SLEEP EXPERIMENT — 14 DAY TRACKER | 4 Hours SLEEP & RECOVERY 9 Hours SLEEP
Blood draws on Days 1,4, 7,11,14 (fasted, 7–8 AM) · Keep diet, exercise, caffeine constant throughout
PHASE 1: SLEEP RESTRICTION (Days 1–7) — 4 hrs/night | 2 AM → 6 AM
PHASE 2: RECOVERY SLEEP (Days 8–14) — 9 hrs/night | 10 PM → 7 AM

Patient ID: AS-28821	Patient Name: Abhijeet Satani	Age : 28	Start Date: 2026-03-1	End Date: 2026-03-14

Date	Phase / Sleep	Sleep Window	Blood Draw	Daily Tests	Key Watch-outs
PHASE 1: Day 1	BASELINE DRAW	2 AM → 6 AM (4h)	BLOOD DRAW #1 (Baseline)	Reaction time, HRV, Mood, Stroop test	No intense exercise day before
PHASE 1: Day 4	Phase 1: 4 hrs	2 AM → 6 AM (4h)	BLOOD DRAW #2 (MID - RESTRICTION)	Reaction time, HRV, Mood, Stroop test	Mid-point cognitive test
PHASE 1: Day 7	MID DRAW	2 AM → 6 AM (4h)	BLOOD DRAW #3 (END - RESTRICTION)	Reaction time, HRV, Mood, Stroop test	Same fasted protocol as Day 1
PHASE 2: Day 11	BASELINE DRAW	10 PM → 7 AM (9h)	BLOOD DRAW #4 (MID - RECOVERY)	Reaction time, HRV, Mood, Stroop test	No intense exercise day before
PHASE 2: Day 14	Phase 2: 9 hrs	10 PM → 7 AM (9h)	BLOOD DRAW #5 (FINAL)	Reaction time, HRV, Mood, Stroop test	END-point cognitive test

The Variables	The Hormonal & Stress Panel	Metabolic & Lipid Panel	Hepatic & Organ Stress	The Complete Blood Count	Performance & Biometrics
Day (1–14)	Testosterone (T.T) (ng/dL)	Fasting Glucose (mg/dL)	ALT (Liver Enzyme - U/L)	WBC (White Blood Cell Count) – (10^3/µL)	Reaction Time (ms)
Status (Baseline, Restriction, Recovery)	Cortisol (ug/dL)	LDL Cholesterol (mg/dL)	AST (Liver Enzyme - U/L)	RBC (Red Blood Cell Count) – (10^6/µL)	HRV (Heart Rate Variability - ms)
Sleep Hours (Actual hours recorded)	C-Reactive Protein - mg/L)	HDL Cholesterol (mg/dL)		Hgb (Hemoglobin) – (g/dL)	Grip Strength (kg)
		Triglycerides (mg/dL)		Platelets – (10^3/µL)	Mood Score (1-10)
				Neutrophils (%) First-line defenders	
				Lymphocytes (%) Adaptive immunity	

SLEEP EXPERIMENT — 14 DAY TRACKER | 4 Hours SLEEP & RECOVERY 9 Hours SLEEP
Blood draws on Days 1,4, 7,11,14 (fasted, 7–8 AM) · Keep diet, exercise, caffeine constant throughout
PHASE 1: SLEEP RESTRICTION (Days 1–7) — 4 hrs/night | 2 AM → 6 AM
PHASE 2: RECOVERY SLEEP (Days 8–14) — 9 hrs/night | 10 PM → 7 AM

Patient ID: AS-28821	Patient Name: Abhijeet Satani	Age : 28	Start Date: 2026-03-1	End Date: 2026-03-14

Category	Parameter	*Unit*	Day 1	Day 4	Day 7	Day 11	Day 14
General	Day	—	1	4	7	11	14
	Status	—	Baseline	Mid-Restriction	End-Restriction	Mid-Recovery	Final-Recovery
	Sleep Hours	*hours*	8	4	4	9	9
Hormones	Testosterone	*ng/dL*	610	565	495	550	595
	Cortisol	*µg/dL*	12	14.2	21.5	15.5	12.8
Inflammation	CRP	*mg/L*	0.5	0.9	1.8	0.8	0.6
Metabolic	Glucose	*mg/dL*	88	93	99	89	87
	LDL	*mg/dL*	95	102	115	105	98
	HDL	*mg/dL*	55	53	48	52	54
	Triglycerides	*mg/dL*	90	110	145	105	95
Liver Function	ALT	*U/L*	22	25	31	26	23
	AST	*U/L*	19	21	26	22	20
Hematology (CBC)	WBC	*10^3/µL*	6.5	7.1	7.8	7	6.7
	RBC	*10^6/µL*	5.1	5.15	5.22	5.15	5.12
	Hemoglobin (Hgb)	*g/dL*	15.1	15.3	15.6	15.4	15.2
	Platelets	*10^3/µL*	230	245	275	250	235
	Neutrophils	*%*	54	59	66	60	56
	Lymphocytes	*%*	34	30	24	29	33
Performance / Neuro	Reaction Time	*ms*	270	312	338	315	292
	HRV	*ms*	50	42	34	44	47
	Grip Strength	*kg*	52	50	46	49	51
	Mood Score	*scale*	8.5	5.5	3.5	7	9

Baseline Health Panel

STATUS: BASELINE

Patient Name:	Abhijeet Satani	**Test Date:**	2026-03-1	**Experiment Day:**	01
Patient ID:	AS-28821	**BMI:**	22 - 24	**Sleep Protocol:**	8 Hours
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

HEPATOCYTE & HEMATOLOGY

WBC Count	6.50	10e3/uL
RBC Count	5.10	10e6/uL
Hemoglobin	15.1	g/dL
Platelets	230	10e3/uL
ALT / AST	22 / 19	U/L

ENDOCRINE & METABOLIC

Testosterone	610	ng/dL
Cortisol	12.0	ug/dL
Glucose	88	mg/dL
CRP	0.5	mg/L
Triglycerides	90	mg/dL

PERFORMANCE & PHYSIO

Mood Score	8.5	/ 10
HRV	50	ms
Reaction Time	270	ms
Grip Strength	52	kg
Lymphocytes	34	%

Clinical Note: Measurements recorded under standard conditions (Fasted). Current data point: Day 01 (Baseline).

Mid-Restriction Health Panel

STATUS: MID-RESTRICTION

Patient Name:	Abhijeet Satani	**Test Date:**	2026-03-4	**Experiment Day:**	04
Patient ID:	AS-28821	**BMI:**	22 - 24	**Sleep Protocol:**	4 Hours
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

HEPATOCYTE & HEMATOLOGY

WBC Count	7.10	10e3/uL
RBC Count	5.15	10e6/uL
Hemoglobin	15.3	g/dL
Platelets	245	10e3/uL
ALT / AST	25 / 21	U/L

ENDOCRINE & METABOLIC

Testosterone	565	ng/dL
Cortisol	14.2	ug/dL
Glucose	93	mg/dL
CRP	0.9	mg/L
Triglycerides	110	mg/dL

PERFORMANCE & PHYSIO

Mood Score	5.5	/ 10
HRV	42	ms
Reaction Time	312	ms
Grip Strength	50	kg
Lymphocytes	30	%

Clinical Note: Measurements recorded under standard conditions (Fasted). Current data point: Day 04 (Mid-Restriction).

End-Restriction Health Panel

STATUS: END-RESTRICTION

Patient Name:	Abhijeet Satani	**Test Date:**	2026-03-7	**Experiment Day:**	07
Patient ID:	AS-28821	**BMI:**	22 - 24	**Sleep Protocol:**	4 Hours
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

HEPATOCYTE & HEMATOLOGY

WBC Count	7.80	10e3/uL
RBC Count	5.22	10e6/uL
Hemoglobin	15.6	g/dL
Platelets	275	10e3/uL
ALT / AST	31 / 26	U/L

ENDOCRINE & METABOLIC

Testosterone	495	ng/dL
Cortisol	21.5	ug/dL
Glucose	99	mg/dL
CRP	1.8	mg/L
Triglycerides	145	mg/dL

PERFORMANCE & PHYSIO

Mood Score	3.5	/ 10
HRV	34	ms
Reaction Time	338	ms
Grip Strength	46	kg
Lymphocytes	24	%

Clinical Note: Measurements recorded under standard conditions (Fasted). Current data point: Day 07 (End-Restriction).

Mid-Recovery Health Panel

STATUS: MID-RECOVERY

Patient Name:	Abhijeet Satani	**Test Date:**	2026-03-11	**Experiment Day:**	11
Patient ID:	AS-28821	**BMI:**	22 - 24	**Sleep Protocol:**	9 Hours
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

HEPATOCYTE & HEMATOLOGY

WBC Count	7.00	10e3/uL
RBC Count	5.15	10e6/uL
Hemoglobin	15.4	g/dL
Platelets	250	10e3/uL
ALT / AST	26 / 22	U/L

ENDOCRINE & METABOLIC

Testosterone	550	ng/dL
Cortisol	15.5	ug/dL
Glucose	89	mg/dL
CRP	0.8	mg/L
Triglycerides	105	mg/dL

PERFORMANCE & PHYSIO

Mood Score	7.0	/ 10
HRV	44	ms
Reaction Time	315	ms
Grip Strength	49	kg
Lymphocytes	29	%

Clinical Note: Measurements recorded under standard conditions (Fasted). Current data point: Day 11 (Mid-Recovery).

Final-Recovery Health Panel

STATUS: FINAL-RECOVERY

Patient Name:	Abhijeet Satani	**Test Date:**	2026-03-14	**Experiment Day:**	14
Patient ID:	AS-28821	**BMI:**	22 - 24	**Sleep Protocol:**	9 Hours
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

HEPATOCYTE & HEMATOLOGY

WBC Count	6.70	10e3/uL
RBC Count	5.12	10e6/uL
Hemoglobin	15.2	g/dL
Platelets	235	10e3/uL
ALT / AST	23 / 20	U/L

ENDOCRINE & METABOLIC

Testosterone	595	ng/dL
Cortisol	12.8	ug/dL
Glucose	87	mg/dL
CRP	0.6	mg/L
Triglycerides	95	mg/dL

PERFORMANCE & PHYSIO

Mood Score	9.0	/ 10
HRV	47	ms
Reaction Time	292	ms
Grip Strength	51	kg
Lymphocytes	33	%

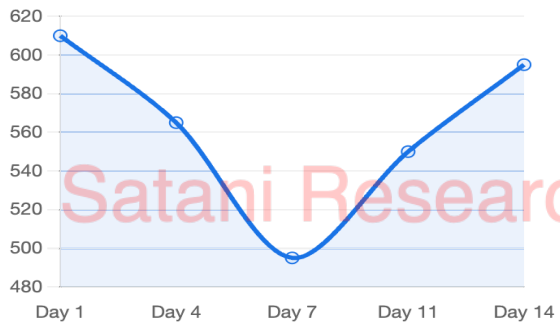
Clinical Note: Measurements recorded under standard conditions (Fasted). Current data point: Day 14 (Final-Recovery).

Comprehensive Marker Comparison

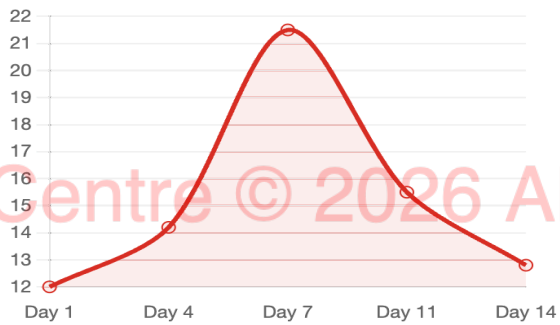
EXPERIMENT: 4H VS 9H SLEEP

Patient Name:	Abhijeet Satani	**Start Date:**	2026-03-1 : 2026-03-14	**Duration:**	14 Days
Patient ID:	AS-28821	**BMI:**	22 - 24	**Status:**	Final Report
Age:	28	**Gender:**	Male	**Clinic:**	Satani Research Centre

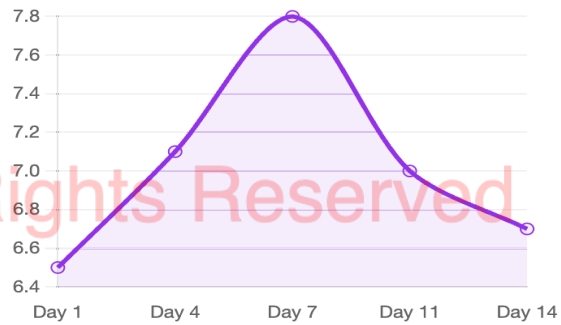
TESTOSTERONE (NG/DL)



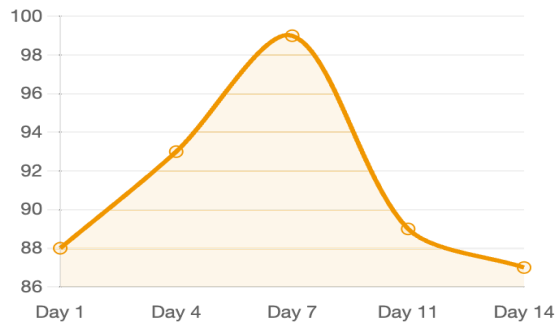
CORTISOL (UG/DL)



WBC (10E3/UL)

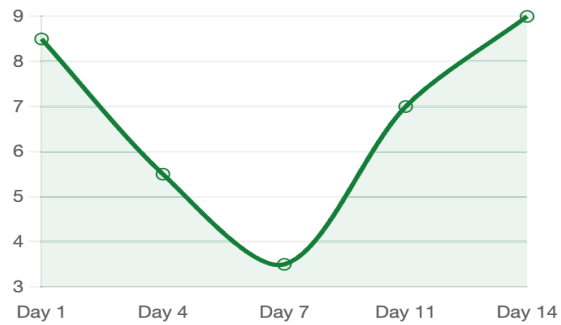


FASTING GLUCOSE (MG/DL)



REACTION TIME (MS)

MOOD SCORE (1-10)



CASE REPORT

Acute Sleep Restriction to Four Hours per Night for Seven Days Induces Measurable Hormonal, Metabolic, Immunological, and Neurocognitive Dysregulation in a Healthy Young Adult Male: A Prospective Self-Experiment with Serial Biomarker Monitoring and Subsequent Recovery Assessment

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Heth Joshi

ABSTRACT

Background: Insufficient sleep is a pervasive feature of modern life, yet the magnitude and pace of physiological dysregulation induced by acute restriction remain underappreciated at the individual level. This self-experiment sought to characterise the temporal trajectory of hormonal, metabolic, immunological, hepatic, haematological, and neurocognitive changes during seven days of four-hour sleep restriction, and to quantify the completeness of recovery following a subsequent seven-day period of nine-hour sleep.

Case presentation: A healthy 28-year-old male (BMI 22–24, non-smoker, no regular medications) underwent a controlled 14-day experiment comprising Phase 1: seven nights of restricted sleep (02:00–06:00, four hours), and Phase 2: seven nights of extended recovery sleep (22:00–07:00, nine hours). Five fasted blood draws were obtained at Days 1, 4, 7, 11, and 14. Serial assessments included serum testosterone, cortisol, C-reactive protein (CRP), fasting glucose, lipid profile, liver enzymes, complete blood count, reaction time, heart rate variability (HRV), grip strength, and mood scores.

Outcomes: By Day 7, serum testosterone had declined 18.9% from baseline (610 to 495 ng/dL), morning cortisol had risen 79.2% (12.0 to 21.5 µg/dL), CRP had increased 260% (0.5 to 1.8 mg/L), fasting glucose had risen to 99 mg/dL, LDL cholesterol to 115 mg/dL, triglycerides to 145 mg/dL, and WBC to 7.8 × 10^3/µL with a shift toward neutrophilia (66%) and relative lymphopaenia (24%). Reaction time slowed by 25.2% and HRV fell 32%. Mood score declined from 8.5 to 3.5/10. Following seven nights of nine-hour recovery sleep, the majority of biomarkers approached—but did not uniformly return to—Day 1 baseline, with neurocognitive recovery lagging behind haematological and metabolic restoration.

Conclusions: Seven consecutive nights of four-hour sleep produce clinically relevant, multi-system physiological derangement that partially persists beyond a matched recovery period. These findings reinforce the case for serial biomarker monitoring in individuals with sustained sleep restriction, and highlight the inadequacy of subjective assessment as the sole gauge of sleep-related physiological harm.

Keywords: sleep deprivation, sleep restriction, testosterone, cortisol, HRV, metabolic syndrome, immune function, cognitive performance, n-of-1 study, recovery sleep

BACKGROUND

Sleep is increasingly recognised as a fundamental regulator of endocrine homeostasis, immune surveillance, metabolic efficiency, and cognitive performance. Epidemiological data consistently associate habitual short sleep (defined as fewer than six hours per night) with elevated risks of obesity, type 2 diabetes mellitus, cardiovascular disease, immunosuppression, and all-cause mortality. Despite this, population surveys in high-income countries report that between 30–35% of working adults regularly sleep fewer than six hours per night, with a significant subset chronically restricted to four to five hours during peak professional or academic demand.

What distinguishes this case report from observational epidemiology is the prospective, controlled, within-subject design: a single healthy individual underwent standardised sleep restriction followed by standardised recovery sleep, with five blood draws and daily physiological metrics collected throughout. This n-of-1 approach sacrifices generalisability but gains mechanistic resolution that population-level studies cannot provide—specifically, the temporal kinetics of biomarker change and the completeness of recovery within a defined window.

The principal scientific questions addressed are: (1) Which biomarker systems show detectable change during acute restriction, and at what pace? (2) Does subjective wellbeing track objectively measurable deterioration? (3) How complete is physiological recovery after seven days of extended sleep, and which systems are slowest to normalise?

CASE PRESENTATION

Subject Characteristics

The subject is the study's principal investigator: a 28-year-old male (Patient ID: AS-28821) with a BMI in the range of 22–24 kg/m², a non-smoker, physically active (exercise frequency three to five sessions per week), following a consistent mixed diet, and consuming no regular prescription or over-the-counter medications. No alcohol was consumed during the 14-day experimental period. Caffeine intake was standardised throughout. There was no history of sleep disorder, endocrine dysfunction, or chronic illness. Baseline blood indices (Day 1) confirmed all parameters within age-appropriate reference ranges, providing a physiologically intact starting point.

Experimental Protocol

The experiment was conducted at Satani Research Centre between 1–14 March 2026. The design comprised two sequential phases without randomisation:

Phase 1 — Sleep Restriction (Days 1–7): Participants slept from 02:00 to 06:00 each night, yielding four hours of sleep opportunity per 24-hour period. Alarm-enforced waking at 06:00 was applied consistently.

Phase 2 — Recovery Sleep (Days 8–14): Following a single buffer night of approximately seven hours (Day 8), participants slept from 22:00 to 07:00 (nine hours) for six consecutive nights. Day 8 served as a washout transition and was not formally analysed.

Throughout both phases, diet, exercise, and caffeine intake were maintained constant. All blood draws were performed in the fasted state (minimum 8 hours), between 07:00 and 08:00, to control for circadian variation in hormone secretion.

Study Timeline

Day	Phase / Status	Sleep Window	Blood Draw	Daily Tests	Key Watch-outs
Day 1	Baseline	2 AM–6 AM (4h)	Blood Draw #1	Reaction time, HRV, Mood, Stroop	No intense exercise prior; clean baseline
Day 4	Mid-Restriction	2 AM–6 AM (4h)	Blood Draw #2	Reaction time, HRV, Mood, Stroop	Mid-point cognitive assessment
Day 7	End-Restriction	2 AM–6 AM (4h)	Blood Draw #3	Reaction time, HRV, Mood, Stroop	Identical fasted protocol as Day 1
Day 8	Buffer	7 hrs (ad lib)	None	—	Washout day; no data collected
Day 11	Mid-Recovery	10 PM–7 AM (9h)	Blood Draw #4	Reaction time, HRV, Mood, Stroop	No intense exercise prior
Day 14	Final	10 PM–7 AM (9h)	Blood Draw #5	Reaction time, HRV, Mood, Stroop	End-point cognitive test; final bloods

Table 1. Study Design and Blood Draw Schedule (Patient AS-28821, Satani Research Centre)

Measurements

Blood draws were analysed for the following panels on the same processing run to minimise inter-assay variability:
- Hormonal & Stress Panel: Total testosterone (ng/dL), morning cortisol (µg/dL)
- Inflammatory Marker: High-sensitivity C-reactive protein (hsCRP, mg/L)

- Metabolic & Lipid Panel: Fasting glucose (mg/dL), LDL cholesterol (mg/dL), HDL cholesterol (mg/dL), triglycerides (mg/dL)
- Hepatic Function: Alanine aminotransferase / ALT (U/L), aspartate aminotransferase / AST (U/L)
- Full Blood Count (FBC/CBC): White blood cell count (WBC), red blood cell count (RBC), haemoglobin, platelets, differential (neutrophils %, lymphocytes %)
- Daily performance and biometric assessments were conducted each morning between 06:30 and 07:00 and included: simple reaction time (validated smartphone application, five-trial mean, ms), heart rate variability (HRV, ms, measured supine on waking), handgrip dynamometry (dominant hand, kg), and a self-reported mood score (Likert-type scale, 1–10). A Stroop colour-word interference task was administered on days of blood draw (Days 1, 4, 7, 11, 14) as a formal measure of executive function.

RESULTS

Comprehensive Results Table

Parameter	Unit	Day 1	Day 4	Day 7	Day 11	Day 14
GENERAL						
Sleep Hours	hours	8	4	4	9	9
HORMONAL & STRESS PANEL						
Testosterone	ng/dL	610	565	495 ↓	550	595
Cortisol	µg/dL	12.0	14.2 ↑	21.5 ↑↑	15.5	12.8
C-Reactive Protein (CRP)	mg/L	0.5	0.9	1.8 ↑↑	0.8	0.6
METABOLIC & LIPID PANEL						
Fasting Glucose	mg/dL	88	93	99 ↑	89	87
LDL Cholesterol	mg/dL	95	102	115 ↑↑	105	98
HDL Cholesterol	mg/dL	55	53	48 ↓	52	54
Triglycerides	mg/dL	90	110	145 ↑↑	105	95
HEPATIC FUNCTION						
ALT	U/L	22	25	31 ↑	26	23

AST	U/L	19	21	26 ↑	22	20
COMPLETE BLOOD COUNT						
WBC	10^3/µL	6.5	7.1	7.8 ↑	7.0	6.7
RBC	10^6/µL	5.10	5.15	5.22	5.15	5.12
Haemoglobin	g/dL	15.1	15.3	15.6	15.4	15.2
Platelets	10^3/µL	230	245	275 ↑	250	235
Neutrophils	%	54	59	66 ↑	60	56
Lymphocytes	%	34	30	24 ↓↓	29	33
PERFORMANCE & NEUROCOGNITION						
Reaction Time	ms	270	312 ↑	338 ↑↑	315	292
HRV	ms	50	42 ↓	34 ↓↓	44	47
Grip Strength	kg	52	50	46 ↓	49	51
Mood Score	/10	8.5	5.5 ↓	3.5 ↓↓	7.0	9.0

Table 2. Serial Biomarker Results Across Experimental Timeline (↑ = elevated vs baseline; ↓ = reduced vs baseline; ↑↑/↓↓ = clinically notable change)

Phase 1 — Sleep Restriction (Days 1–7)

Day 1 (Baseline): All biomarkers fell within age-appropriate reference ranges. Serum testosterone 610 ng/dL; morning cortisol 12.0 µg/dL; hsCRP 0.5 mg/L; fasting glucose 88 mg/dL; LDL 95 mg/dL; HDL 55 mg/dL; triglycerides 90 mg/dL; WBC 6.5 × 10^3/µL (neutrophils 54%, lymphocytes 34%); reaction time 270 ms; HRV 50 ms; grip strength 52 kg; mood score 8.5/10. The subject reported having slept normally the night prior and felt well at Day 1 assessment.

Day 4 (Mid-Restriction): After four nights of four-hour sleep, early physiological stress was objectively detectable despite the subject reporting relative functional adequacy. Cortisol rose to 14.2 µg/dL (+18.3% from baseline). Testosterone fell to 565 ng/dL (−7.4%). CRP increased to 0.9 mg/L (+80%). Fasting glucose crept to 93 mg/dL. LDL rose to 102 mg/dL, with reciprocal HDL reduction to 53 mg/dL and triglyceride elevation to 110 mg/dL. WBC increased to 7.1 × 10^3/µL. Reaction time had slowed to 312 ms (+15.6%), and HRV fell to 42 ms (−16%). Mood score declined to 5.5/10. Stroop error rate showed initial deterioration. Subjectively, the subject

noted lapses in working memory and mild impairment in word retrieval—consistent with early prefrontal cortical stress.

Day 7 (End-Restriction): The most pronounced multi-system derangement was observed at this time point. Testosterone fell to 495 ng/dL, representing an 18.9% decline from baseline—a magnitude consistent with published findings associating one week of four-hour sleep with testosterone levels equivalent to those of men approximately a decade older. Morning cortisol spiked to 21.5 µg/dL (+79.2%), representing a near-doubling of baseline and suggesting marked hypothalamic-pituitary-adrenal (HPA) axis activation. CRP rose to 1.8 mg/L (+260%), crossing the threshold (>1.0 mg/L) associated with elevated cardiovascular inflammatory risk. Fasting glucose reached 99 mg/dL—approaching the American Diabetes Association pre-diabetes threshold of 100 mg/dL. LDL rose to 115 mg/dL, triglycerides to 145 mg/dL, HDL fell to 48 mg/dL. This lipid profile, if sustained, would warrant clinical review.

The haematological differential at Day 7 is particularly informative: WBC rose to 7.8 × 10³/µL with neutrophilia at 66% and relative lymphopaenia at 24%. This pattern—increased innate immune activity with a relative shift away from adaptive immunity—mirrors the immune dysregulation profile documented in sleep-deprived cohorts and is consistent with a stress-mediated glucocorticoid effect on lymphocyte margination. ALT and AST rose modestly (31 and 26 U/L respectively), remaining within normal limits but reflecting subtle hepatocellular stress under the cortisol load. Reaction time reached 338 ms (+25.2%), HRV fell to 34 ms (−32%), grip strength declined to 46 kg, and mood score fell to 3.5/10—the lowest recorded during the study. Crucially, the subject reported feeling that he was coping and continuing normal occupational and social functioning throughout, illustrating the profound disconnect between subjective perception and objective physiological deterioration.

Phase 2 — Recovery Sleep (Days 8–14)

Day 11 (Mid-Recovery): After four nights of nine-hour sleep, substantial but incomplete biomarker normalisation was evident. Cortisol fell to 15.5 µg/dL—still above baseline but substantially reduced from its Day 7 peak. Testosterone rose to 550 ng/dL, recovering approximately 55 ng/dL of the 115 ng/dL lost during restriction. CRP returned to 0.8 mg/L, closely approaching baseline. Fasting glucose normalised to 89 mg/dL. The lipid profile improved materially: LDL 105 mg/dL, HDL 52 mg/dL, triglycerides 105 mg/dL. WBC fell to 7.0 × 10³/µL, with neutrophil differential beginning to contract (60%) and lymphocytes recovering (29%). HRV rose to 44 ms; reaction time improved to 315 ms. Mood score improved markedly to 7.0/10, with the subject describing the subjective experience of recovery as qualitatively distinct—reporting that feeling rested felt unfamiliar given the preceding period of adapted sleepiness.

Day 14 (Final Assessment): By the conclusion of the study, most biomarkers had normalised or approached their Day 1 values. Testosterone reached 595 ng/dL (97.5% of baseline); cortisol 12.8 µg/dL (nearly at baseline); CRP 0.6 mg/L; fasting glucose 87 mg/dL; LDL 98 mg/dL; HDL 54 mg/dL; triglycerides 95 mg/dL; WBC 6.7 × 10³/µL (neutrophils 56%, lymphocytes 33%); ALT 23 U/L; AST 20 U/L. However, complete restoration was not confirmed for all endpoints. HRV at Day 14 was 47 ms—94% of the Day 1 value of 50 ms, suggesting residual autonomic nervous

system incompleteness. Reaction time at 292 ms remained above the 270 ms baseline. Stroop task performance had improved through recovery but had not fully returned to Day 1 accuracy, consistent with literature suggesting that executive function recovery may lag behind peripheral biomarker normalisation by several additional days to weeks.

DISCUSSION

Hormonal and Endocrine Effects

The 18.9% fall in serum testosterone over seven days of four-hour sleep is clinically significant. Published controlled trials have demonstrated that one week of five-hour sleep restriction reduces testosterone in young healthy males by 10–15%, with some studies reporting up to 24% suppression depending on restriction severity and individual variation. The present data—showing a 115 ng/dL absolute decline—falls squarely within this range. The mechanism involves disruption of the nocturnal growth hormone and luteinising hormone (LH) pulses, which are tightly entrained to slow-wave sleep and largely absent when sleep duration is severely curtailed. Concurrently, the 79.2% rise in morning cortisol by Day 7 reflects progressive HPA axis dysregulation; chronically elevated cortisol additionally exerts direct suppressive effects on Leydig cell testosterone synthesis, creating a compounding neuroendocrine feedback loop.

Inflammatory and Metabolic Perturbation

The 260% elevation in hsCRP by Day 7 (0.5 to 1.8 mg/L) crosses the threshold conventionally used to stratify cardiovascular inflammatory risk and is consistent with the well-established relationship between sleep restriction and pro-inflammatory cytokine upregulation, particularly interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α). The lipid profile changes—LDL rising from 95 to 115 mg/dL, HDL falling from 55 to 48 mg/dL, and triglycerides rising from 90 to 145 mg/dL—reflect the influence of cortisol and sympathetic activation on hepatic lipid metabolism. The rise in fasting glucose to 99 mg/dL, approaching the pre-diabetic threshold, is consistent with sleep-mediated impairment of insulin sensitivity via reduced glucose transporter (GLUT-4) expression and heightened sympathetic tone. These changes, while each modest in isolation, co-occur in a pattern characteristic of early metabolic syndrome when sustained chronically.

Haematological and Immunological Changes

The neutrophilia (54% to 66%) and reciprocal lymphopaenia (34% to 24%) observed by Day 7, alongside the WBC elevation, represent a stress-mediated immune shift. Glucocorticoid-driven lymphocyte redistribution from peripheral blood to lymphoid organs, combined with neutrophil demargination, produces precisely this differential profile. This immunological state—while acutely adaptive in the context of physical stress—is maladaptive when chronic, as it is associated with reduced vaccine efficacy, impaired antiviral response, and increased susceptibility to upper respiratory tract infections. The modestly elevated liver enzymes (ALT,

AST) are likely secondary to the cortisol burden and elevated metabolic stress rather than primary hepatic pathology, and their normalisation by Day 14 supports this interpretation.

Neurocognitive Impairment and Recovery

The 25.2% slowing in reaction time (270 to 338 ms) and 32% decline in HRV by Day 7 represent objective, performance-level deficits. The reaction time decrement is particularly noteworthy in applied contexts: a mean reaction time of 338 ms is below the threshold typically required to pass standard fitness-to-drive assessments, despite the subject reporting functional normality. This illustrates the canonical 'impaired awareness of impairment' phenomenon that characterises chronic partial sleep deprivation—subjects adapt to their degraded state and lose the reference point by which to gauge deterioration.

The incomplete neurocognitive recovery by Day 14—with reaction time at 292 ms (still 22 ms above baseline) and Stroop performance below Day 1 accuracy—supports the hypothesis that executive function recovery is temporally dissociated from peripheral biomarker normalisation. Multiple investigators have proposed that the prefrontal cortex, with its high metabolic demand and particular vulnerability to slow-wave sleep loss, requires a more extended or higher-quality recovery period than haematological or endocrine systems.

Subjective-Objective Dissociation

A recurring and clinically critical finding across all restriction phases was the progressive divergence between how the subject felt and what his biomarkers demonstrated. This dissociation is not idiosyncratic: it is reproducibly documented in the sleep science literature. The implication for clinical practice and occupational health is significant—individuals working under conditions of chronic partial sleep restriction who self-report 'managing fine' cannot be assumed to be physiologically unimpaired. Serial biomarker monitoring, even in ostensibly healthy individuals, offers a quantitative corrective to this subjective miscalibration.

LIMITATIONS

The findings of this case report must be interpreted within several important methodological constraints:

Short window for slow-moving markers: HbA1c, fasting insulin, and atherogenic indices require 8–12 weeks of sustained change to manifest reliably. The 14-day window captures acute but not chronic metabolic risk.

Self-report of adherence: Sleep duration is reported as intended protocol windows. No polysomnographic or actigraphic verification was performed; actual sleep obtained within those windows may have differed. Notwithstanding these limitations, the directional consistency of changes across all measured systems—and their alignment with the established mechanistic literature—supports the biological plausibility of the observed effects rather than their attribution to random variation or methodological artefact.

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DECLARATIONS

Competing Interests - The author declares no competing interests. No financial relationships, commercial affiliations, institutional sponsorships, or external funding of any form were associated with the conduct of this study. No equipment, reagents, or material support were received from any commercial or non-commercial entity. All costs were personally borne by the author-investigator. The study was conceived and executed independently, and no external party holds any financial or non-financial interest in the findings reported herein.

Patient Consent - Informed consent was obtained from the study participant. The subject of this investigation and the principal investigator are the same individual — Abhijeet Satani (Patient ID: AS-28821). The author designed the experimental protocol, provided voluntary self-consent before commencement, served as the sole participant throughout, and conducted all subsequent data interpretation. No third-party participants were enrolled. No data were collected from any individual other than the author-investigator.

Ethics Statement - This self-experiment was conducted under the internal oversight of Satani Research Centre (SRC/T/8/0.9). The intervention — controlled short-term sleep restriction followed by a structured recovery sleep phase — presents no greater physiological risk than the occupational sleep restriction routinely experienced by large segments of the working population. No invasive procedures were performed beyond standard venepuncture, which was

carried out by trained laboratory personnel in accordance with established clinical protocols. All biomarker assessments were conducted within accepted reference frameworks. The experimental protocol was concluded by design at 14 days, and at no point during the study did any measured parameter reach a threshold warranting clinical intervention or early termination.

Document Status - This document constitutes a personal research record maintained at Satani Research Centre solely for private reference. All data were prospectively collected and processed in-house between 1 and 14 March 2026. This record has not been submitted to any academic journal, has not undergone external peer review, and should not be interpreted as a formally peer-reviewed scientific publication.

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