Biological Stress Test

Why Alignment Theory May Generalize Beyond Psychology and AI

Michael Nathan Bower — alignmenttheory.org

Abstract

Frameworks that claim broad explanatory scope should remain coherent when translated across domains. Alignment Theory proposes that human flourishing depends on the relationship between internal regulation (capacities maintained within the system itself) and external scaffolding (structures that assist or guide behavior from outside the system). If the framework describes a real structural dynamic rather than a domain-specific metaphor, similar patterns should appear in biological systems.

This paper conducts a conceptual stress test by translating the framework into biological contexts and examining several well-understood phenomena: muscular atrophy under disuse, immune calibration, chronic stress and allostatic overload, hormesis and adaptive stress, microbiome co-regulation, neural plasticity, and developmental critical periods. Across these cases, a consistent pattern appears: adaptive systems remain robust when external support scaffolds internal regulatory processes without replacing them. When external systems persistently substitute for internal regulatory capacity, systems may remain temporarily stable while becoming progressively less adaptive and less resilient.

These findings suggest that the internal–external regulation dynamic identified in Alignment Theory may reflect a general property of adaptive systems rather than a principle limited to psychology or moral development.

---

1. Introduction: A Cross-Domain Test

Alignment Theory emerged as an attempt to describe patterns in human moral development, cognition, and institutional life. At its center is a structural distinction between two sources of order: internal regulation, where coherence arises from capacities carried within the system itself, and external regulation, where coherence is maintained by structures that guide or constrain the system from outside.

External regulation is not inherently harmful. Institutions, technologies, and environments routinely enhance stability and capability. The critical distinction is how external support relates to internal regulatory capacity over time. The three modes of that relationship are clarified in section 2. When substitution occurs persistently, systems may remain operational while becoming progressively less adaptive, less resilient, and less capable of independent regulation.

If this dynamic reflects a real structural property of adaptive systems, similar patterns should appear in biological contexts. Biology therefore provides a powerful test environment for the framework.

2. Three Modes of External Support

Before examining biological cases, it is helpful to clarify how external support can interact with internal regulation. Biological systems reveal three distinct modes.

Supportive Co-Regulation

External interaction strengthens internal regulatory systems. Examples include symbiotic relationships, environmental feedback loops, and ecological interactions that stimulate adaptive development. In these cases the external element does not replace internal regulation but helps stabilize and train it.

Temporary Scaffolding

External support compensates for a limitation while preserving the pathway back to autonomous function. Medical interventions often operate in this mode. A cast stabilizes a broken bone while the body repairs itself. Ventilation can support breathing while respiratory systems recover. The scaffold is temporary and oriented toward restoration of internal function.

Substitutive Regulation

External systems perform functions that the organism would otherwise perform internally. When this substitution becomes persistent, internal capacity may weaken through disuse. This third pattern is the focus of the paper. The claim is not that external support is harmful, but that long-term substitution for load-bearing internal processes can gradually degrade adaptive capacity.

3. Muscle Atrophy and the Cost of Disuse

One of the clearest biological examples of load-bearing capacity is muscle tissue. Muscles are maintained through mechanical load. When the body experiences resistance through movement or exercise, biochemical signaling preserves and strengthens muscle fibers. When load disappears, the body reallocates resources and muscle mass declines. This process has been documented extensively in bedrest studies and spaceflight research, where the removal of gravitational load produces measurable atrophy within days (Booth & Criswell, 1997; LeBlanc et al., 1992).

This process is not pathological; it is an adaptive response to changing demands. However, it demonstrates an important principle: capacity is not preserved indefinitely in the absence of use. Protection from load may preserve short-term comfort while reducing long-term capability. The biological system reallocates resources away from capacities that are no longer exercised.

4. Immune Calibration and Over-Intervention

The immune system provides a more complex example of the same structural dynamic. Immune regulation depends on continual calibration through exposure and interaction. Signals from pathogens, environmental microbes, and internal physiological processes help the immune system maintain its ability to distinguish threat from tolerance. Strachan (1989) proposed that reduced microbial exposure in early childhood was associated with increased rates of allergic disease, a finding developed further by Rook (2010) into the old friends hypothesis, which argues that the immune system requires co-evolution with specific microbial partners to calibrate correctly.

External intervention is often necessary and beneficial. However, when regulation is persistently replaced by external control mechanisms — such as long-term suppression of immune signaling — the system may become less capable of independent discrimination and response. This phenomenon illustrates the difference between supporting a system's regulatory processes and replacing them entirely.

5. Chronic Stress and Allostatic Overload

Stress physiology provides a second pathway through which regulatory capacity can degrade. Under normal conditions, organisms respond to stress through adaptive regulatory responses that allow the organism to maintain stability in changing conditions, a process Sterling and Eyer (1988) termed allostasis and McEwen and Stellar (1993) developed into a framework for understanding stress-related disease.

Over time, repeated activation of these stress responses produces allostatic load: the cumulative physiological wear associated with sustained stress signaling. When the load exceeds the organism's capacity to restore equilibrium, the system enters allostatic overload (McEwen, 1998). Under these conditions the organism narrows its regulatory priorities toward short-term stabilization. Processes associated with growth, exploration, and integration decline.

The organism remains functional but becomes less flexible and less capable of adaptive response.

6. Hormesis and the Role of Adaptive Stress

Not all stress weakens biological systems. Many systems become stronger when exposed to moderate stressors. This phenomenon, known as hormesis, occurs when challenges such as exercise, temperature variation, or metabolic stress stimulate adaptive responses that increase resilience. Calabrese and Baldwin (2002) documented hormetic dose-response relationships across a wide range of biological systems, and Mattson (2008) extended the concept to neurological contexts, showing that moderate cognitive and metabolic challenges can strengthen neural resilience.

The key variable is not the presence of stress but the calibration of challenge. Too little challenge produces underdevelopment and fragility. Excessive challenge produces breakdown. Within an appropriate range, however, challenge stimulates strengthening and integration.

Adaptive development therefore depends not on eliminating stress entirely but on maintaining an environment where challenge remains within a range the system can integrate.

7. Microbiome Co-Regulation

The relationship between organisms and their microbiomes provides a particularly important refinement of the framework. Humans do not regulate themselves in isolation. The microbiome participates in metabolic regulation, immune signaling, and neurological processes. Health depends on continuous interaction between host systems and microbial communities (Turnbaugh et al., 2007). Cryan and Dinan (2012) documented the extent of gut-brain axis signaling, demonstrating that microbial communities influence mood, cognition, and stress responsivity through multiple neurochemical pathways.

This relationship demonstrates that not all external regulation is optional or temporary. Some forms of external interaction are constitutive components of internal regulation itself. The relevant distinction is therefore not simply internal versus external regulation, but the nature of the interaction between them.

Healthy microbiome relationships exemplify supportive co-regulation: external systems that help stabilize and maintain internal regulatory architecture rather than displacing it.

8. Neural Plasticity and Cognitive Capacity

Cognitive neuroscience provides a direct bridge between biological and psychological levels of analysis. Hebb (1949) proposed that synaptic connections strengthen when neurons fire together, establishing the foundational principle that neural structure is shaped by use. Subsequent research confirmed that learning and cognitive challenge produce measurable structural changes in neural tissue, including synaptic strengthening and cortical remapping. Draganski et al. (2004) demonstrated that skill acquisition produces detectable changes in gray matter density in humans, and Merzenich and colleagues documented extensive cortical remapping in response to altered sensory experience (Merzenich et al., 1984).

Conversely, capacities that are not exercised may decline through synaptic pruning or reduced neural activation. This use-it-or-lose-it principle demonstrates that cognitive capacities are not merely abstract functions. They are biological processes embedded in neural structure and maintained through repeated engagement.

Neural plasticity therefore provides one of the clearest examples of load-bearing capacity operating simultaneously at biological and cognitive levels — which is precisely what the generalization claim of the framework requires.

9. Developmental Critical Periods

Biology also shows that the timing of experience matters. Many regulatory capacities develop during critical periods of heightened plasticity. Hubel and Wiesel (1970) demonstrated that visual cortex development in cats required appropriate sensory input during a defined early window; deprivation during this period produced permanent functional deficits that could not be fully reversed by later experience. Hensch (2004) identified molecular mechanisms governing critical period opening and closure across multiple sensory systems. Kuhl (2004) documented analogous windows in human language acquisition, showing that sensitivity to phonetic distinctions declines sharply after the first year of life.

If the relevant experiences do not occur during these windows, development may be incomplete or permanently altered. This phenomenon suggests that the displacement of certain developmental challenges — particularly early in life — may prevent capacities from forming fully rather than merely weakening them through later disuse.

10. Toward a General Principle of Adaptive Systems

Across these biological cases, a consistent structural pattern appears.

> **Adaptive systems remain robust when external conditions support but do not replace the internal processes through which the system maintains its own coherence.**

Conversely, when external systems persistently substitute for load-bearing internal capacities, the system may remain stable while becoming progressively less adaptive, less resilient, and less capable of independent regulation.

The repeated appearance of this pattern across biological contexts suggests that the internal–external regulation dynamic may represent a general principle of adaptive systems rather than a concept limited to human cognition or moral development. Biological load-bearing capacity and psychological load-bearing capacity may therefore be understood not as metaphorical parallels but as instances of the same underlying structural dynamic operating at different levels of organization.

11. Conclusion

Biological systems provide a powerful environment for testing conceptual frameworks. When Alignment Theory is translated into biological contexts, its central distinction between internal regulation and external scaffolding aligns with several well-established phenomena: muscular atrophy under disuse, immune calibration, chronic stress responses, hormetic adaptation, microbiome co-regulation, neural plasticity, and developmental critical periods.

Across these cases the same structural principle appears repeatedly: capacities that are not exercised tend to degrade, while adaptive systems remain robust when external support strengthens rather than replaces internal regulatory processes.

If this pattern generalizes across biological, cognitive, and social domains, it suggests that the framework may be describing a common architecture of adaptive systems rather than a domain-specific theory. Establishing the empirical scope of this principle remains an open research question, but the biological evidence indicates that the structural dynamic Alignment Theory identifies is not merely metaphorical. It may be a recurring feature of how complex adaptive systems maintain their coherence over time.

References

Booth, F. W., & Criswell, D. S. (1997). Molecular events underlying skeletal muscle atrophy and the development of effective countermeasures. *International Journal of Sports Medicine, 18*(S1), S265–S269.

Calabrese, E. J., & Baldwin, L. A. (2002). Defining hormesis. *Human and Experimental Toxicology, 21*(2), 91–097.

Cryan, J. F., & Dinan, T. G. (2012). Mind-altering microorganisms: The impact of the gut microbiota on brain and behaviour. *Nature Reviews Neuroscience, 13*(10), 701–712.

Draganski, B., Gaser, C., Busch, V., Schuierer, G., Bogdahn, U., & May, A. (2004). Neuroplasticity: Changes in grey matter induced by training. *Nature, 427*(6972), 311–312.

Hebb, D. O. (1949). *The Organization of Behavior: A Neuropsychological Theory.* Wiley.

Hensch, T. K. (2004). Critical period regulation. *Annual Review of Neuroscience, 27*, 549–579.

Hubel, D. H., & Wiesel, T. N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *Journal of Physiology, 206*(2), 419–436.

Kuhl, P. K. (2004). Early language acquisition: Cracking the speech code. *Nature Reviews Neuroscience, 5*(11), 831–843.

LeBlanc, A., Rowe, R., Schneider, V., Evans, H., & Shackelford, L. (1992). Regional muscle loss after short duration spaceflight. *Aviation, Space, and Environmental Medicine, 63*(12), 1143–1146.

Mattson, M. P. (2008). Hormesis defined. *Ageing Research Reviews, 7*(1), 1–3.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840*, 33–44.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine, 153*(18), 2093–2101.

Merzenich, M. M., Kaas, J. H., Wall, J. T., Sur, M., Nelson, R. J., & Felleman, D. J. (1984). Progression of change following median nerve section in the cortical representation of the hand in areas 3b and 1 in adult owl and squirrel monkeys. *Neuroscience, 10*(3), 639–665.

Rook, G. A. (2010). 99th Dahlem conference on infection, inflammation and chronic inflammatory disorders: Darwinian medicine and the ‘hygiene’ or ‘old friends’ hypothesis. *Clinical and Experimental Immunology, 160*(1), 70–79.

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of Life Stress, Cognition and Health* (pp. 629–649). Wiley.

Strachan, D. P. (1989). Hay fever, hygiene, and household size. *BMJ, 299*(6710), 1259–1260.

Turnbaugh, P. J., Ley, R. E., Hamady, M., Fraser-Liggett, C. M., Knight, R., & Gordon, J. I. (2007). The human microbiome project. *Nature, 449*(7164), 804–810.