Behavioral Changes):** Finally, the actual health outcome x (or its proxy) is measurable: wound size, recovery time, symptom severity, blood pressure, fatigue, etc., as well as health-related behaviors (physical activity, medication adherence). The model suggests these outcomes will differ as a function of the interplay between mindset, attention, and evidence. Indeed, numerous studies support this: e.g., placebo studies show that when expectations are high and attention is managed, patients experience real improvements in symptoms; conversely, in nocebo scenarios, negative expectations and symptom-focusing can produce real side effects or worsening of health (47; 48; 49; 50).

By quantitatively fitting this model to data - for example, using Bayesian computational modeling to estimate each participant's w and prior strength from their responses - researchers can assess how well the framework explains individual differences in mind-body responses. The model's parameters each map to an intervention lever: *beliefs* can be shifted via reframing or information (mindset interventions), *attention* can be trained or directed (mindfulness, distraction techniques, gamification), and *contextual priming* can be adjusted (creating more healing-conducive environments). In practice, the EMH model suggests that to improve health outcomes, one can either change the prior (instill positive expectations and empowering health beliefs) or change attention (teach individuals to attend in ways that support those positive beliefs, for instance by mindfulness that neither exaggerates nor ignores bodily signals, but interprets them calmly).

In summary, this simple mathematical model encodes a conceptually rich idea: health is a continuously negotiated prediction, forged between our mind's expectations and our body's sensations, with attention as the tuning knob. It formalizes how mindsets, priors, and present-moment evidence combine to shape our lived health outcomes, and how adjusting our focus of attention can tip the scales towards one or the other. By defining each component in measurable terms, the model also offers a blueprint for research - a way to predict when changing a belief or an attentional focus

will translate into real physiological change. Such a framework moves us closer to an integrated, empirically grounded understanding of mind-body unity, where interventions can be designed to harness the beneficial loops of belief, expectation, and attention for better health. In the following section, we test this framework on empirical datasets, assessing whether expectations, attention, and context interact to predict measurable changes in physiology and health.

Expectation and attention jointly shape healing in the perceived-time bruise study. Aungle and Langer's (32) cupping-bruise experiment offers a concise test of the EMH mechanism. Twenty-five participants first completed a baseline survey-at enrollment, *before any lab visit*-rating how fast they heal relative to other people. We treat this session-independent score as each person's *expectation prior* $P_{\text{prior}}(h \mid M)$.

Each participant attended three laboratory sessions in which a standardized mild bruise was induced, and healing was monitored over a fixed 28-minute period. During each session, a visible desk timer was manipulated to display either 14 minutes (*Slow Time*), 28 minutes (*Normal Time*), or 56 minutes (*Fast Time*), thereby altering participants' perception of how much time had passed, while the actual elapsed time remained constant. Every four minutes they completed a brief *healing-observation* survey about color, size, irritation, visibility, and severity. Digital photographs provided an objective index of bruise lightening.[1]

Two participant-level covariates capture the EMH channels:

1. **Expectation prior.** The baseline healing-expectation score recorded *prior to all sessions*.

2. **Attention weight proxy.** The within-session *variance of healing-survey ratings*-how much a respondent noticed the bruise changing from check to check-serves as a behavioral index of the attention weight $w(M)$ [2].

[1] Henri Bergson's concept of *dur'ee*-time as qualitatively experienced rather than mechanically measured-offers a philosophical analogue to the timer manipulation here. By altering the externally signaled "amount of time elapsed," the *fast* and *slow* clocks reshape participants' priors about how much healing *should* have occurred, while the attention weight $w(M)$ governs how strongly concurrent bodily evidence updates that belief. See Bergson (1889/1910) *Time and Free Will*.

[2] *Attention to variance in healing* gauges how often participants *noticed any change* in their cupping marks from one check-in to the next. Each session contained six survey moments ("waves"); from wave 2 onward, participants indicated for *each of five dimensions*-intensity, color, irritation, visibility, severity-whether that feature had become *better, worse, or stayed the same* since the previous wave. This yields $5 \times 5 = 25$ binary change/no-change judgments per session. We code a "1" whenever a dimension is reported as either better or worse (i.e. *any* change) and a "0" when

Figure 1 shows that participants with stronger baseline priors tend to monitor the bruise more closely ($r \approx 0.35$). Figures 2 and 3 display the *Expectation* $\times$ *Timer* and *Attention* $\times$ *Timer* interactions: when the timer ran *fast*, both a stronger prior and greater attentional weighting to healing variance predicted markedly greater objective healing, whereas slopes flattened or reversed under the *slow* timer. Thus the external fast "elapsed-time" cue produced the largest physiological benefit precisely when it aligned with a positive prior *and* received high attentional weighting-exactly as Eq. (1) anticipates.

Together, these visual results indicate (a) priors and attention covary, reflecting a shared mindset, and (b) both factors interact with contextual cues (e.g., perceived time) to modulate health-relevant outcomes, rather than exerting simple main effects. These results provide intuitive and empirical support for the EMH framework, in which priors and attention jointly shape the interpretation of bodily signals and consequent physiological change.

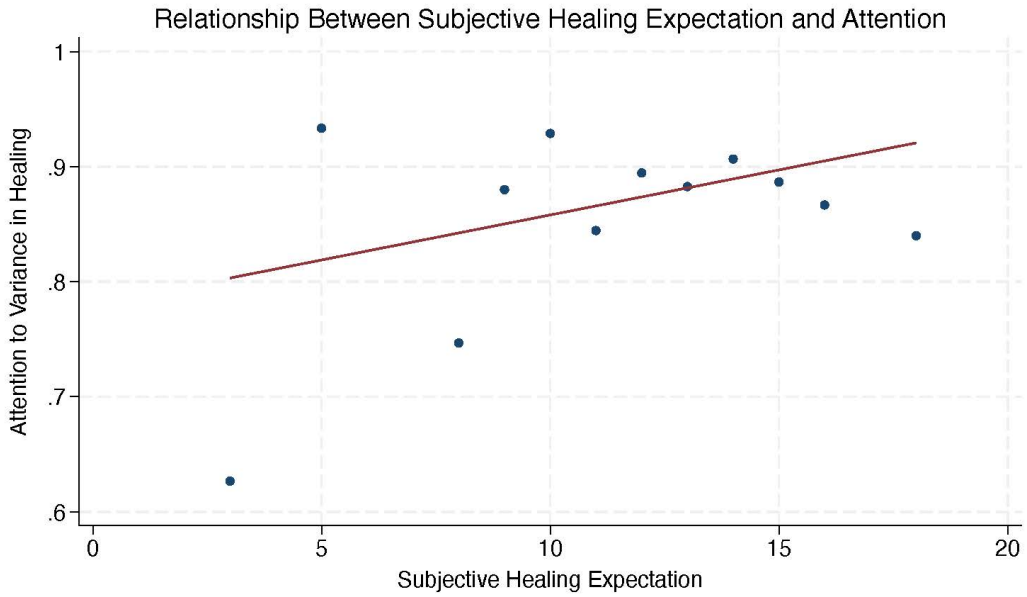

Figure 1: **Relationship between subjective healing expectations and attention.** Individuals who expect faster recovery also devote more attention to subtle changes in their bruises ($r \approx 0.35$). Each dot is a participant; the line is an OLS fit.

it is unchanged, then take the *mean fraction of dimensions flagged as changed*. Scores therefore range from 0 (no change ever noticed) to 1 (every dimension judged different at every wave); higher values reflect closer, more variance-sensitive monitoring-analogous to a passenger who continually registers subtle speed shifts in a plane overhead.

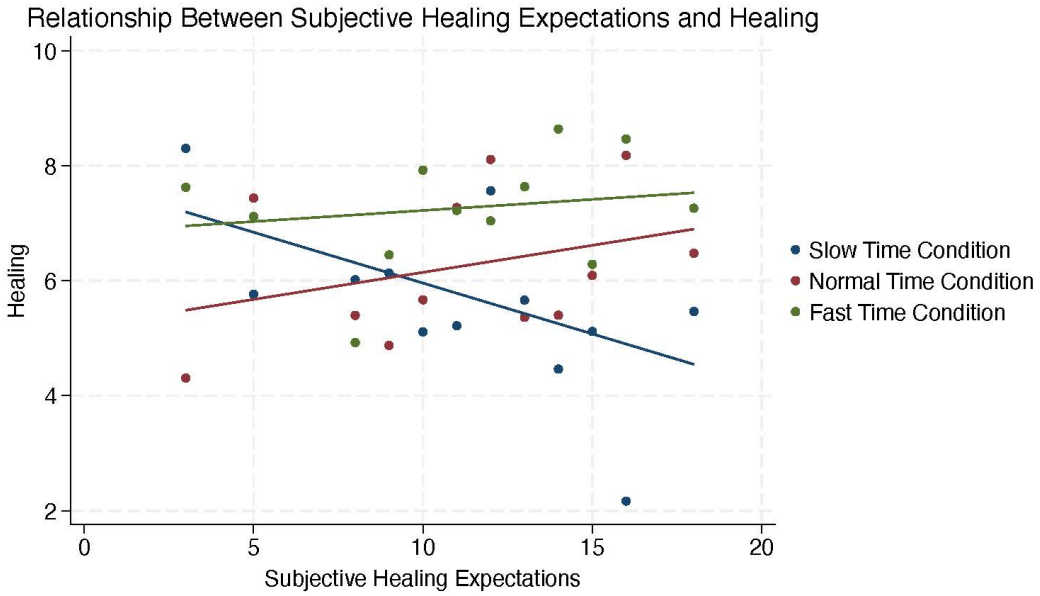

Figure 2: **Expectation moderates the timer cue.** Higher subjective expectations predict greater objective healing in the *fast-time* session (green) and in the *normal* session (red), but a negative slope in the *slow-time* session (blue). Lines show within-condition OLS fits; points are participant-sessions.

Regression evidence for prior– and attention–moderation. Table 2 summarizes two subject-fixed-effects specifications that mirror the binscatter patterns.

- **Expectation channel.** In Column (1) the interaction between *Subjective Expectation* and the *Fast-timer* dummy is positive and significant ($\beta = 0.237$, SE $= 0.105$, $p = 0.02$). The accelerated time cue therefore yields the largest objective healing among participants who initially believed they would heal quickly, exactly as the EMH model predicts when a strong prior aligns with a pro-healing context.

- **Attention channel.** Column (2) adds the *Attention Proxy* and the binary *High-Expectation* indicator (above-median prior). The triple interaction HighExp × Attention Proxy × Fast is large and highly significant ($\beta = 3.10$, SE $= 1.21$, $p < 0.01$). Participants who both expected rapid recovery and attended closely to small changes in their bruise experienced the greatest physiological benefit from the fast timer.

- **Joint implication.** Healing is maximized when the external context (fast timer) signals substantial elapsed time, the participant's prior favors rapid recovery, and their attention

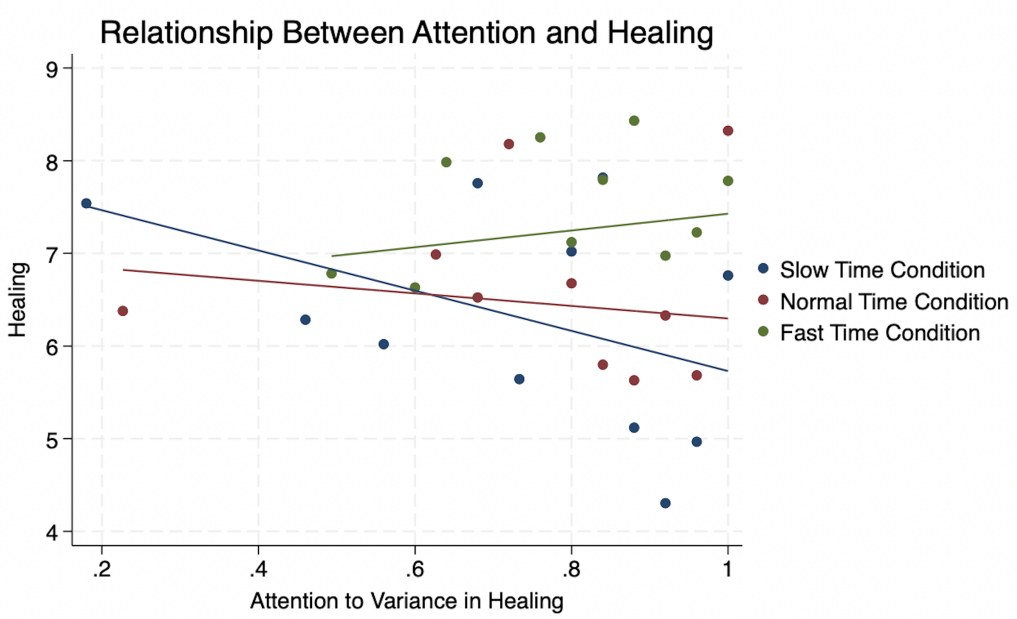

Figure 3: **Attention moderates the timer cue.** Greater moment-to-moment attention to variance in the bruise boosts healing under the *fast-time* cue but dampens healing under the *slow-time* cue, mirroring the model's prediction that the attention weight $w(M)$ amplifies contextually relevant signals.

weight $w(M)$ is high. This convergence of prior and attention echoes the prior-plus-weight mechanism formalized in Eq. (1).

Table 2: Expectations, attention, and objective healing

	(1) Healing Cont. Exp.	(2) Healing HighExp × Attn
Main regressors		
Subjective expectation (cont.)	−1.919*	—
	(0.996)	
High Expectation (> median)	—	−0.843
		(0.541)
Attention proxy	—	−0.616
		(0.399)
Timer dummies (baseline = slow 14 min)		
Normal 28 min	−2.919	−2.302
	(1.797)	(1.760)
Fast 56 min	−1.096	−0.964
	(1.161)	(1.147)
Key interactions		
Expectation × 28 min	0.295*	—
	(0.153)	
Expectation × 56 min	0.237**	—
	(0.105)	
HighExp × Attention	—	0.421
		(0.265)
HighExp × 28 min	—	0.553
		(0.484)
HighExp × 56 min	—	0.879*
		(0.491)
Attention × 28 min	—	1.063
		(0.673)
Attention × 56 min	—	2.247***
		(0.757)
HighExp × Attn × 28 min	—	−0.471
		(0.619)
HighExp × Attn × 56 min	—	3.103***
		(0.981)
Subject fixed effects	Yes	Yes
Session-order controls	Yes	Yes
Observations	1 825	1 825
Participants (clusters)	25	25
Within R^2	0.120	0.173

Robust standard errors clustered by participant in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. *High Expectation* = 1 if a participant's baseline expectation of healing speed relative to others exceeds the sample median. The *Attention proxy* is the within-session variance of five healing-observation items (details in main-text footnote). Reference timer condition is the *slow* 14-minute clock.

Heterogeneous treatment effects

Estimating heterogeneous treatment effects (HTEs) is a central goal across numerous fields, including personalized medicine and economics. In both randomized controlled trials and observational studies, random forests have emerged as a versatile and effective tool for uncovering such effect heterogeneity (51; 30). HTEs manifest as interaction effects between the treatment and covariates, and the number of possible interactions—especially higher-order and non-linear ones—can quickly become overwhelming in high-dimensional data. Manually specifying and testing such models is not only tedious and time-consuming but also prone to model misspecification and overfitting. Random forests, by contrast, offer a data-driven and flexible alternative. In particular, "causal forests" introduced by (30), and implemented in the R package `grf` (52), provide a principled framework for nonparametric HTE estimation. These methods rely on a two-stage procedure—often termed "local centering" or "orthogonalization"—that begins by removing the influence of observed covariates on both the outcome and the treatment assignment. This allows the forest to focus exclusively on estimating the residual treatment effect heterogeneity, improving both robustness and interpretability in complex settings such as health data.

In addition to their predictive flexibility, causal forests in `grf` also provide tools for interpretability, including variable importance measures tailored to HTE estimation, which help identify which covariates contribute most to treatment effect variation. Since HTEs in casual forests are not constrained to be linear, `grf` furthermore supports *partial dependence plots* (PDPs) (53) in order to generate smooth and simpler summaries of complex, high-dimensional HTE surfaces. A PDP shows how the estimated treatment effect varies with one specific covariate, averaging over the distribution of all other covariates in the data. More formally, it plots the expected conditional average treatment effect (CATE) as a function of a focal variable, marginalizing over the remaining features. This allows researchers to visualize and interpret the marginal relationship between a given covariate and the heterogeneity in treatment effects. Such plots can help detect important effect modifiers and support scientific insight into which subpopulations benefit most or least from a treatment.

Who benefits most from the fast-timer cue?

Moderators of the 56-vs-14-minute effect. To explore why some people heal more under the *fast* 56-minute clock than under the *slow* 14-minute clock we fit a *causal forest*[3] to the session-level healing scores ($N = 75$). The model predicts, for each participant-session, the expected difference $\widehat{\text{ATE}}_{56-14}$ in objective healing if the timer were fast rather than slow.

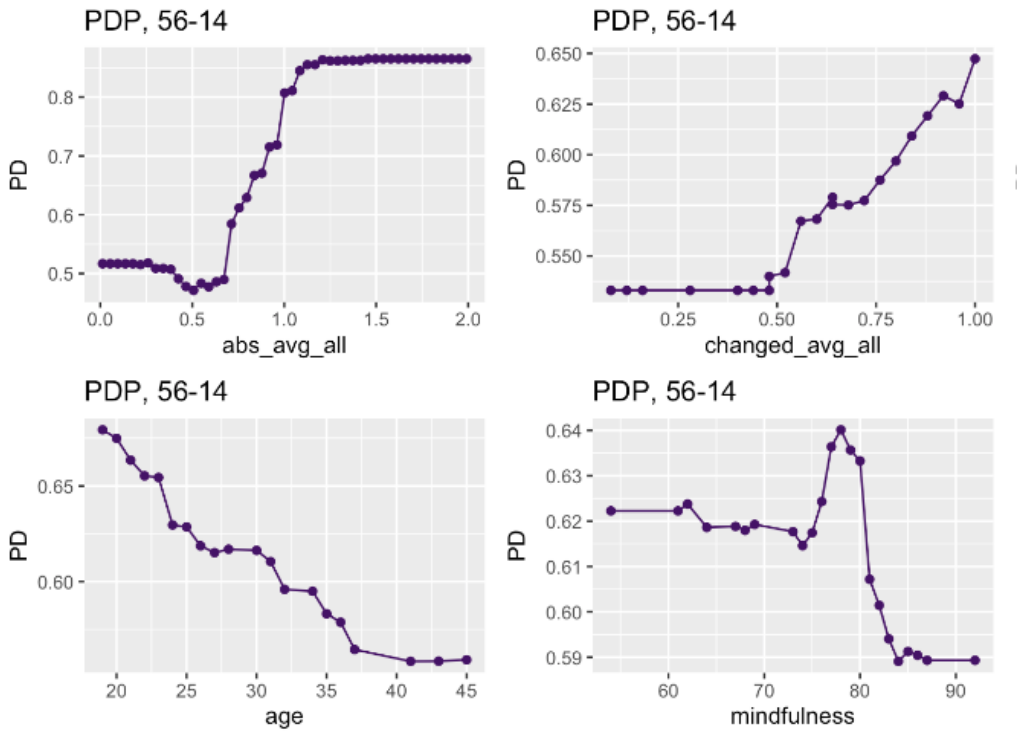

Figure 4: **Partial-dependence plots (PDPs) from the causal forest.** Each quadrant traces the predicted healing advantage of the *fast* (56-min) timer over the *slow* (14-min) timer as one moderator varies and all others are held at their observed values. Points are forest means; lines connect them for clarity.

Figure 4 shows the PDPs for four key covariates :

- **Absolute variance noticed (*abs_avg_all*).** Participants who perceived larger *absolute* swings in healing intensity across observations (top-left panel) showed sharply larger benefits once the metric exceeded ≈ 0.8. In other words, keen observers of change profited most from the fast-timer suggestion.

[3]We used the `grf` package (52) with default honest splitting and 2 000 trees; standard errors come from the built-in jack-knife. We cluster on the subject level and choose a minimum node size of 20.

- **Change frequency (*changed_avg_all*).** This index is simply the *fraction* of the 25 wave-by-dimension checks in which any change (better *or* worse) was reported. Participants who flagged change more often also gained more from the fast-timer cue, although the slope is gentler than for *abs_avg_all*.
- **Age.** Younger participants were more sensitive to the time cue (bottom-left). The forest slope flattens after about 35 years, suggesting developmental or motivational differences in how strongly elapsed-time information is trusted.
- **Trait mindfulness.** The mindfulness scale (LMS) shows an inverted-U pattern (bottom-right): moderate trait mindfulness ($\approx$ 75-80) is associated with the largest gains, whereas very low or very high scores yield smaller effects.

Taken together, the forest results reinforce the main EMH finding: the external "more time has passed" cue yields its strongest physiological impact when the individual *attends to healing variance* and holds a *present-focused mindset*. Age further gates this pathway, hinting at developmental factors worth exploring in future interventions.

Mindfulness as a second-order moderator. Does close, non-judgemental attention (high LMS scores) potentiate the *variance-gated* response we observed above? To answer this we re-estimated heterogeneous timer effects in the sub-sample scoring $\geq$ 80 on the Mindful Attention Awareness Scale (top quartile, $N = 46$ sessions). Figure 5 plots the partial-dependence of the 28-14 and 56-14 contrasts on the same absolute-variance index *abs_avg_all*.

The pattern reinforces the EMH prediction that *context, prior, and attention must align*. Even among already attentive, mindful individuals, a strong context cue (*fast* timer) *and* salient evidence of change ($abs\_avg\_all \gtrsim 0.8$) help unlock the maximal placebo-like benefit.

Belief-Reset in the Synergistic-Mindsets Trial. To illustrate how EMH can sharpen inference from existing research, we re-examined data from Yeager and colleagues' synergistic-mindsets trial (54). In that multicohort study, more than 2,500 U.S. adolescents completed a 30-minute online lesson that reframed both intelligence and stress as malleable and potentially growth-enhancing. The original authors showed robust average gains in academic performance and stress-related

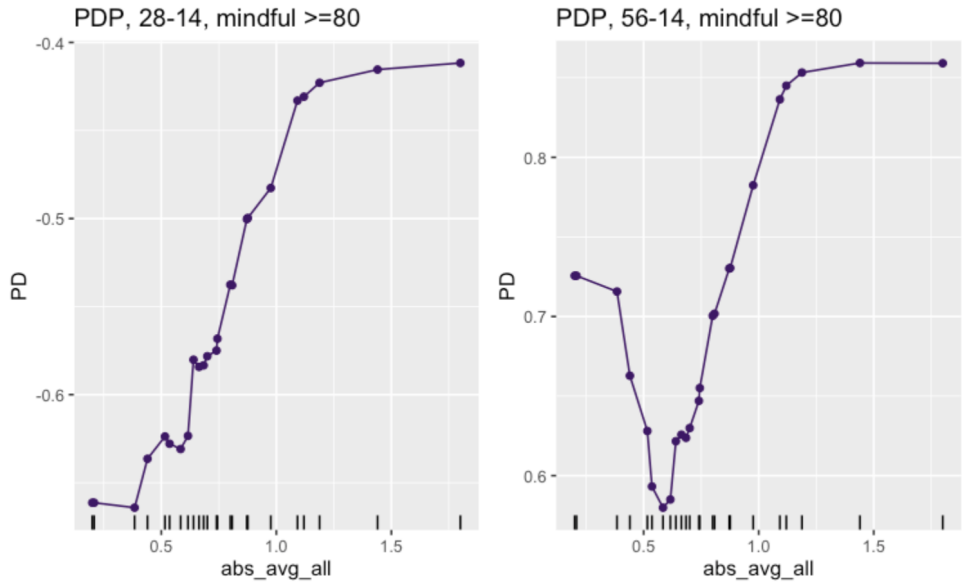

Figure 5: **Highly mindful participants respond only to the *fast* time cue-and only when they notice sizeable absolute change.** Each line is the causal-forest partial-dependence of the 28 min–14 min (left) or 56 min–14 min (right) contrast, conditional on LMS $\geq$ 80. Dots mark decile averages; rugs show the distribution of *abs_avg_all* within this sub-sample. For the *normal* timer (28 min) the effect remains negative across the range, but under the *fast* timer (56 min) the effect climbs steeply once participants report noticing $\gtrsim$ 0.8 units of absolute change.

physiology. Because the intervention explicitly targets priors about struggle ("fixed" $\rightarrow$ "growth") and about bodily arousal ("harmful" $\rightarrow$ "helpful"), it offers a natural laboratory for the EMH claim that belief shifts redirect attention and, in turn, modulate health-relevant bodily processes. Below we re-model their open dataset to ask not just whether the lesson worked, but for whom and through which EMH pathway it worked.

As shown in Table 3, a single 30-minute "mindsets" module does its best work on the students who start out most convinced that talent is fixed: for every one-unit rise in their baseline fixed-mindset score, the intervention's benefit climbs by roughly a tenth of a standard-deviation. In plain English, the kids who were most certain that "you either have it or you don't" re-interpreted classroom stress the most once they were shown that abilities grow and that a racing heart can fuel learning. The pattern is consistent with an EMH account in which updating a strongly negative prior redirects attention and downstream stress responses. Far from being a blanket pep talk, the lesson acts like a targeted "prior reset" that unlocks healthier appraisals and physiology precisely where the gap between old belief and new evidence is widest.

Table 3: Reanalysis of the synergistic-mindsets dataset 1 (54) reveals a significant interaction between a fixed mindset and the treatment (highlighted in bold letters); each unit of fixed mindset adds about 0.1 SD to the intervention benefit.

	Dependent variable:
	Positive Stress Mindset (sd)
Mindset Intervention	−0.568**
	(0.280)
baseline Positive Mindset	0.154***
	(0.029)
baseline Fixed Mindset	0.060**
	(0.024)
baseline Test Anxiety	0.037
	(0.025)
baseline PSS (Perceived Stress Scale)	0.213***
	(0.046)
baseline Expectancy	−0.095***
	(0.015)
Grade	−0.026*
	(0.014)
Female	−0.003
	(0.040)
First Generation	0.104***
	(0.039)
Non-Native Good English	0.167***
	(0.043)
Non-Native Poor English	0.184**
	(0.084)
Intervention × baseline Positive Mindset	0.010
	(0.041)
Intervention × baseline Fixed Mindset	**0.102*** **
	(0.035)
Intervention × baseline Test Anxiety	0.006
	(0.035)
Intervention × baseline PSS	−0.038
	(0.066)
Constant	2.556***
	(0.261)
Observations	2,534
R^2	0.172
Adjusted R^2	0.167
Residual Std. Error	0.955 (df = 2518)
F Statistic	34.859*** (df = 15; 2518)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 4: Reanalysis of the synergistic-mindsets dataset 5 (54) shows a significant overwhelm × intervention interaction (highlighted in bold letters): each unit increase in perceived stress reduced the intervention's effect by about one standard deviation.

	Dependent variable:
	Positive Stress Mindset (sd)
Mindset Intervention	−3.308*
	(1.861)
baseline Cortisol	0.007
	(0.011)
baseline Fixed Mindset	0.120
	(0.144)
baseline Positive Mindset	0.339**
	(0.144)
baseline Selfesteem	−0.198
	(0.144)
baseline PSS (Perceived Stress Scale)	−0.588*
	(0.320)
baseline Test Anxiety	0.061
	(0.102)
Grade	−0.037
	(0.169)
Sex	−0.504**
	(0.193)
Age	0.041
	(0.154)
Intervention × baseline Cortisol	−0.008
	(0.011)
Intervention × baseline Fixed Mindset	0.034
	(0.190)
Intervention × baseline Positive Mindset	−0.279
	(0.190)
Intervention × baseline Selfesteem	0.151
	(0.182)
Intervention × baseline PSS	**1.076****
	(0.422)
Intervention × baseline Test Anxiety	0.010
	(0.157)
Constant	1.497
	(5.018)
Observations	114
R^2	0.249
Adjusted R^2	0.125
Residual Std. Error	0.918 (df = 97)
F Statistic	2.005** (df = 16; 97)
Note:	*p<0.1; **p<0.05; ***p<0.01

In a laboratory follow-up, the same 30-minute video swung teenagers' views of stress toward the "can-be-helpful" side by more than three standard deviations—an eye-popping shift for such a brief intervention. Yet the gain was muted in students who arrived already feeling chronically overwhelmed: every notch higher on a perceived-stress scale trimmed the lesson's impact by about one standard deviation (See Table 4). The EMH explains the pattern neatly: a persuasive new story can rewrite a threat-based prior, but when the body is already signaling high distress, those noisy sensations compete for attention and blunt the narrative's reach.

Open-Label Placebo Effects To further demonstrate how EMH can enhance inferential precision in existing research, we reexamined data from the investigation by Leibowitz et al. on the role of patient beliefs in open-label placebo effects (55). In this study, 148 participants underwent a

Table 5: Reanalysis of the open-label placebo study (55) confirms a significant interaction between low placebo belief and treatment (highlighted in bold letters).

	Dependent variable:
	wheal postTreatment
rationale	−0.38
	(0.25)
low placebo belief	−2.98
	(1.96)
wheal baseline	0.95***
	(0.23)
rationale × low placebo belief	**1.74*** **
	(0.64)
low placebo belief × wheal baseline	0.39
	(0.38)
Constant	2.03*
	(1.10)
Observations	77
R^2	0.37
Adjusted R^2	0.32
Residual Std. Error	1.003 (df = 71)
F Statistic	8.177** (df = 5; 71)
Note:	*p<0.1; **p<0.05; ***p<0.01

histamine skin prick test and were randomly assigned to one of four conditions that incorporate

key components of open-label placebo interventions: a supportive provider relationship, a medical ritual, positive expectations and a placebo rationale. (The main outcome of interest is the size of the resulting red raised bump from the allergic reaction referred to as a 'wheal'.) Although the original study did not find main effects of the condition on allergic responses, the rational intervention significantly attenuated physiological allergic reactions among participants with strong placebo beliefs. In addition, we find that the rational intervention significantly increased allergic responses among participants with especially low prior beliefs about the effectiveness of placebo, which is shown in Table 5.

The findings from (55) closely align with the Bayesian Embodied Model of Health, which views physiological responses as the result of integrating prior beliefs with bodily evidence. In their study, the placebo rationale did not produce uniform effects; instead, its impact depended on participants' existing beliefs about placebo effectiveness. Those with strong placebo beliefs showed reduced allergic responses, consistent with a mindset that generates more positive priors and down-weights threat-related bodily cues. Conversely, those with especially low placebo beliefs showed increased allergic reactions, indicating that the same intervention can amplify negative priors or heighten attention to symptoms. This pattern illustrates EMH's central claim: identical inputs can lead to opposite physiological outcomes when interpreted through different belief-driven priors and attention weights.

Discussion and Conclusion

Across three very different contexts—a laboratory bruise-healing task, a 2,500-student field trial and a placebo study —we find that health-relevant outcomes follow the logic of an Embodied Model of Health in which *priors* (mindsets) and an *attention weight* jointly determine how incoming sensations revise belief and physiology. The fast-timer cue sped healing more when participants both expected rapid recovery and closely monitored change; the synergistic-mindsets lesson helped most when it directly contradicted a fixed-ability prior and when bodily distress was not shouting over the new narrative; and in the placebo study, the same intervention decreased allergic reactions among participants with strong placebo beliefs but increased them among those with very low beliefs, demonstrating how identical inputs can produce divergent outcomes through the interplay

of prior expectations and attentional weighting.

Theoretical contribution. EMH unifies the literature on placebo, stress, and mindset within a balanced Bayesian update. Every parameter—prior strength, attention weight, context cue—maps onto a measurable or manipulable quantity, enabling systematic tests rather than post-hoc story-telling.

Practical implication. Mind-body interventions can be *tuned with precision*: shift beliefs when priors are strongly negative, train attention when bodily noise is high, and align both whenever possible.

Limitations and future work. While the EMH framework offers a comprehensive model, the complementary empirical studies we conducted face several limitations. First, much of the evidence is correlational, making it difficult to establish causal direction between priors, attention, and physiological outcomes. Second, many measures relied on questionnaires or brief behavioral assessments, which may not fully capture the complexity of embodied inference. Third, most studies focused on short-term effects, leaving the dynamics of longer-term feedback loops largely unexplored. Future work should therefore (i) embed continuous or repeated physiological recording in large-scale trials, (ii) manipulate attention directly (e.g., mindfulness vs. distraction) alongside belief, and (iii) extend EMH modeling and empirical tests to chronic conditions where feedback loops unfold over longer timescales.

In sum, treating health as an embodied inference problem does more than tidy up disparate findings—it provides a quantitative blueprint for interventions that harness beneficial cognitive loops while disrupting harmful ones.

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