

Clinical Outcomes White Paper — No. 1 of 5

The Brain Reset Protocol

A Longitudinal Analysis of Neurological Symptom Change in ADHD-Pattern Adults

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Brain Health Assessment Outcomes Dataset

93.1%

Improved on Program

$p < .0001$

vs. Non-Program

n=89

Retested Adults

The Brain Reset Protocol: A Longitudinal Analysis of Neurological Symptom Change in ADHD-Pattern Adults

What 89 repeat assessments reveal about system correction versus symptom management.

You do not have an attention problem. You have an unmeasured system. Most people who struggle with focus, follow-through, and mental fatigue are told to try harder, sleep more, build better habits. None of it addresses what is actually happening at the neurological level — and almost none of it is measured before and after. This paper is not a testimonial. It is data.

Section 01

The Industry Runs on Before-and-After Photos. This Runs on Before-and-After Data.

Brain health, wellness, and executive-coaching programs report outcomes almost exclusively through anecdote: a quote, a photo, a testimonial video. Repeat, standardized measurement — the same person, the same instrument, months apart — is rare, because most programs never ask the client to test twice.

Fascia Training Institute's Brain Health Assessment (BHA) is a 118-item self-report instrument that scores four neurologically-linked systems — Dopamine, Acetylcholine, GABA, and Serotonin — along with a composite Total Symptom Score (0–115, lower is better). Over multiple years of intake, a subset of clients retested voluntarily: some after enrolling in the Brain Reset Protocol, some without enrolling at all. That second group is not a plant. It is what remained once the data was pulled — and it is what makes this analysis unusual: a real comparison group, inside a real client population.

This paper reports on **89 adults** who completed the BHA at least twice — **58 of whom enrolled in the Brain Reset Protocol** after their first assessment, and **31 who retested without enrolling**.

Section 02

What the Four Systems Represent

Symptoms are not the problem. Symptoms are the output of a system under load. The BHA maps self-reported symptoms onto four systems long associated with prefrontal and limbic function:

- **Dopamine** — focus, motivation, follow-through, impulse control. The system most consistently implicated in attention and executive function.
- **Acetylcholine** — memory, mental clarity, word-retrieval, learning speed.
- **GABA** — the brain's primary inhibitory (calming) system; regulates nervousness, tension, and the ability to downshift.
- **Serotonin** — mood stability and emotional regulation.

The anatomical substrate for executive function is the prefrontal cortex, and dopamine and norepinephrine — with acetylcholine and serotonin playing supporting roles — are the neurotransmitters that activate it. Executive-function disruption of this kind is documented across ADHD and related conditions [1].

Section 03

The Findings

93.1%

of program clients improved

-30.7%

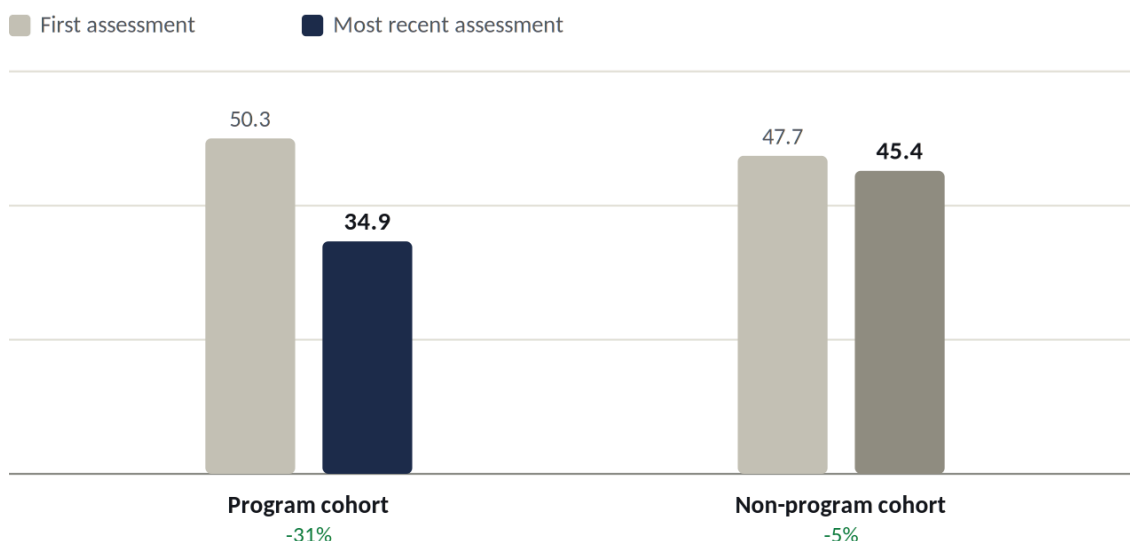
mean symptom score change

p<.0001

program vs. non-program

Clients who enrolled in the Brain Reset Protocol saw their Total Symptom Score fall from an average of **50.28** to **34.86** — a 30.7% reduction — over an average of 246 days (roughly 8 months) between first and most recent assessment. Clients who retested without enrolling moved from 47.68 to 45.42: essentially flat, and not statistically significant (p=0.3948).

Total Symptom Score — Program Cohort vs. Non-Program Cohort

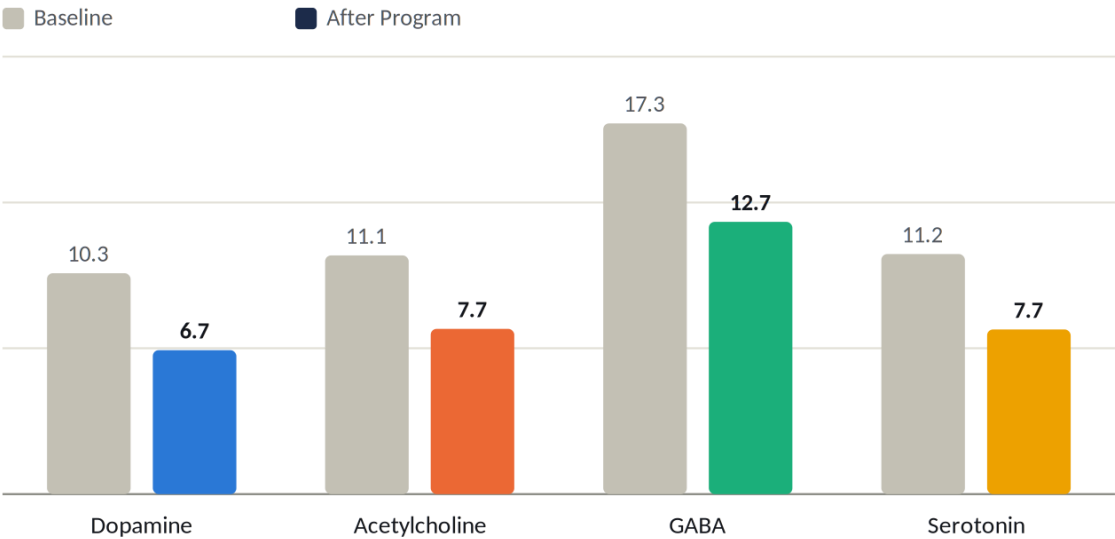


n=58 program clients, n=31 non-program retesters. Lower score = fewer/lighter symptoms. Between-group difference: p<.0001, Cohen's d=1.03 (large effect).

The gap between the two lines above is the finding. Time alone, or simply having taken the assessment once, does not explain the change in the program cohort. Enrollment does.

Every System Moved — Not Just One

All Four Systems, Program Cohort — Baseline vs. Most Recent Assessment



All four subscale improvements were statistically significant for the program cohort and significantly larger than the non-program cohort (Dopamine $p=0.0003$; Acetylcholine $p<.0001$; GABA $p=0.0007$; Serotonin $p=0.0068$).

System	Program: First	Program: Latest	Change	Non-Program Change	Between-Group Sig.
Total Symptom Score	50.28	34.86	-30.7%	-4.7%	$p<.0001$
Dopamine	10.31	6.72	-34.8%	-5.0%	$p=0.0003$
Acetylcholine	11.14	7.71	-30.8%	-0.3%	$p<.0001$
GABA	17.31	12.71	-26.6%	-2.4%	$p=0.0007$
Serotonin	11.21	7.69	-31.4%	-8.8%	$p=0.0068$
Severe-range items	1.16	0.41	-64.7%	-7.4%	$p=0.0074$

The Dopamine system — the one most tied to attention, follow-through, and impulse control — improved 34.8% in the program cohort, more than six times the 5.0% drift seen in clients who did not enroll. A deeper breakdown of the attention-and-executive-function pattern specifically is the subject of White Papers 2 and 3 in this series.

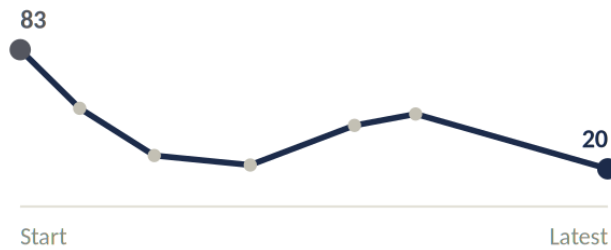
Section 04

Five Clients, De-Identified

Averages can hide the texture of real change — including the fact that recovery is rarely a straight line. The five cases below are drawn directly from the program cohort and use anonymized codes, not names.

Case GABA-1

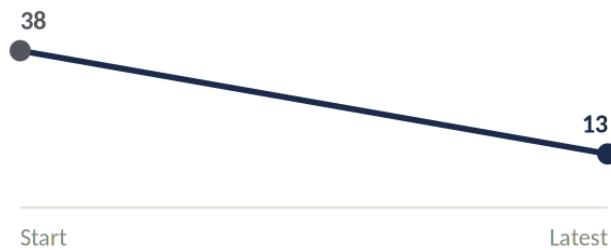
7 assessments over 434 days • Dominant system at intake: Gaba



83 → 20 total symptom score (-76%). Severe-range items: 4 → 0.

Case GABA-2

2 assessments over 89 days • Dominant system at intake: Gaba



38 → 13 total symptom score (-66%). Severe-range items: 0 → 0.

Case GABA-3

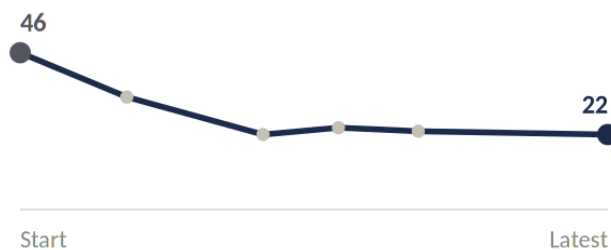
6 assessments over 397 days • Dominant system at intake: Gaba



27 → 11 total symptom score (-59%). Severe-range items: 0 → 0.

Case ACh-1

6 assessments over 375 days • Dominant system at intake: Acetylcholine



46 → 22 total symptom score (-52%). Severe-range items: 0 → 0.

Case SER-1

4 assessments over 392 days • Dominant system at intake: Serotonin



32 → 16 total symptom score (-50%). Severe-range items: 0 → 0.

Case GABA-1 is included deliberately because it is not a clean downward line — the score rose between the third and fifth assessments before dropping to its lowest point. Real physiology under real-life stress looks like this. A program that only shows straight lines is showing you a highlight reel, not data.

What This Data Does Not Prove

This is an outcomes analysis of a self-report instrument, not a randomized clinical trial. Three limits matter: **(1)** the Brain Health Assessment is an educational self-report tool, not a validated diagnostic instrument for ADHD or any other condition; **(2)** clients were not randomly assigned to the program — those who enrolled chose to, which may reflect motivation or severity differences that also affect outcomes; **(3)** this has not been through independent peer review. What the data does show, credibly: a real cohort of adults who retested the same instrument, months apart, and a program-enrolled group whose improvement was significantly and substantially larger than a same-population group who did not enroll — on every system measured.

Section 05

Why This Matters Beyond One Program

Adult attention and executive-function difficulty is not a niche complaint. Global meta-analytic estimates put symptomatic adult ADHD at roughly 6.8% of the adult population — on the order of 350 million adults worldwide — with executive-functioning deficits that are frequently under-captured by standard diagnostic checklists [2,3]. Most of those adults are never offered a system-level explanation. They are offered productivity apps and willpower.

The Position

The industry treats attention, follow-through, and emotional regulation as personality. This dataset treats them as systems — measurable, trackable, and, in this cohort, significantly more responsive to structured correction than to time alone. That is the entire argument of the Brain Reset Protocol, and now it has a number attached to it.

References

1. Papazian O, Alfonso I, Luzondo RJ. Executive function disorders. *Rev Neurol*. 2006;42(Suppl 3):S45-50. Anatomical substrate of executive function is the prefrontal cortex; dopamine and norepinephrine (and, to a lesser degree, acetylcholine and serotonin) are the neurotransmitters activating prefrontal neurons. Executive-function disorders are documented in ADHD. <https://pubmed.ncbi.nlm.nih.gov/16642451/>

2. Song P, Zha M, Yang Q, Zhang Y, Li X, Rudan I. The prevalence of adult attention-deficit hyperactivity disorder: A global systematic review and meta-analysis. *J Glob Health*. 2021;11:04009. Global prevalence of persistent adult ADHD estimated at 2.58% and symptomatic adult ADHD at 6.76% — roughly 140–366 million adults worldwide. <https://doi.org/10.7189/jogh.11.04009>
3. Anbarasan D, Kitchin M, Adler LA. Screening for Adult ADHD. *Curr Psychiatry Rep*. 2020;22(12):72. Adult ADHD is characterized by executive-functioning deficits not explicitly captured in DSM-5 criteria; the condition carries substantial personal, familial, and societal cost. <https://doi.org/10.1007/s11920-020-01194-9>

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Clinical Outcomes White Paper — No. 2 of 5

Why ADHD Is a Systems Failure, Not a Character Flaw

The attention-dopamine circuit, and the case for brain-based correction over willpower

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to the Brain Reset Protocol Outcomes Series

-35%

Dopamine-Linked Symptoms

p=0.0003

vs. Non-Program

30+ yrs

Elite & Executive Practice

Why ADHD Is a Systems Failure, Not a Character Flaw

The attention-dopamine circuit, and the case for brain-based correction over willpower.

It is not that you lack discipline. It is that your system is under load. Every adult who has ever been told to "just focus," "just be consistent," or "just build better habits" has been handed a character diagnosis for a neurological condition. This paper makes the case — mechanistically and with outcomes data — for why that framing fails, and what correcting the system actually requires.

Section 01

The Character Diagnosis

Late deadlines. Half-finished projects. A calendar that looks organized and a mind that does not feel it. For most adults who live this way, the explanation on offer is moral: not disciplined enough, not motivated enough, not trying hard enough. It is the most common misdiagnosis in high-performance culture, and it is wrong in a specific, correctable way.

What you are feeling is the output, not the cause. Inconsistent focus, task paralysis, emotional reactivity, and the "I know exactly what to do and still don't do it" experience are downstream symptoms of a dysregulated attention-and-executive-function system. They are not evidence of weak character. They are evidence of a system that has not been corrected.

Section 02

Why the Willpower Model Fails

Willpower-based interventions — productivity systems, accountability apps, "just do it" coaching — share one assumption: that the executive-function system is intact, and the problem is application. For a system that is genuinely under neurological load, this assumption is backwards. It asks a compromised system to override itself through more effort, which is precisely the resource that system struggles to deploy consistently. This is the gap no one is addressing: intervention aimed at behavior, applied to a problem that originates below behavior.

Section 03

The Attention-Dopamine Circuit

The prefrontal cortex is the brain's executive-function center — the seat of planning, inhibition, working memory, and self-monitoring. It runs on a specific neurochemical supply: primarily dopamine and norepinephrine, with acetylcholine and serotonin in supporting roles [1]. When

dopaminergic signaling to the prefrontal cortex is insufficient or dysregulated, the executive functions it powers — sustained attention, task initiation, impulse control — degrade, regardless of intelligence, ambition, or intent.

This is why attention and follow-through problems in adults are so often accompanied by a specific, recognizable cluster: a "good attention span" that comes and goes; starting tasks easily and finishing them rarely; impulses that create consequences the person did not intend; a private sense of being smarter than their output shows. That cluster is not a personality. It is a circuit.

This is where it changes: the moment attention stops being treated as a virtue you either have or don't, and starts being treated as a circuit you can measure and correct.

Adult ADHD is frequently under-diagnosed precisely because its defining impairment — executive dysfunction — is not fully captured by symptom checklists built around childhood hyperactivity [2]. Clinical prevalence estimates for adult ADHD run from roughly 4% to nearly 7% of the general population depending on methodology [2,3] — which means the conference room, the leadership team, and the client roster are statistically guaranteed to contain more of this circuit pattern than anyone has named out loud.

Section 04

Thirty Years at the Systems Level

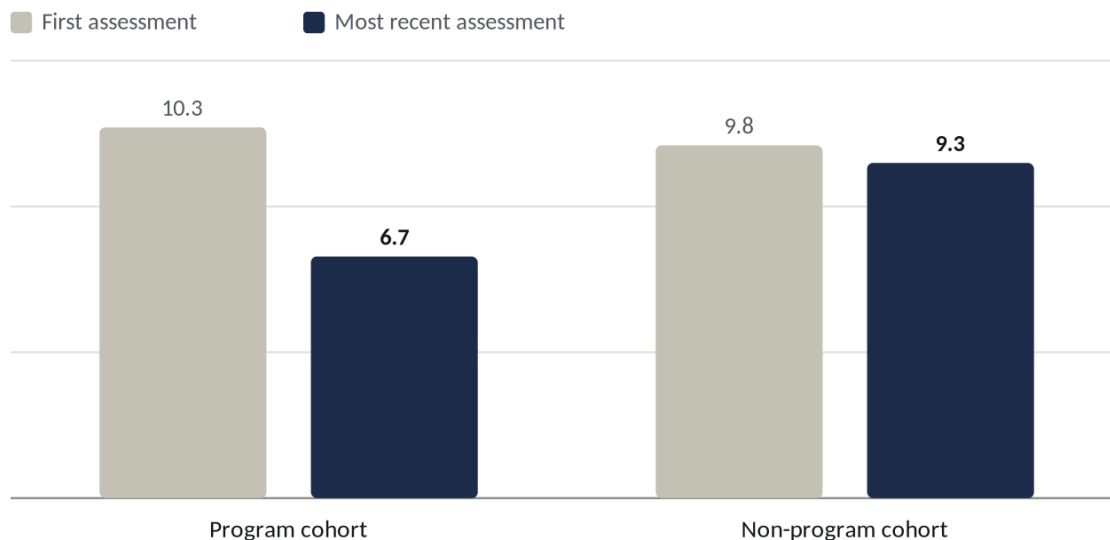
Fascia Training Institute was not built around symptom management. Across three decades working with elite athletes — NFL, NHL, and Olympic-level competitors — and, more recently, high-performing entrepreneurs and executives, the same pattern repeated: the people with the most willpower in the room were often the ones burning out fastest, because willpower was being asked to compensate for a system that needed correction, not motivation. Dynamic Brain Healing™ and Quantum NeuroFascial Release™ were built from that repeated observation — that performance breakdown happens at the neurological level first, and that you do not force change into a dysregulated system. You create the conditions for it.

Section 05

What Correction Looks Like, Measured

The Brain Reset Protocol is built to correct the system rather than manage its symptoms. Across 58 adults who enrolled in the protocol and retested their Brain Health Assessment months later, the Dopamine-linked symptom cluster — attention, drive, follow-through, impulse control — improved by 34.8% on average, a change $p=0.0003$ more significant than what a same-population comparison group of 31 non-enrolled retesters showed on their own.

Dopamine-Linked Symptom Score — First vs. Most Recent Assessment



Lower score = fewer attention/drive/impulse-control symptoms. Program cohort n=58; non-program cohort n=31.

A parallel, item-level analysis built specifically around attention span, task completion, working memory, and impulsivity — the hallmark presentation of ADHD-pattern executive dysfunction — showed the same pattern: program-enrolled clients improved 24.9% on this index ($p < .0001$), and the improvement was significantly larger than the comparison group's ($p = 0.0297$). White Paper 1 in this series carries the full outcomes analysis; White Paper 3 applies this same circuit to the specific demands of business and executive performance.

System-Level Insight

You do not force change. You create the conditions for change. Willpower interventions try to override a struggling circuit through more effort. System correction changes the neurological conditions the circuit is operating under — nutrition, stress load, movement, sleep architecture, and targeted regulation work — so the circuit itself functions differently. That distinction is the entire difference between a program that produces a testimonial and a program that produces a number.

The Position

Execution failure is a systems issue, not a discipline issue. When the system changes, everything downstream of it changes with it — focus, follow-through, mood, sleep, and the quiet, corrosive sense of underperforming your own intelligence. That is not motivation. That is neurology, corrected.

References

1. Papazian O, Alfonso I, Luzondo RJ. Executive function disorders. *Rev Neurol*. 2006;42(Suppl 3):S45-50. Anatomical substrate of executive function is the prefrontal cortex; dopamine and norepinephrine (and, to a lesser degree, acetylcholine and serotonin) are the neurotransmitters activating prefrontal neurons. Executive-function disorders are documented in ADHD. <https://pubmed.ncbi.nlm.nih.gov/16642451/>

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Clinical Outcomes White Paper — No. 3 of 5

The Business ADHD Brain

Executive function, decision fatigue, and performance recovery in high-achieving founders

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to the Brain Reset Protocol Outcomes Series

-25%

Attention/EF Index

p=0.0297

vs. Non-Program

77.6%

Improved on Program

The Business ADHD Brain

Executive function, decision fatigue, and performance recovery in high-achieving founders and executives.

The highest-performing person in the room is often running the least regulated system in the room. Founders and executives with an ADHD-pattern brain do not look impaired — they look relentless, until the cost of running on emergency fuel catches up with them. This paper applies the Brain Reset Protocol's outcomes data to that specific population: capable, high-output, and quietly inconsistent in a way no productivity system has fixed.

Section 01

Capable but Inconsistent

Founders and senior operators with an ADHD-pattern brain tend to share a specific signature: exceptional performance under acute pressure (the pitch, the launch, the deadline), and a disproportionate struggle with the unglamorous middle — the follow-up email, the admin, the maintenance work that has no deadline attached to it. From the outside this reads as inconsistency, or a discipline problem. It is neither. It is a dopamine-driven reward system that responds to urgency and novelty, and goes quiet without them.

The cost compounds at the executive level in ways it does not for an entry-level employee: a founder's inconsistency becomes the team's roadmap uncertainty, the board's confidence question, the household's financial variable. Executive-function deficits in adults are frequently missed by standard screening because high intelligence and strong compensatory habits mask them for years — often until the cognitive load of running something increases past the point those habits can cover [1].

Section 02

Decision Fatigue Is Not a Time-Management Problem

Every decision a founder makes draws on the same prefrontal resource: working memory, inhibition, and sustained attention, running in parallel, all day. In a well-regulated system, that resource replenishes. In a dysregulated attention-executive system, it does not — which is why the same founder who closes a critical negotiation at 10am can be reduced to indecision over a routine hire by 4pm. That is not a time-management failure. It is a systems failure occurring in real time, and it is measurable.

What you're feeling by 4pm is the output, not the cause. The cause is a system that was never given the conditions to regulate.

Section 03

What the Data Shows in This Population

Fascia Training Institute's Brain Health Assessment data does not tag clients by occupation, so the figures below reflect the full retested client cohort rather than a founder-only subset — but the underlying circuit is the same one driving business performance: the attention-and-executive-function pattern captured by items on sustained attention, task completion, working memory under pressure, and impulsivity.

25%

Reduction in attention/EF index

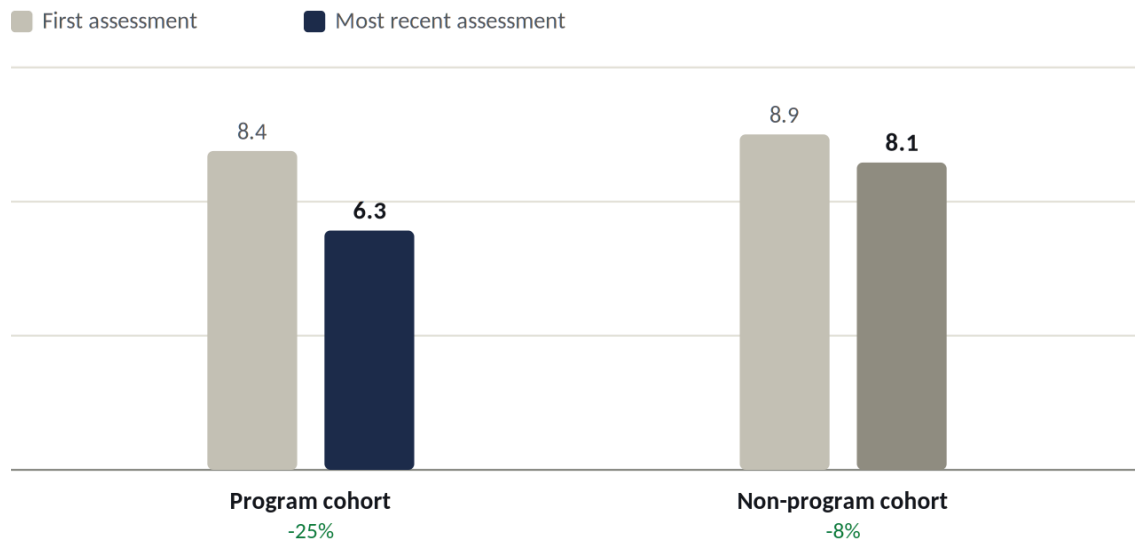
77.6%

of program clients improved

p=0.0297

vs. non-program comparison

Attention & Executive Function Index (0–15, higher = more symptoms)



Custom index built from 15 Brain Health Assessment items on attention span, task completion, working memory, and impulsivity. Program cohort n=58; non-program n=31.

Clients who enrolled moved from an average of 8.43 to 6.33 on this index — clients who did not enroll moved only from 8.87 to 8.13. The Dopamine-linked subscale specifically, the system most tied to drive and follow-through, improved 34.8% in the program cohort (p=0.0003 vs. non-program).

Case ACh-1 — Total Symptom Trajectory



6 assessments over 375 days. 46 → 22 (-52%).

Section 04

What Correction Requires at the Executive Level

You cannot coach a dysregulated dopamine system into consistency with more accountability calls. Founders have usually already tried that — a coach, an assistant, a system, a app — and arrived here anyway. The Brain Reset Protocol works at the level beneath behavior: nutrition, stress-load modulation, movement, sleep architecture, and targeted neurofascial regulation, engineered to change what the prefrontal circuit has to work with, rather than asking the same under-resourced circuit to try harder.

This is the gap no one is addressing in executive performance work: coaching optimizes behavior on top of a system. It does not correct the system underneath it. Founders who have plateaued despite excellent coaching are, more often than not, plateaued at the systems level — not the behavioral one.

The Position

A founder who is capable but inconsistent does not need another framework. They need the neurological conditions under their own performance corrected. When the system changes, the inconsistency changes with it — not because discipline improved, but because the circuit producing the inconsistency no longer has to run on emergency fuel.

References

1. Anbarasan D, Kitchin M, Adler LA. Screening for Adult ADHD. *Curr Psychiatry Rep.* 2020;22(12):72. Adult ADHD is characterized by executive-functioning deficits not explicitly captured in DSM-5 criteria; the condition carries substantial personal, familial, and societal cost. <https://doi.org/10.1007/s11920-020-01194-9>
2. Song P, Zha M, Yang Q, Zhang Y, Li X, Rudan I. The prevalence of adult attention-deficit hyperactivity disorder: A global systematic review and meta-analysis. *J Glob Health.* 2021;11:04009. Global prevalence of persistent adult ADHD estimated at 2.58% and symptomatic adult ADHD at 6.76% — roughly 140–366 million adults worldwide. <https://doi.org/10.7189/jogh.11.04009>

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Clinical Outcomes White Paper — No. 4 of 5

Anxiety Is Not a Personality Trait

GABA regulation, the ADHD-anxiety overlap, and what the data actually shows

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to the Brain Reset Protocol Outcomes Series

-27%

GABA System, Program

p=0.0007

vs. Non-Program

82.8%

Improved on Program

Anxiety Is Not a Personality Trait

GABA regulation, the ADHD-anxiety overlap, and what the Brain Reset Protocol data actually shows.

"I'm just a worrier" is not a personality trait. It is a description of an under-regulated inhibitory system, reported in the first person. GABA is the brain's primary braking system — the neurochemical signal that tells an overactive circuit to stand down. When that signal is weak, the brain does not experience calm as a resting state. It experiences everything as a threat that has not yet been ruled out.

Section 01

The System Underneath "I'm Just Anxious"

Chronic anxiety, a racing mind at bedtime, a body that stays braced even when nothing is wrong — these are usually described as temperament. They are more accurately described as a regulation deficit. GABA(A) receptors provide both moment-to-moment inhibition and a constant background "tonic" inhibition that keeps neural networks from running hot by default [1]. Chronic stress measurably degrades that background inhibition, which is why sustained pressure — a launch, a diagnosis, a difficult year — can leave someone permanently keyed up long after the acute event has passed [2].

This matters directly for the ADHD-pattern population this white paper series focuses on: attention dysregulation and anxiety are frequent co-travelers, not coincidences. A dopamine-driven attention system running without adequate inhibitory backup does not just lose focus — it runs anxious while doing it.

Section 02

What the Data Shows — and Where It Is Honestly Limited

The GABA system is one of the four Brain Health Assessment subscales tracked across every retested client. Program-enrolled clients improved 26.6% on this system, against 2.4% for clients who retested without enrolling — a gap that is statistically significant ($p=0.0007$).

-27%

GABA system, program cohort

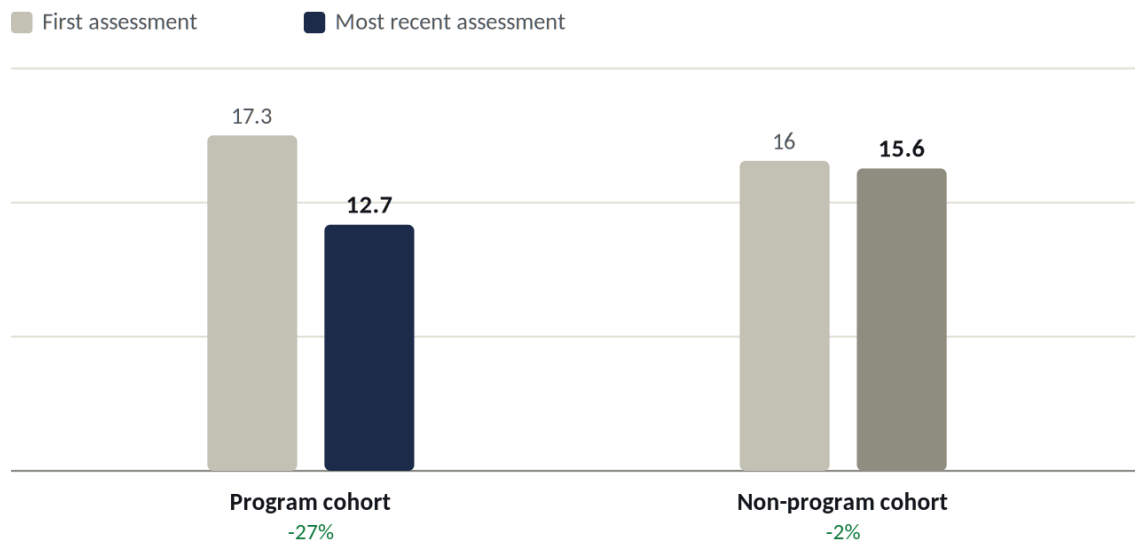
82.8%

of program clients improved

$p=0.0007$

vs. non-program comparison

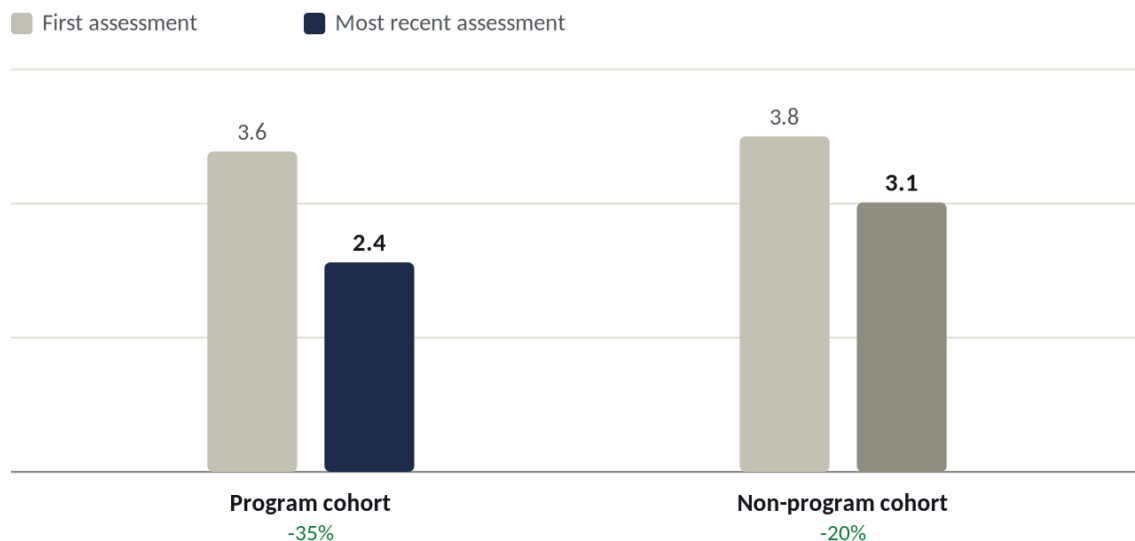
GABA (Regulation) System — First vs. Most Recent Assessment



Program cohort n=58; non-program cohort n=31. Between-group difference: $p=0.0007$.

We also built a narrower, exploratory index from the 10 Brain Health Assessment items most specific to anxious symptoms — chronic anxiety, nervousness, trembling, racing mood. On this narrower cut, program clients improved substantially and significantly on their own (34.6% reduction, $p<.0001$) — but the difference against the non-program comparison group did not reach statistical significance ($p=0.3483$), because both groups showed some improvement over time on this smaller, noisier slice of items.

Anxiety-Pattern Item Index (0–10, higher = more symptoms) — Exploratory



Program cohort n=58; non-program cohort n=31. Between-group difference not statistically significant ($p=0.3483$) — reported for transparency.

Read This Number Correctly

We are not going to round this corner. On the broader, official GABA subscale, the Brain Reset Protocol shows a real and statistically significant advantage over the non-program comparison group. On the narrower anxiety-specific item cut, the program cohort improved significantly on its own, but we cannot currently claim, from this dataset, that the program outperformed the comparison group on anxiety symptoms specifically — the sample is smaller and the signal noisier at that resolution. Both statements are true. We are reporting both, because a white paper that only shows you the number that flatters the program is marketing, not evidence.

Section 03

Why Regulation Work Outperforms Reassurance

Most approaches to chronic anxiety operate at the level of thought: reframe the worry, challenge the catastrophizing, talk it through. Useful, and incomplete — because if the underlying inhibitory system is still under-resourced, the brain will generate a new anxious thought the moment the old one is resolved. The Brain Reset Protocol works underneath the thought: nutrition, stress-load modulation, movement, sleep architecture, and targeted neurofascial regulation designed to restore the tonic inhibition a chronically stressed system has lost. When the braking system itself is restored, there are fewer thoughts left that need to be managed in the first place.

The Position

Anxiety is not who you are. It is what an under-regulated system produces under load. The GABA data here shows that load is correctable — measurably, in a matter of months. What it does not show, and what we will not claim, is that every anxious symptom resolves faster with structured correction than with time. Some do. The system-level ones — the chronic, physiological, braced-for-no-reason ones — are where this protocol shows its clearest advantage.

References

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Clinical Outcomes White Paper — No. 5 of 5

The Sleep-Attention Circuit

How sleep disruption fuels the ADHD pattern, and what moves when the system is corrected

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to the Brain Reset Protocol Outcomes Series

-21%

Sleep Index, Program

p=0.0459

vs. Non-Program

43.1%

Improved on Program

The Sleep-Attention Circuit

How sleep disruption fuels the ADHD pattern — and what moves when the system is corrected.

An unrested ADHD-pattern brain is not a milder version of a rested one. It is a materially worse one. Sleep and attention run on overlapping neurochemistry — dopamine and GABA both — which means a system that is already dysregulated during the day tends to sleep worse at night, and a night of poor sleep tends to make the next day's dysregulation worse. This is not two problems. It is one circuit, seen at two different hours.

Section 01

The Loop Nobody Names

Ask an ADHD-pattern adult about their sleep and the story is usually familiar: a mind that will not downshift at bedtime, a body that wakes at 2am already thinking, mornings that never feel like they delivered real rest. Sleep and ADHD maintain a documented bidirectional relationship: disrupted sleep worsens attention and emotional regulation the next day, and the same circuitry driving daytime attention difficulty also disrupts the ability to fall and stay asleep at night [1]. Reviews of the shared circuitry point to dopamine and GABA dysregulation, within the same cortico-striatal-thalamic pathways implicated in ADHD, as a common mechanism behind both [2].

Which means: you cannot fully correct the daytime pattern without addressing the nighttime one. A protocol that only targets focus during waking hours is treating half the circuit.

Section 02

What the Data Shows

Program-enrolled clients improved 21.2% on a Brain Health Assessment index built from 7 sleep-specific items — insomnia, waking repeatedly through the night, difficulty falling back asleep, early waking — a change that was statistically significant on its own ($p=0.0009$) and significantly larger than the comparison group's minimal movement ($p=0.0459$).

-21%

Sleep-pattern index, program

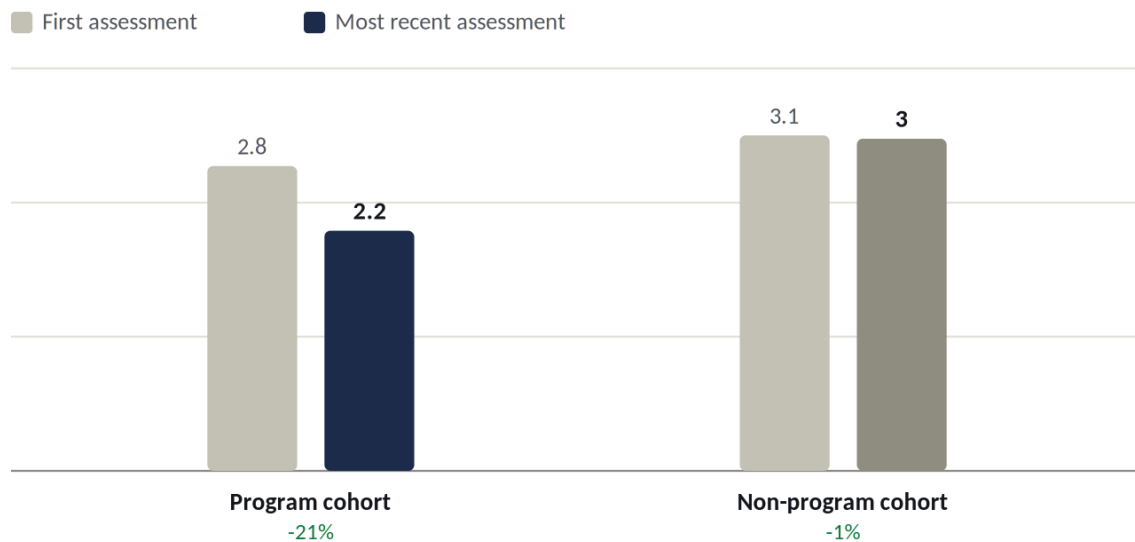
43.1%

of program clients improved

$p=0.0459$

vs. non-program comparison

Sleep-Pattern Item Index (0–7, higher = more symptoms)



Program cohort n=58; non-program cohort n=31. Between-group difference: $p=0.0459$.

Read This Number Correctly

The sleep-specific effect is real and statistically significant, but it is the most modest of the improvements reported across this white paper series — a smaller item count than the Dopamine or GABA subscales, and a between-group result that clears significance without much room to spare ($p=0.0459$). Sleep architecture tends to improve as a downstream consequence of broader system regulation, which is consistent with what the shared-circuitry research would predict [2] — but it is not, on this dataset, the single fastest-moving marker in the protocol.

Section 03

Why "Just Get More Sleep" Fails the Same Way "Just Focus" Does

Sleep advice aimed at behavior — a consistent bedtime, less screen time, a cooler room — is not wrong, but it addresses the environment around the circuit, not the circuit itself. For a system whose dopamine and GABA signaling is already under load during the day, that same dysregulation does not switch off at 10pm because the lights did. This is the same pattern-interrupt as the rest of this series: the intervention has to reach the neurochemistry, not just the schedule around it.

What you're calling insomnia is very often the nighttime shift of the exact circuit that ran your attention all day. It is not a separate problem. It is the same system, unregulated at a different hour.

Section 04

Closing the Loop

The Brain Reset Protocol treats sleep architecture as one output of system regulation rather than a separate problem to solve in isolation — the same nutritional, stress-load, movement, and neurofascial regulation work driving the Dopamine (34.8% improvement, $p=0.0003$) and GABA (26.6%

improvement, $p=0.0007$) findings reported elsewhere in this series is the same work moving the sleep numbers above. Correct the circuit, and both ends of the day move together.

The Position

You do not have a sleep problem and an attention problem. You have one dysregulated circuit that shows up twice a day. Correct the system, and it stops showing up at either hour the way it used to.

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Clinical Outcomes White Paper — Series II, No. 1 of 3

The Signal Everyone Missed

A full statistical re-ranking of the Brain Reset Protocol dataset — and the finding that outranks the one we led with

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Brain Health Assessment Outcomes Dataset, Series II

d=1.14

Acetylcholine — Largest Effect

p<.0001

vs. Non-Program

#1 of 10

Ranked Metrics

The Signal Everyone Missed

A full re-ranking of the Brain Reset Protocol dataset by statistical strength — and the finding that outranks the one we led with.

Every white paper we have published on this dataset so far has led with attention. That was not wrong — Dopamine moved substantially, and attention is what most clients say brought them in the door. But it was incomplete. When every tracked system in this dataset is ranked the same way a statistician would rank it — by effect size and significance, not by which story is easiest to tell — one system outranks all of them, including the aggregate score. This paper reports that finding directly, without softening it to fit the narrative we had already built.

Section 01

How "What Improved the Most" Should Actually Be Measured

Percent change alone is a weak way to rank clinical improvement — it is sensitive to the scale a subsystem happens to be measured on and can make a noisy, small-sample result look dramatic. The more defensible standard, and the one used throughout the peer-reviewed literature, combines three things: the between-group effect size (Cohen's d , comparing the program cohort's change to the non-program cohort's change), the statistical significance of that between-group difference, and the consistency of the effect (the percentage of clients who moved in the improved direction). Ranking the full Brain Health Assessment dataset — four neurotransmitter systems, the aggregate score, severe- and moderate-range item counts, and three custom symptom-pattern indices — by all three criteria at once produces a single, unambiguous answer.

Section 02

The Full Ranking

Rank	Metric	% Change	Effect Size ($ d $)	Significance	% Improved
1	Acetylcholine	-30.8%	1.14	p<.0001	87.9%
2	Total Symptom Score	-30.7%	1.03	p<.0001	93.1%
3	Dopamine	-34.8%	0.9	p=0.000250	84.5%
4	GABA	-26.6%	0.84	p=0.000675	82.8%
5	Serotonin	-31.4%	0.65	p=0.0068	82.8%
6	Severe-range items	-64.7%	0.64	p=0.0074	48.3%
7	ADHD-Pattern Index	-24.9%	0.51	p=0.0297	77.6%
8	Sleep-Pattern Index	-21.2%	0.45	p=0.0459	43.1%

9	Moderate-range items	-8.6%	0.24	p=0.2693	41.4%
10	Anxiety-Pattern Index	-34.6%	0.22	p=0.3483	65.5%

Ranked by between-group Cohen's *d* (program cohort change vs. non-program cohort change), largest effect first. n=58 program, n=31 non-program.

-31%

Acetylcholine, program cohort

1.14

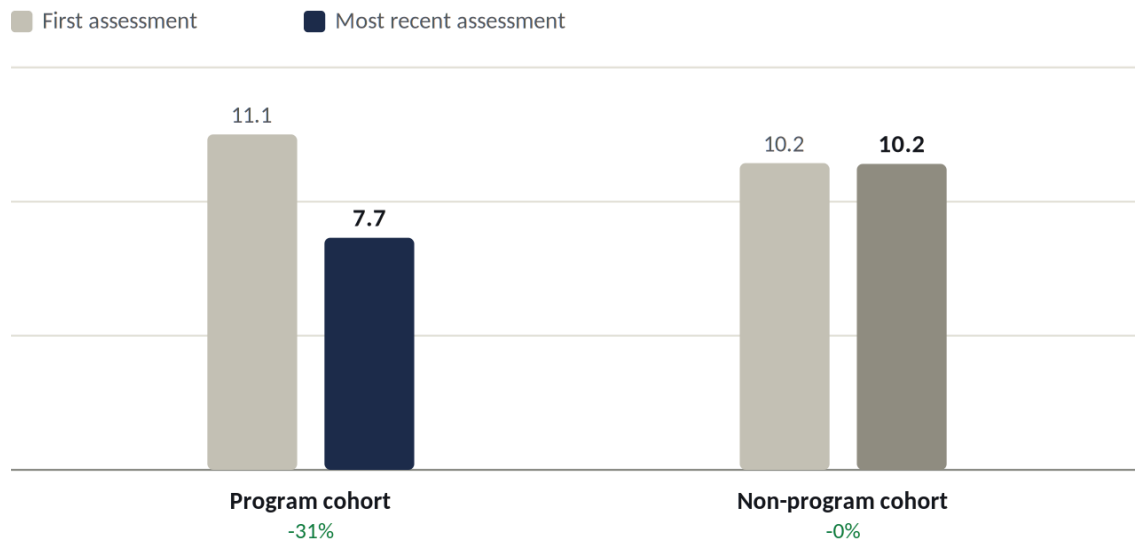
Cohen's *d* — the largest in the dataset

p<.0001

vs. non-program — the strongest signal

Acetylcholine — the system governing memory, mental clarity, word-retrieval, and learning speed — shows the largest between-group effect size and the smallest p-value of any metric in the entire Brain Health Assessment, including the composite Total Symptom Score itself. Program clients' Acetylcholine scores fell from an average of 11.14 to 7.71 (30.8% reduction, 87.9% of clients improved), while the non-program comparison group moved from 10.19 to 10.16 — functionally flat (p=0.9527).

Acetylcholine — Program Cohort vs. Non-Program Cohort



Between-group difference: p<.0001, Cohen's *d*=1.14.

Section 03

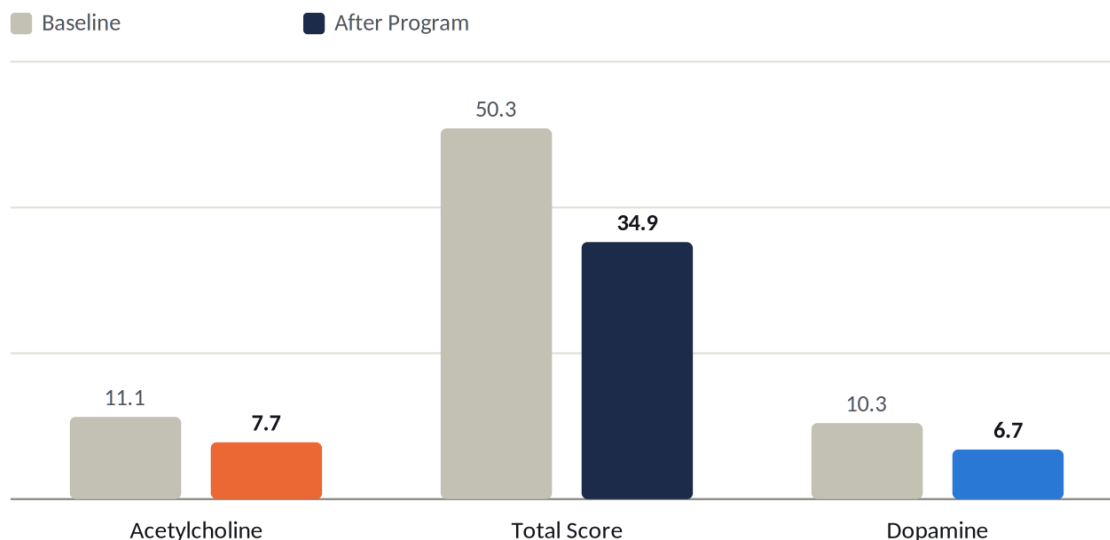
What Acetylcholine Actually Governs

Acetylcholine is not a background system. At the cortical level, it does two things simultaneously: it amplifies the brain's response to whatever is directly in front of it — a conversation, a task, a page of text — while suppressing the pull of background noise and prior, already-learned patterns. That combination is what encodes new memory cleanly and retrieves an existing one on demand [1].

Reduced function in this system's primary receptor pathway is specifically associated with a cluster of conditions that includes attention-deficit hyperactivity disorder [2].

Put in the language clients actually use: this is the system behind "the word is right there and I can't get to it," walking into a room and losing the reason you walked in, re-reading the same paragraph three times. Those are not attention failures in the classic hyperactive-distractible sense. They are retrieval and encoding failures — a different circuit, doing a different job, and, in this dataset, the one that moved the most.

Acetylcholine vs. Total Score vs. Dopamine — Program Cohort, Baseline to Latest



Acetylcholine's between-group effect size ($d=1.14$) exceeds both the aggregate Total Symptom Score ($d=1.03$) and Dopamine ($d=0.9$).

Section 04

Why We Are Correcting Our Own Framing

The earlier papers in this series were not wrong to feature Dopamine and attention — that system improved substantially and significantly, and it is the presenting complaint for most clients who arrive at Fascia Training Institute describing themselves as distracted or inconsistent. But a program built on measurement does not get to stop measuring once the first result is flattering. Run the full ranking, and the strongest, most statistically defensible claim this dataset supports is not "this program helps you focus." It is: **this program restores cognitive clarity, and clarity is frequently the upstream condition attention problems were mistaken for.**

What This Ranking Does Not Prove

All ten metrics ranked here come from the same 118-item self-report instrument, completed by the same clients — they are correlated with each other, not ten independent experiments. Acetylcholine ranking first is a real, robust finding within this dataset; it is not evidence that acetylcholine "matters more" than the other three systems in any absolute physiological sense, or that every client's primary gain will be in this domain. It is the single most consistent and statistically strongest signal this specific cohort produced.

The Position

We built this program to fix attention. The data says it is doing something more foundational than that — and more foundational is not a smaller claim. It is a larger one, stated more precisely.

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1. Hasselmo ME, Giocomo LM. Cholinergic modulation of cortical function. *J Mol Neurosci*. 2006;30(1-2):133-5. Acetylcholine enhances the cortex's response to incoming sensory/task input while suppressing background and prior-learned activity — heightening attention to what is in front of you and enhancing encoding of new memory, while reducing interference from what you already know. <https://doi.org/10.1385/JMN:30:1:133>
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Mechanism & Authority White Paper — Series II, No. 2 of 3

Why Clarity Moves First

The neurological mechanism behind the strongest signal in the Brain Reset dataset

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to “The Signal Everyone Missed”

-31%

Acetylcholine, Program

$p < .0001$

vs. Non-Program

30+ yrs

Clinical & Elite-Performance Application

Why Clarity Moves First

The neurological mechanism behind the strongest signal in the Brain Reset dataset — and how it connects to the nervous-system regulation work underneath the protocol.

Brain fog is not a vague complaint. It is a specific, describable signal failure in a specific neurochemical system — and White Paper 6 in this series showed that system, Acetylcholine, is the one that moved the most under structured correction. This paper explains why. Not to romanticize a neurotransmitter, but because the mechanism matters: it is the difference between a program that happens to correlate with improvement and one built on a defensible model of why the improvement occurs.

Section 01

What Acetylcholine Does at the Synapse

Acetylcholine performs two functions in the cortex at the same time, and the combination is the point. It amplifies the brain's response to whatever input is directly in front of it — the sentence you are reading, the person you are talking to, the task on the desk — while simultaneously suppressing background cortical noise and interference from prior, already-consolidated learning [1]. High acetylcholine tone means new information gets encoded cleanly, with less competition from everything the brain already knows. Low tone means the opposite: input and memory blur together, retrieval slows, and the felt experience is exactly what clients describe as "fog" — not an inability to pay attention, but an inability to cleanly separate the present moment from everything else competing for the same circuit.

This is not a static trait. Acetylcholine release in the memory system tracks moment-to-moment engagement in real time — when engagement drops, cholinergic signaling drops with it, and the precision of the brain's working memory degrades measurably. Pharmacologically blocking acetylcholine receptors reproduces this same disengaged, imprecise state directly [2]. The system is dynamic, state-dependent, and — critically for this protocol — responsive to changes in the conditions surrounding it, not fixed.

Section 02

The Regulation Connection: Acetylcholine Is Not Only a Memory Chemical

Acetylcholine has a second job that has nothing to do with memory directly, and it is where this gets structurally interesting for a fascia- and nervous-system-based protocol. The primary efferent pathway of the vagus nerve — the body's main parasympathetic regulation channel — signals through acetylcholine. This is the cholinergic anti-inflammatory pathway: vagal acetylcholine release is the

mechanism by which the nervous system directly suppresses systemic inflammatory signaling in real time [3,4]. The same neurotransmitter this dataset tracks as a cognitive-clarity marker is also the chemical messenger of the body's primary calm-and-regulate circuit.

This is why nervous-system regulation work — the kind aimed at restoring parasympathetic tone through fascia, breath, and structured stress-load correction rather than through willpower or supplementation alone — is a plausible mechanism for why a cognitive system would respond this strongly to a program that was never marketed as a "memory protocol." A body under chronic sympathetic load is a body with suppressed vagal-cholinergic tone system-wide. Restore the regulation, and the same chemical messenger that calms inflammation downstream is available in greater supply upstream, in the cortex, doing its cognitive job.

Where the Evidence Ends and the Model Begins

Everything above this box is supported by published neuroscience: acetylcholine's cortical role in attention and memory encoding is well established [1,2], and its role as the vagus nerve's primary anti-inflammatory signal is well established separately [3,4]. What is *not* directly measured in this dataset is vagal tone, inflammatory markers, or a biological pathway linking the two in these specific clients. The connection offered here is a physiologically grounded, plausible mechanism — the most defensible explanation we currently have for why a nervous-system regulation protocol would produce its strongest cognitive effect in exactly this system. It is a hypothesis built on real science, not a claim this dataset proves directly.

Section 03

What Thirty Years With High Performers Confirms

Long before this dataset existed, the pattern was visible on the field and in the boardroom. Elite athletes describe their worst performances not as a failure of effort but as a loss of clarity — the game "speeding up," a step behind, unable to read what used to be obvious. Executives describe the same thing in different language: "I used to be sharp and now I'm guessing." In both populations, the complaint that arrives first is usually framed as attention or focus. The complaint underneath it, almost every time, is a loss of clean signal — clarity, not attention, is the higher lever. This dataset is the first time that observation has had a number attached to it.

Case ACh-1 — Trajectory, Total Symptom Score



6 assessments over 375 days. De-identified, drawn from the program cohort.

Section 04

The Practical Implication

If clarity is the upstream lever, then a protocol aimed only at attention symptoms is treating the downstream output. The Brain Reset Protocol's nutrition, stress-load, and neurofascial regulation components were never built exclusively around dopamine or classic attention training — they were built around restoring system-wide regulation. This dataset is the first evidence that the strongest measurable return on that broader approach shows up exactly where the mechanism predicts it should: in the cholinergic system, not the dopaminergic one.

The Position

You do not force clarity by trying harder to focus. Clarity is what a regulated system produces on its own, once the load that was suppressing it is removed. Attention is the symptom people notice first. Clarity is the system this data says actually moved.

References

1. Hasselmo ME, Giocomo LM. Cholinergic modulation of cortical function. *J Mol Neurosci.* 2006;30(1-2):133-5. Acetylcholine enhances the cortex's response to incoming sensory/task input while suppressing background and prior-learned activity — heightening attention to what is in front of you and enhancing encoding of new memory, while reducing interference from what you already know. <https://doi.org/10.1385/JMN:30:1:133>
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Statistical Methods White Paper — Series II, No. 3 of 3

Does It Work Better If You're Worse Off?

An honest statistical look at baseline severity and improvement in the Brain Reset dataset

Simone Fortier, Founder — Fascia Training Institute

August 2026 • Companion to “The Signal Everyone Missed”

$r = -0.477$

Non-Program Correlation — Stronger

38%

Mild-Third Improvement, Program

+18%

Mild-Third Change, Non-Program

Does It Work Better If You're Worse Off?

An honest statistical look at baseline severity and improvement — including the confound most programs would not show you.

Here is the tempting story: clients who started in worse shape saw bigger point-drops in their Total Symptom Score. It would be easy to market that as "the more you're struggling, the more this helps you." A rigorous look at the data does not support that story cleanly — and the reason why is more useful than the story would have been.

Section 01

The Pattern That Looks Like a Finding

Split the program cohort into thirds by baseline severity and the raw numbers do show larger absolute point-drops in the more severe groups: the Severe third (baseline ≈ 70) improved by 16.7 points; the Mild third (baseline ≈ 33) improved by 12.6. Correlating baseline score against total change across the full program cohort confirms it statistically: $r = -0.298$, $p = 0.0231$, $n = 58$ — clients who started higher (more symptomatic) tended to drop more.

On its own, that correlation looks like a severity-response finding. It is not one — or at least, not only that. Before attributing it to the program, it has to survive one test: does the exact same pattern also show up in people who received no structured intervention at all?

Section 02

The Confound: Regression to the Mean

An extreme score on any noisy, repeated measurement — a mood scale, a blood pressure reading, a symptom inventory — tends to drift toward the average on a second measurement, for purely statistical reasons, with no intervention required. Someone who scores unusually high on a bad week is statistically likely to score somewhat lower next time regardless of what they do in between, simply because some of that first high score was noise. This is regression to the mean, and any dataset built on repeat self-report measurement has to check for it before claiming a severity-response effect is real.

We ran the identical correlation on the 31 clients who retested **without** enrolling in the Brain Reset Protocol: $r = -0.477$, $p = 0.0066$, $n = 31$. That correlation is not weaker than the program cohort's. It is stronger.

Read This Number Correctly

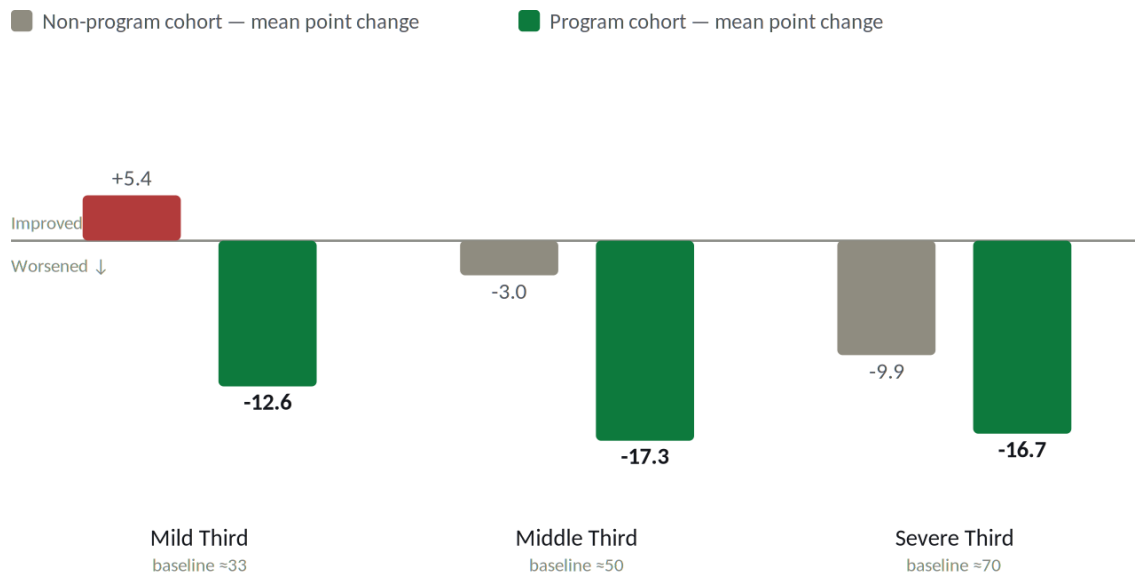
If "the program helps more severe clients more" were the true, program-specific effect, the baseline-severity-to-improvement correlation should appear only — or at least more strongly — in the enrolled group. It does not. It appears more strongly in the group that received no structured intervention at all. The most defensible statistical conclusion is that most of what looks like a severity-response pattern in this dataset is regression to the mean, not a program effect. We are reporting this plainly because a white paper that quietly keeps the flattering correlation and drops the inconvenient comparison group is marketing, not evidence.

Section 03

What Survives the Confound — and It Is the More Useful Finding

Regression to the mean explains why both groups show a baseline-severity correlation. It does not explain what happens when you compare the two groups directly, tercile by tercile, at matched levels of starting severity.

Mean Point Change by Baseline-Severity Third — Program vs. Non-Program



Total Symptom Score, mean change from first to most recent assessment, split into thirds by baseline severity within each cohort.

Severity Third	Baseline (avg)	Program n	Program Δ	Non-Prog n	Non-Prog Δ
Mild Third	32.7	n=21	-12.6 (-38.4%)	n=11	+5.4 (+18.2%)
Middle Third	49.8	n=18	-17.3 (-34.8%)	n=10	-3.0 (-6.0%)
Severe Third	70.2	n=19	-16.7 (-23.9%)	n=10	-9.9 (-15.1%)

Look at the Mild third specifically, because it is the most revealing cell in this table. Left untreated, the mildest non-program clients did not hold steady — their scores **worsened** on average (+5.4 points, +18.2%). The mildest program clients, over a comparable window, improved by -12.6 points (38.4%). That gap — not the severity correlation — is the part of this pattern that regression to the mean cannot

explain, because regression to the mean predicts the mildest untreated cases should drift *toward* worse, which is exactly what happened in the non-program group. Enrollment reversed that drift.

The same directional gap — program cohort improving more than the non-program cohort at the same starting severity — holds in all three thirds. Subgroup sizes here are small (n=10–21 per cell) and these are descriptive comparisons, not independently powered tests; we are not claiming statistical significance within each cell. What the pattern supports, consistently, is this: **severity does not reliably predict who benefits more from the program. Enrollment reliably outperforms non-enrollment, at every starting severity level.**

Section 04

Why This Version of the Finding Is More Valuable, Not Less

A program that only works for its most severe clients is a rescue service. A program that shows a real, consistent advantage over doing nothing — across mild, moderate, and severe presentations alike — is a system. The second claim is harder to make honestly, because it requires ruling out the more dramatic-sounding story first. It is also the claim that matters more to the audience this protocol is built for: high-functioning people who do not yet consider themselves "severe," who are exactly the population most likely to be told, correctly, that doing nothing carries its own risk of drift.

The Position

We are not going to claim this program works best on the people who are struggling most. The data does not clearly support that, and a program built on measurement does not get to keep a finding that does not survive the check. What the data does support, at every severity level tested: showing up and doing the work outperforms waiting to see what happens on your own.

References

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This white paper is provided for educational purposes and describes outcomes observed in Fascia Training Institute's Brain Health Assessment program dataset. It is not a peer-reviewed clinical trial, and the Brain Health Assessment is a self-report educational instrument, not a validated diagnostic tool for ADHD, anxiety, or any other medical or psychiatric condition. Program enrollment in this dataset was self-selected, not randomized, which may contribute to the differences reported between cohorts. Simone Fortier and Fascia Training Institute do not provide medical advice, diagnosis, or treatment; consult a qualified healthcare professional regarding any medical or psychiatric condition. Client data referenced throughout is aggregated and de-identified; case examples use anonymized codes, not real names.