

Stress as a Recovery Speed System

Oct 2, 2026 · Charles B. Morris III, Ph.D. (ORCID <https://orcid.org/0009-0008-8507-9235>) · White paper, not peer reviewed

Abstract

Stress is widely treated as a load to be reduced. This white paper argues that the more useful question for human performance is not how much stress a person carries but how quickly their physiology returns toward baseline once the demand ends. Reviewing published research on allostatic load, stress physiology, cardiovascular and vagal recovery, and autonomic flexibility, the paper sets out a recovery speed model: acute stress responses deploy capacity, and the cost associated with stress appears most clearly when activation is prolonged and recovery is incomplete. Three lines of evidence support treating recovery as a distinct variable. Short-term and long-term stress exposures have different physiological consequences. Prolonged activation driven by worry and rumination has been proposed as a mechanism linking stressors to health outcomes. Delayed physiological recovery after a stressor has been associated with adverse cardiovascular outcomes independently of the size of the initial response. The paper also states plainly where the evidence is thin. Recovery has historically been measured far less often than reactivity, pooled effects for recovery from psychological challenge are small, and most of the relevant research is associative rather than causal. The practical implication for executives, athletes and operators is that recovery between demands deserves the same deliberate attention normally given to effort, and that recovery speed can be tracked, and may be trainable, although direct evidence for training it is still missing.

Keywords

stress recovery, allostatic load, heart rate variability, autonomic regulation, human performance, recovery speed, resilience, nervous system regulation, occupational performance, slow breathing

Introduction

The dominant public model of stress is subtractive. Stress is framed as a quantity to be lowered, and the advice that follows is to do less, escape more, and find calm. For people whose work is built on sustained demand, that advice fails in practice, because the demand is not optional and is often the point.

There is a second model available in the research literature, and it is more useful. Stress responses are coordinated adaptations that mobilize energy and attention to meet a demand. The autonomic and neuroendocrine systems that drive them are regulated through limbic, hypothalamic and

brainstem circuits, and prolonged exposure changes those circuits rather than simply adding wear (Ulrich-Lai and Herman, 2009). The response itself is not the liability. The liability accumulates when the response does not resolve.

This distinction has a name in the stress literature. Allostasis describes the process of maintaining stability through change, and allostatic load describes the physiological cost of repeated or poorly regulated allostatic activity (McEwen and Stellar, 1993). A systematic review of 267 original studies found that higher allostatic load is consistently associated with worse physical and mental health outcomes in adults (Guidi et al., 2020). The concept is well established. What is less often made operational, especially outside clinical settings, is the variable that sits inside it: how long activation persists after a demand ends, and how completely the system returns toward baseline.

This paper makes the case for treating that variable, recovery speed, as a primary performance metric rather than an afterthought. The argument runs in four steps. First, the current public model misdirects effort toward reducing exposure. Second, the research supports a framework in which capacity is deployed through activation and restored through recovery, with regulation understood as the ability to move between those states. Third, there is published evidence that incomplete or delayed recovery carries measurable cost, alongside clear limits on how strongly that evidence can be stated. Fourth, recovery can be observed, and there are plausible ways to practice it, which makes it worth acting on while the direct evidence for training it is still being built.

The framework presented here draws on the author's methodology in applied human performance work with executives, athletes and military personnel, and on the argument developed in the author's book *Stress: Our Human Superpower*. Where the paper makes physiological claims, it cites published research and distinguishes what that research establishes from what practitioner observation suggests.

The Problem With the Current View

Three assumptions in the prevailing view of stress send capable people toward interventions that cannot work for them.

The first assumption is that less stress is the goal. This treats activation itself as the problem. The research does not support that reading. Short-term stress exposure, lasting minutes to hours, has been reviewed as enhancing rather than suppressing immune function, with suppression and dysregulation associated with long-term exposure instead (Dhabhar, 2014). Earlier theoretical work proposed that intermittent exposure to demand produces what the author called physiological toughness, a pattern of low resting sympathetic activity combined with strong arousal when challenged (Dienstbier, 1989). Both lines of work point the same direction. The pattern associated with durability is not low exposure. It is exposure followed by return.

The second assumption is that recovery happens on its own once conditions improve.

For people whose conditions do not improve, this is a plan that never activates. It also misses something the literature has flagged for decades. Worry and rumination have been proposed to prolong stress-related physiological activation after the stressor itself has passed, which would mean the duration of activation is partly decoupled from the duration of the event (Brosschot, Gerin and Thayer, 2006). On that account, waiting for circumstances to change does not reliably end activation, because the activation is being sustained internally.

The third assumption is that stress is primarily a psychological matter, addressed by thinking differently. Appraisal clearly matters. But the systems involved are autonomic and neuroendocrine, and they are regulated by circuits that respond to physiological input, not only to interpretation (Ulrich-Lai and Herman, 2009). A model that offers only cognitive tools leaves the faster physiological inputs unused.

There is a fourth problem, and it is methodological rather than conceptual. Recovery has been measured far less often than reactivity. A review of laboratory stress reactivity protocols found that while 63 percent of reviewed studies collected recovery data, only 23 percent reported it, and the authors argued that recovery deserved equal standing with reactivity as an object of study (Linden et al., 1997). Nearly three decades later, a scoping review of cardiac vagal recovery after acute psychological stress covering 294 studies concluded that inconsistent recovery windows, posture reporting and stressor types across the literature still limit quantitative synthesis (Roddick et al., 2025). Recovery is not a neglected idea. It is an under-measured one, which is a different and more tractable problem.

The Framework: Stress as a Recovery Speed System

The framework has four components. Each is stated as a proposition, with its evidential standing noted.

1. The stress response is a capacity deployment system

When a demand appears, sympathetic activity rises and neuroendocrine signaling mobilizes energy. Attention narrows, reaction time shortens, and stored fuel becomes available. These are the coordinated outputs of a system built to meet demand, regulated through limbic, hypothalamic and brainstem circuits (Ulrich-Lai and Herman, 2009). Framing this deployment as damage is a category error. The appropriate question is not whether the system activates but whether it also stands down.

2. The functional distinction is activation that resolves versus activation that persists

The table below sets out the distinction as this framework uses it. The physiological asymmetry between short-term and long-term exposure is supported by review evidence in the immune literature (Dhabhar, 2014). The behavior of the recovery phase is where the framework places its emphasis.

	Activation that resolves	Activation that persists
Duration	Bounded by the demand	Outlasts the demand
Recovery phase	Completes, system returns toward baseline	Incomplete or absent
Functional role	Deploys capacity, then restores it	Capacity deployed without restoration
Associated physiology	Short-term exposure associated with enhanced immune function (Dhabhar, 2014)	Long-term exposure associated with suppression and dysregulation (Dhabhar, 2014); cumulative cost described as allostatic load (McEwen and Stellar, 1993)
Subjective signal	Felt and then released	Often quiet, experienced as a raised baseline

The second column is not a more intense version of the first. It is the first column with the return phase missing. This is the sense in which the paper treats duration without recovery as the more actionable variable, and the limits on that claim are stated in the Limitations section below.

3. Regulation is the capacity to move between states, not to stay in one

A well-regulated system is not a quiet one. It is one that can shift. Heart rate variability has been used as an index of the flexibility of autonomic input to the heart, and the standards document that established its measurement conventions is explicit that it reflects fluctuation in autonomic input rather than mean autonomic level (Task Force of the ESC and NASPE, 1996). Higher vagally mediated variability has been theorized to index the integrity of the circuits supporting self-regulation and executive function (Thayer and Lane, 2000; Thayer et al., 2009).

This matters for how the goal is defined. If the target were low arousal, the intervention would be sedation. If the target is flexibility, the intervention is training the transition in both directions, which is what the practical section addresses.

4. Recovery speed is the variable that compounds across a day

When demands arrive in sequence and each one resolves, the system meets each from a comparable starting point. When they arrive in sequence and activation persists between them, each demand begins from a higher baseline than the last. Over a working day, that difference accumulates not because the demands grew but because the gaps stopped functioning.

This is the component of the framework that rests most heavily on practitioner observation rather than on direct experimental demonstration. The supporting research establishes that prolonged activation is associated with cost (Guidi et al., 2020; McEwen and Stellar, 1993), that internal processes can sustain activation past the stressor (Brosschot, Gerin and Thayer, 2006), and that delayed recovery has been associated with adverse outcomes (Panaite et al., 2015; Whittaker et al., 2021). The specific within-day accumulation model is presented as a working framework consistent with that evidence, not as a demonstrated finding.

Supporting Evidence

Four lines of published research bear on the recovery speed framework. Each is reported with the strength of evidence it actually carries.

Cumulative physiological cost is well established

The allostatic load construct, introduced to describe the physiological cost of repeated or poorly regulated adaptive activity (McEwen and Stellar, 1993), has accumulated substantial supporting evidence. A systematic review of 267 original studies searched through December 2019 found higher allostatic load or overload consistently associated with worse physical and mental health in adults (Guidi et al., 2020). The relevant caveat is that allostatic load is operationalized differently across studies, and the review does not isolate the duration of activation from its intensity. It establishes that cumulative physiological cost is real and measurable. It does not by itself establish which component of exposure drives that cost.

Short-term and long-term exposure differ in kind

A review of stress and immune function concluded that short-term stress responses, on the order of minutes to hours, tend to enhance immune function, while long-term stress is associated with suppression and dysregulation (Dhabhar, 2014). This is the clearest published support for treating duration as a functional dividing line rather than a matter of degree. Two limits apply. It is a narrative review rather than a pooled analysis, and it frames the contrast as short versus long exposure rather than as recovered versus unrecovered.

A separate theoretical account proposed that intermittent exposure to demand, as distinct from continuous exposure, builds a physiological profile combining low resting sympathetic activity with strong arousal under challenge (Dienstbier, 1989). This paper is dated and the evidence it draws on is mixed, so it is cited here as a framework consistent with the recovery speed model rather than as confirmation of it.

Activation can outlast the stressor, and delayed recovery has been linked to outcomes

The perseverative cognition hypothesis proposed that worry and rumination prolong stress-related physiological activation beyond the stressor itself, and that this prolongation may be part of how stressors affect health (Brosschot, Gerin and Thayer, 2006). This is a mechanistic hypothesis paper rather than an effect estimate, and it should be cited as such.

On outcomes, a meta-analysis of 37 studies covering 125,386 participants found that poor cardiovascular recovery after a stressor predicted adverse cardiovascular outcomes and mortality, with a summary correlation of r equals 0.17 (Panaite et al., 2015). Two features of that result matter for honest reporting. The effect was stronger for recovery from physical exercise, r equals 0.19, than for recovery from psychological challenge, r equals 0.09. The psychological-challenge studies were relatively few. A narrative review and opinion paper later argued that delayed blood pressure recovery predicts later hypertension, atherosclerosis and adiposity, and may function as a predictor separable from reactivity magnitude (Whittaker et al., 2021), while noting that the evidence is largely correlational and that no standardized recovery thresholds exist.

One meta-analysis bears on both sides of this question at once, and it is the most useful single source in this paper. Pooling prospective cohort evidence through December 2009, it examined 169 associations from 36 articles on stress reactivity and 30 associations from 5 articles on poststress recovery, each in relation to later cardiovascular status. Both predicted outcomes. Reactivity was associated with poorer later cardiovascular status at r equals 0.091, 95 percent confidence interval 0.050 to 0.132, and poor poststress recovery at r equals 0.096, 95 percent confidence interval 0.058 to 0.134, both p less than 0.001 (Chida and Steptoe, 2010). Incident hypertension was the outcome most consistently predicted by reactivity, and increased carotid intima-media thickness the outcome most consistently predicted by poor recovery.

Two conclusions follow, and the second is the argument of this paper. Intensity carries real predictive information, so any claim that it does not is unsupportable. And recovery carries predictive information of its own, at an effect size similar to reactivity's (r equals 0.096 against 0.091, a difference far smaller than the confidence intervals), drawn from far fewer associations (30 from 5 articles against 169 from 36). That asymmetry, comparable prediction from far less evidence, is the clearest available case for treating recovery as a variable worth measuring in its own right rather than as a footnote to reactivity.

Recovery can be observed and influenced

A scoping review of cardiac vagal recovery following acute psychological stress, covering 294 studies and 23,533 participants across 33 countries, found faster vagal recovery associated with resilience, attentional control and cognitive reappraisal, and blunted recovery associated with depression, cardiovascular risk and emotion dysregulation (Roddick et al., 2025). As a scoping review it yields no pooled effect sizes, and the authors identify wide variation in recovery windows,

posture reporting and stressor types as a barrier to meta-analysis. It is nevertheless the closest published match to the recovery speed construct, and the associations run in the expected direction.

On deliberate intervention, a systematic review and meta-analysis of 223 studies found that vagally mediated heart rate variability increased during slow breathing, immediately after a single session, and following multi-session training (Laborde et al., 2022). For RMSSD the pooled effect was Hedges g equals 0.530, 95 percent confidence interval 0.430 to 0.620, during practice; g equals 0.140, confidence interval 0.030 to 0.240, immediately after one session; and g equals 0.320, confidence interval 0.080 to 0.560, after multi-session interventions. One caveat belongs with those figures and is stated in the Limitations section: overall risk of bias was rated high in 215 of the 223 included studies. A meta-analysis of 12 randomized controlled trials with 785 adults found breathwork reduced self-reported stress relative to controls, with Hedges g equals minus 0.35, 95 percent confidence interval minus 0.55 to minus 0.14, and moderate heterogeneity at I^2 equals 42 percent (Fincham et al., 2023). The authors of that analysis explicitly urge caution and warn against a mismatch between enthusiasm and evidence: three trials were rated at high risk of bias and nine at some concerns, with none rated low risk overall. A separate systematic review and meta-analysis of heart rate variability biofeedback across 58 studies reported a small to moderate effect favoring the intervention, largest for anxiety, depression, anger and athletic or artistic performance, with effects larger against inactive than active controls (Lehrer et al., 2020). An earlier meta-analysis of 24 studies with 484 participants reported a considerably larger effect, Hedges g of roughly 0.81 pre-post and 0.83 against controls (Goessl, Curtiss and Hofmann, 2017), which rests on self-reported outcomes and variable study quality and is best read alongside the more conservative Lehrer estimate.

Taken together, these results support a modest and useful claim. Autonomic recovery is observable, it responds to deliberate respiratory intervention, and the measured effects on self-reported stress are real but small to moderate and drawn from a literature with acknowledged quality problems.

Practical Implications

The framework changes what gets measured and what gets scheduled. Four implications follow.

Treat the gap between demands as working time. If recovery carries predictive information distinct from reactivity, then the interval after a demand is not residual time. In applied performance work it is the point at which activation either resolves or carries forward. The practical instruction is to place a deliberate downshift at transitions, particularly at the close of a demand and at the end of the working day, rather than relying on the gap to do that work unprompted.

Use respiration as the accessible input. Among the inputs available to a person mid-day without equipment, voluntary slow breathing has the most direct published association with increased vagally mediated heart rate variability, observed during practice, immediately after a

single session, and following repeated sessions (Laborde et al., 2022). Effects on self-reported stress are small to moderate and drawn from a literature with quality limitations (Fincham et al., 2023), so the honest framing is a low-cost, low-risk input with measurable autonomic effect, not a decisive intervention.

Train the transition under load, not only at rest. The target is flexibility, the ability to shift state, rather than low arousal (Task Force of the ESC and NASPE, 1996; Thayer and Lane, 2000). A practice rehearsed only in quiet conditions is rehearsed in conditions that do not resemble the moment it is needed. In the author's methodology, regulation reps are run while engaged and on the move, so that the trained response is available under the conditions it was built for. This specific transfer claim comes from applied practice and is not established by the research cited here.

Measure recovery, with appropriate caution about the tools. Heart rate variability offers a window into autonomic flexibility, but the measurement conventions matter. Values recorded over different durations are not interchangeable, and age, sex, posture, respiration and health status all shift baselines (Shaffer and Ginsberg, 2017). A meta-analysis of portable device accuracy against electrocardiography found small pooled error overall but meaningful variation by metric, posture and sex, with high heterogeneity, and it predates current wrist and ring devices (Dobbs et al., 2019). The defensible use of consumer measurement is as a non-medical, week-over-week trend for an individual against their own baseline, not as a diagnostic value or a comparison between people.

For organizations, the implication is structural. If recovery between demands shapes the quality of the output that follows, then schedule density is a performance variable and not only a capacity question. Back-to-back scheduling removes the intervals in which activation would otherwise resolve. This follows from the framework rather than from a cited trial, and is offered as a hypothesis worth testing inside organizations that can measure it.

Limitations

This paper is a white paper. It has not been peer reviewed, and it should be read as a framework argument supported by published research rather than as original empirical work.

The central claim is stated more strongly in popular discussion than the evidence supports. The formulation that the line between acute and chronic stress is duration without recovery and not intensity overstates what the literature shows. Greater cardiovascular reactivity to laboratory stress has itself been associated with poorer later cardiovascular risk status (Chida and Steptoe, 2010), so intensity carries predictive information too. The position this paper defends is narrower: recovery is a distinct and under-measured factor alongside intensity, and it is the factor that may be more open to deliberate training.

The recovery-to-outcome evidence is strongest for cardiovascular measures and modest in size. The pooled correlation between poor cardiovascular recovery and adverse outcomes was r equals 0.17 overall and r equals 0.09 for recovery from psychological challenge specifically (Panaite et al., 2015). No systematic review or meta-analysis establishing that cortisol or heart rate variability recovery predicts health outcomes was located in preparing this paper, which is itself a finding worth stating.

Most of the relevant evidence is associative. The scoping review of cardiac vagal recovery reports associations, not causal effects, and its authors note that inconsistent methods across 294 studies prevent quantitative synthesis (Roddick et al., 2025). Faster recovery being associated with resilience and attentional control does not establish that training recovery produces those qualities.

The intervention literature has serious quality problems. In the breathwork meta-analysis, no trial was rated at low overall risk of bias, and the authors urge caution against overstatement (Fincham et al., 2023). In the largest slow breathing review, overall risk of bias was rated high in 215 of the 223 included studies, with some concerns in 5 and low risk in 1, and no formal certainty-of-evidence assessment was performed (Laborde et al., 2022). That is the most important caveat on the respiratory evidence cited here: the direction of effect is consistent across a very large literature made up largely of low-quality studies. Effect size estimates for heart rate variability biofeedback also vary substantially between analyses (Goessl, Curtiss and Hofmann, 2017; Lehrer et al., 2020), which suggests the true effect is not yet well characterized.

The within-day accumulation model is a working framework, not a demonstrated result. The proposition that unresolved activation from one demand raises the starting point for the next, compounding across a working day, is consistent with the cited research but has not been directly tested in the form presented here.

Practitioner observation is not evidence. Where this paper refers to patterns observed in applied work with executives, athletes and military personnel, those are uncontrolled observations from a practice with a self-selected population and no comparison group. They are offered to describe how the framework is applied, not to support its validity.

Scope. This paper addresses performance and wellness in generally healthy adults. It does not address clinical populations, and nothing in it constitutes guidance for any diagnosed condition.

Conclusion

The prevailing model asks how much stress a person is under. The more productive question for human performance is how fast they come back.

That shift has practical consequences. It moves attention from exposure, which high performers cannot reduce without giving up the work, to recovery, which they may be able to train. It identifies the interval after a demand as the place where capacity is either restored or quietly spent. And it

supplies a variable that can be observed over time rather than estimated by feel.

The published research supports this direction without settling it. Cumulative physiological cost is well established. Short-term and long-term exposure differ in kind rather than only in degree. Activation can persist past the stressor through internal processes. Delayed physiological recovery has been associated with adverse cardiovascular outcomes, modestly but consistently. Autonomic recovery is observable, and it responds to voluntary slow breathing with measured effects that are real, small to moderate, and drawn from a literature that acknowledges its own quality limits.

What is missing is direct work on recovery speed as a trainable performance variable in healthy, high-demand populations. Recovery has been collected far more often than it has been reported (Linden et al., 1997), and the methodological inconsistency that still prevents pooled analysis of vagal recovery data (Roddick et al., 2025) marks a clear opportunity. The framework set out here is offered as a basis for that work, and as a working model for practitioners who cannot wait for it.

The operative reframe is simple. Capacity is not the absence of demand. It is the speed of return.

Disclosure and Notice

Competing interests. The author founded and operates Fulcrum Performance Lab, LLC, a human performance business, and is the creator of the Morris Method, a commercial methodology. The author is also the author of the book *Stress: Our Human Superpower*, from which parts of this framework are drawn, and has a commercial interest in Nova Breath, an application that delivers breathing protocols and tracks heart rate variability. Readers should weigh these interests when assessing the framework presented here.

Status of this document. This is a white paper. It has not been peer reviewed and does not report original empirical research. Nothing in it should be represented as a peer-reviewed finding.

Not medical advice. This paper is educational and addresses wellness and human performance in generally healthy adults. It is not medical advice, does not diagnose or treat any condition, and is not a substitute for care from a qualified professional. Readers with any medical concern should consult a qualified healthcare professional before changing their practices.

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Sources consulted for verification

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