Uncovering sleep-related barriers to motor recovery after stroke:

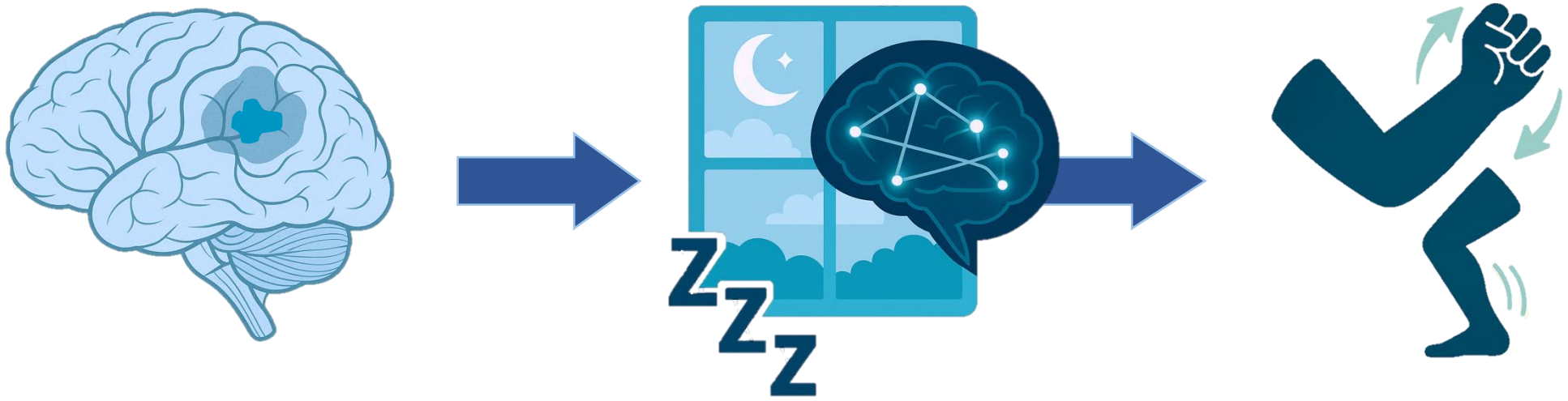
Behavioural, neurophysiological, and structural insights.

UNIVERSITY OF OXFORD

CENTRE for INTEGRATIVE NEUROIMAGING

Background

- Stroke is a leading cause of worldwide disability, with over 50% of stroke survivors experiencing long-term motor impairment.
- As good outcomes after stroke are dependent on the ability of the brain to adapt and repair, it is critical we understand how neuroplasticity can be influenced shortly after stroke to maximise recovery.
- Sleep has long been thought to offer a unique window of opportunity for neuroplasticity and thus may be an important and modifiable target for post-stroke recovery



Sleep after Stroke

- Sleep disruption is commonplace after stroke
- Estimated prevalence of post-stroke insomnia has been reported to be as high as 60%

Brain Injury

Medications

Daily Routines

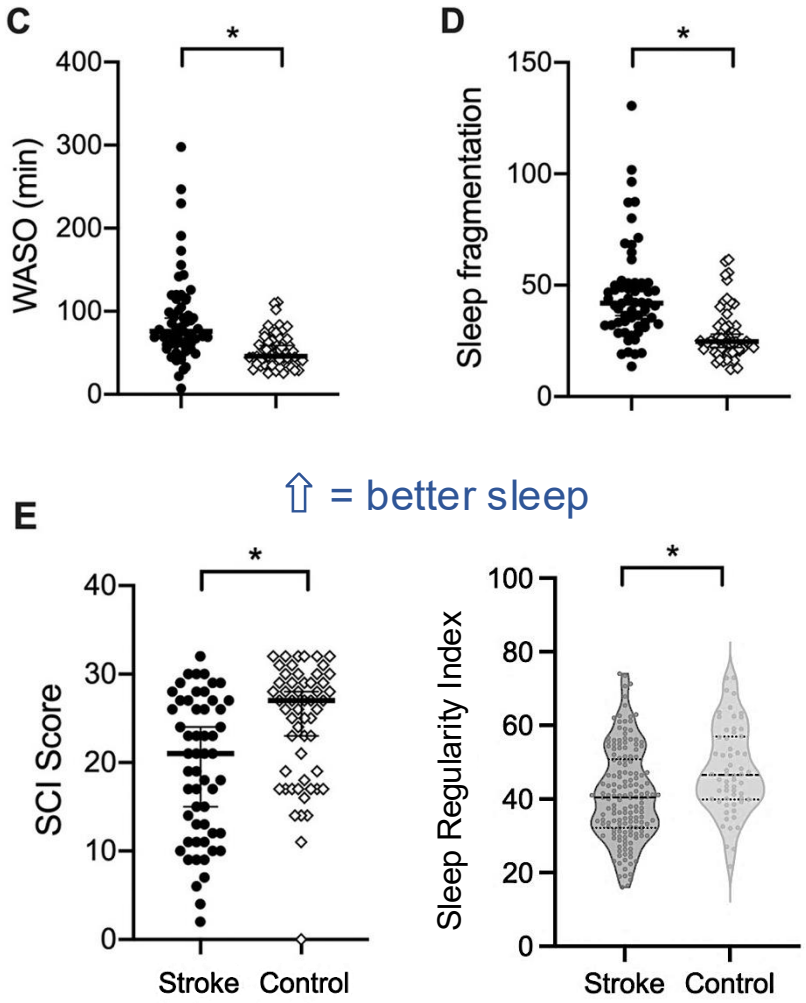
Mood

Incontinence

Pain

Co-morbidities

⇩ = less disrupted sleep

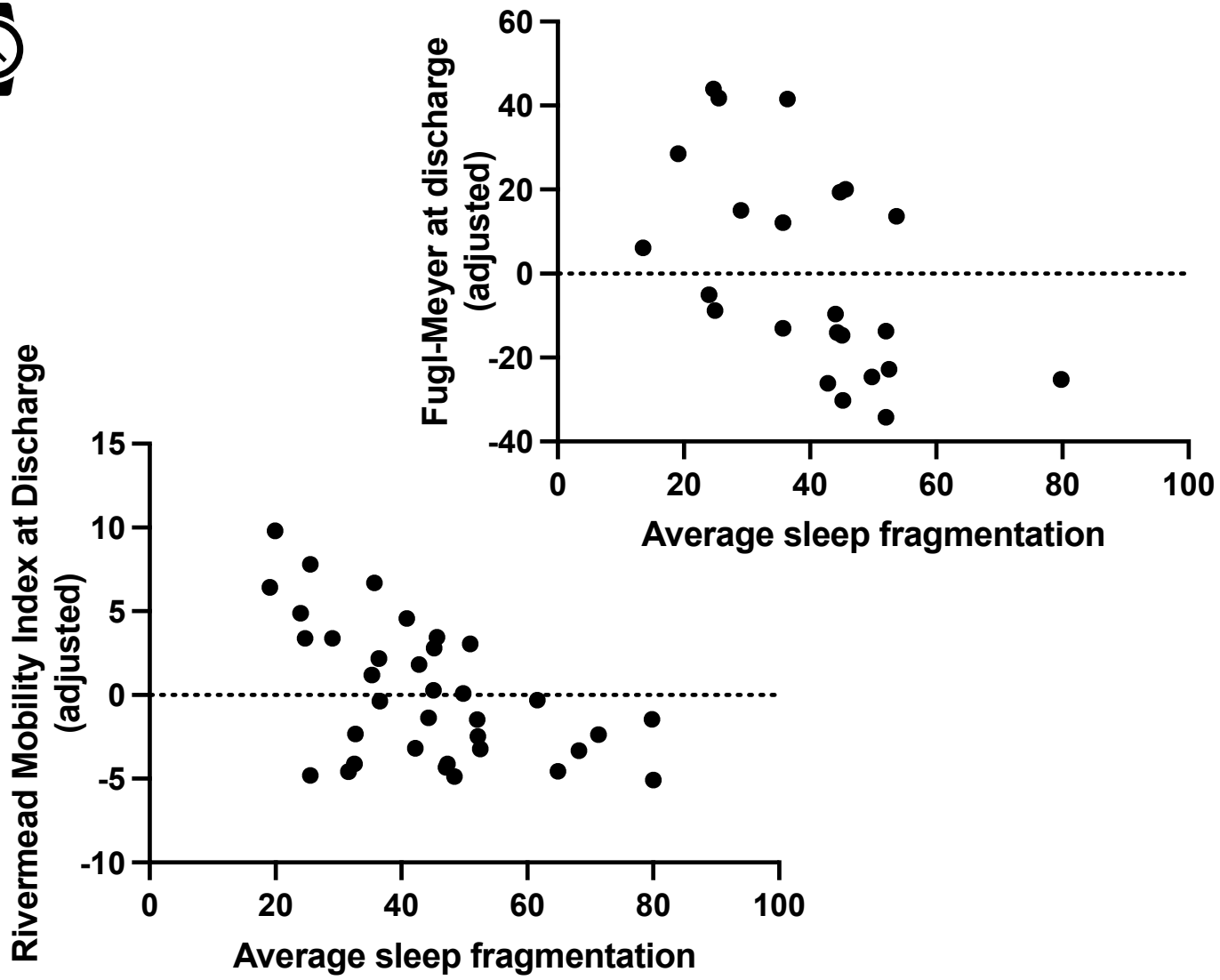


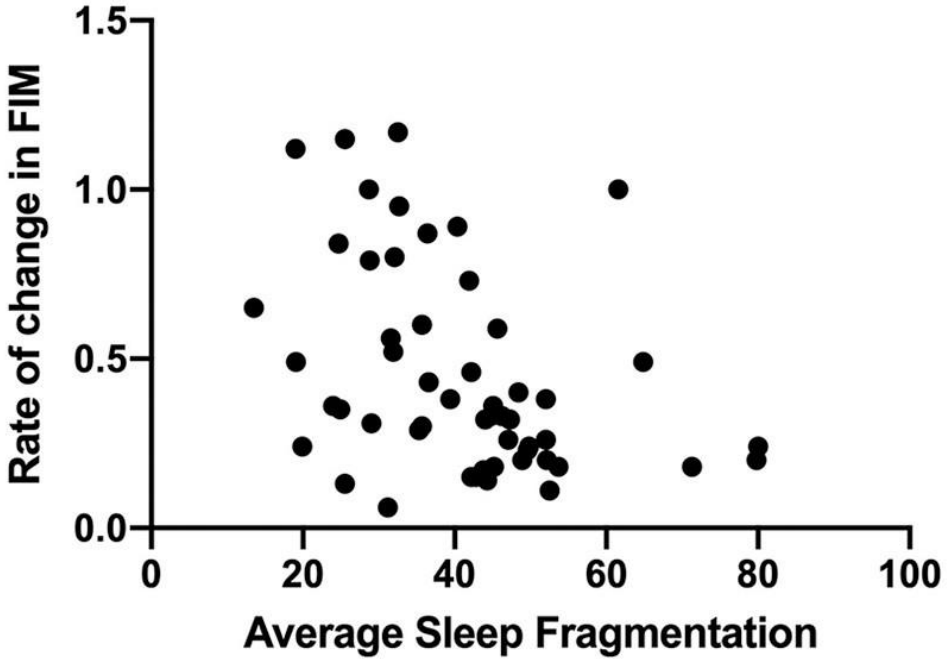
Fleming et al., 2020; Schruers et al., 2025

Sleep after Stroke

⇦ = less disrupted sleep

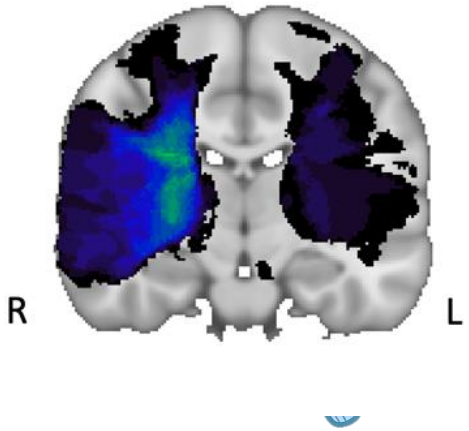
⇧ = better motor outcome

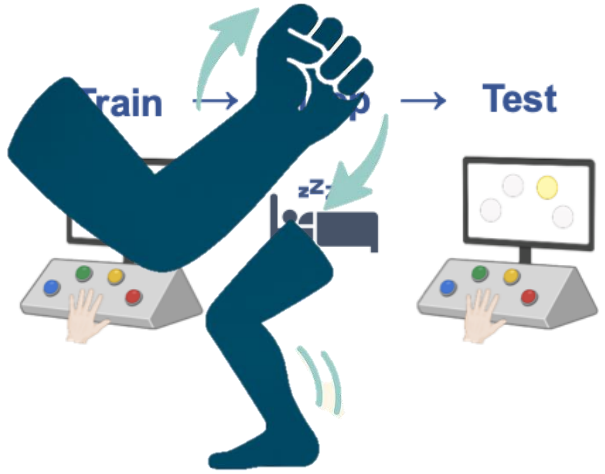




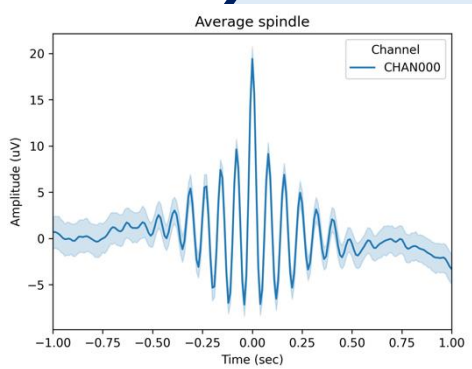
Fleming et al., 2020

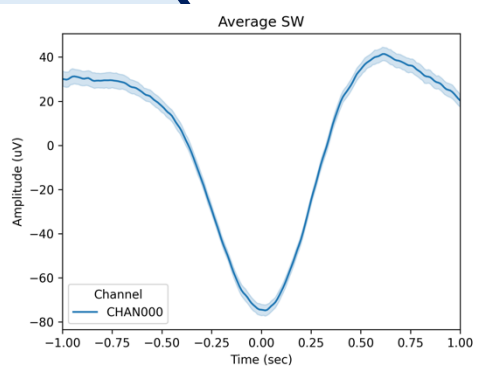
Missing Link ?!





Memory Consolidation





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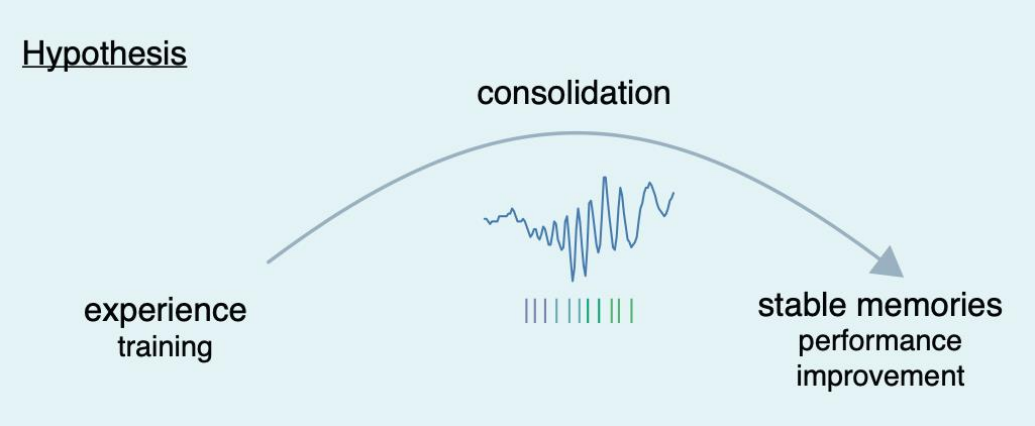
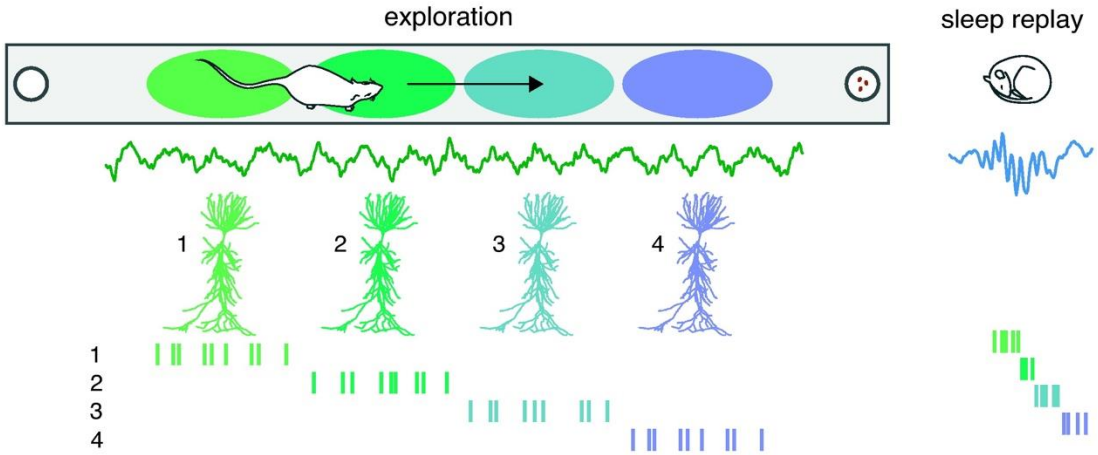
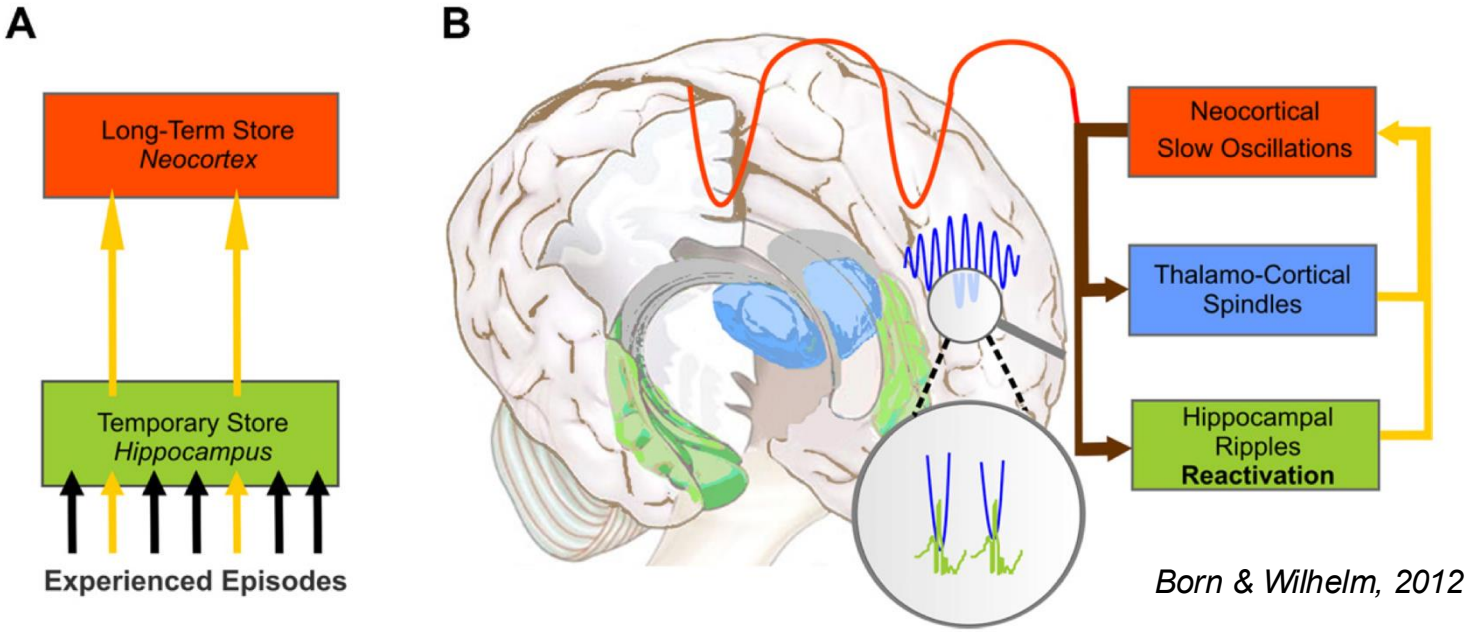


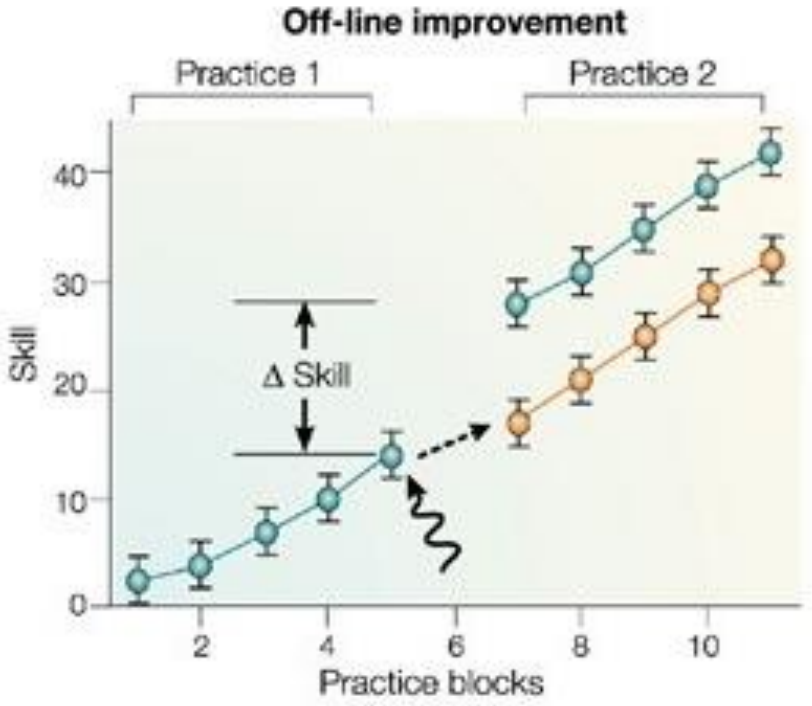


Girardeau & Zugaro, 2011

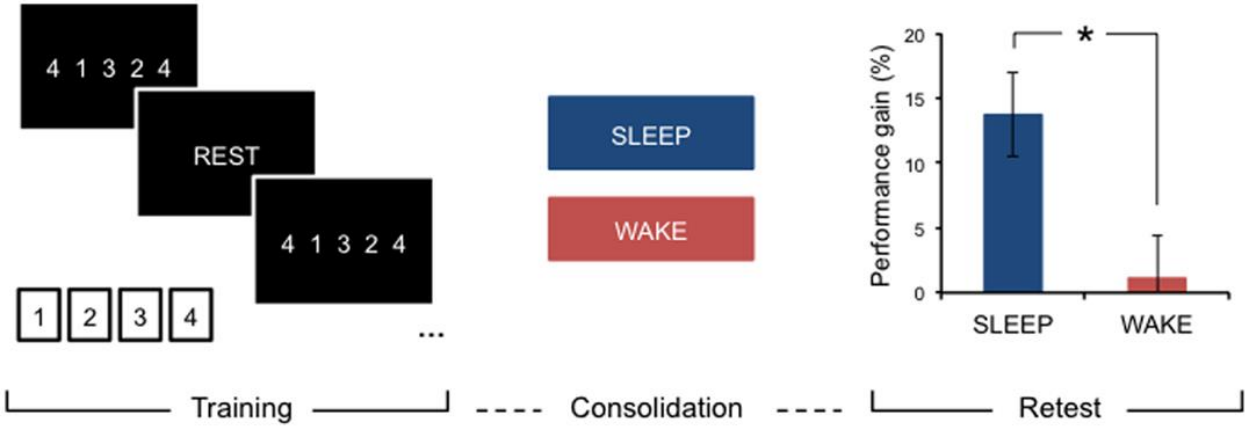


Born & Wilhelm, 2012

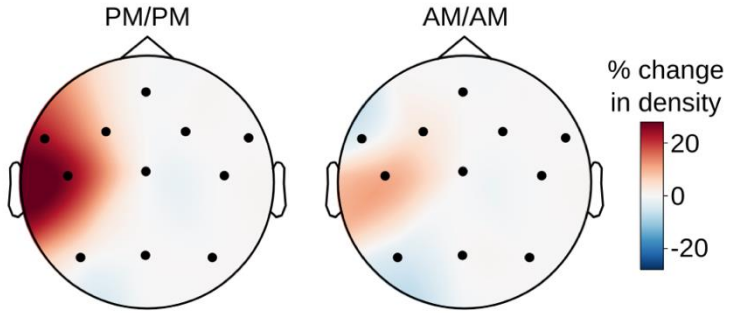
Motor Consolidation



Robertson, Pascual-Leone & Miall, 2004



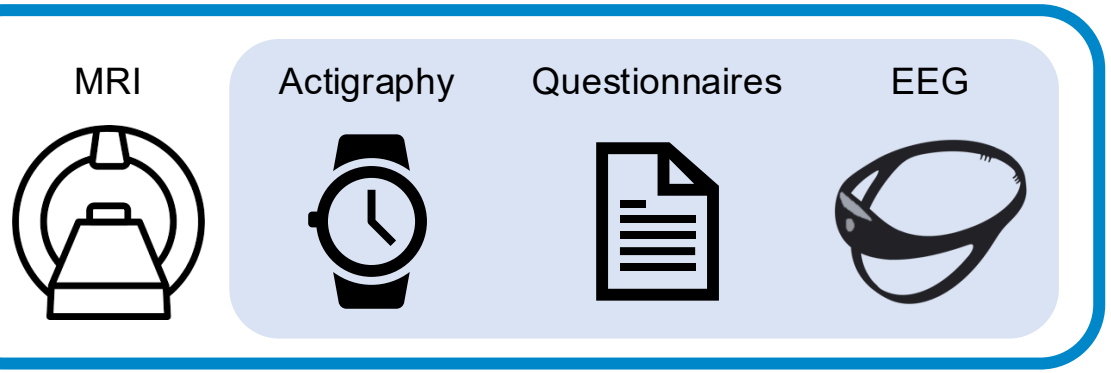
Gudberg & Johansen-Berg, 2015

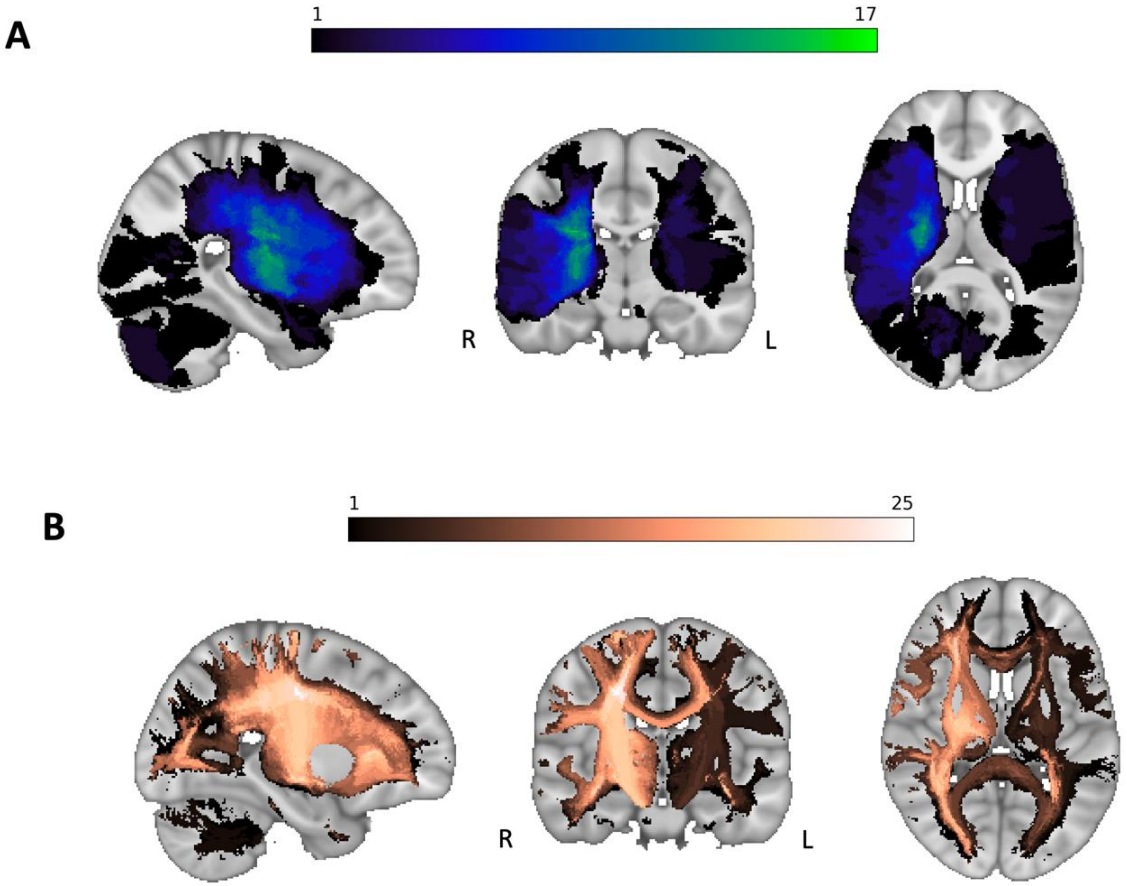


Solano et al., 2024

Stroke Lesions

	Mean (SD) or count
Age, years	64 (12)
Male/female	27/11
Time since stroke, months	95 (66)
Lesioned left/right hemisphere	11/27
Estimated Body Mass Index (N=25)	27 (4)
Type of stroke (N=26)	
Ischaemic	16
Haemorrhagic	4
Unknown	6

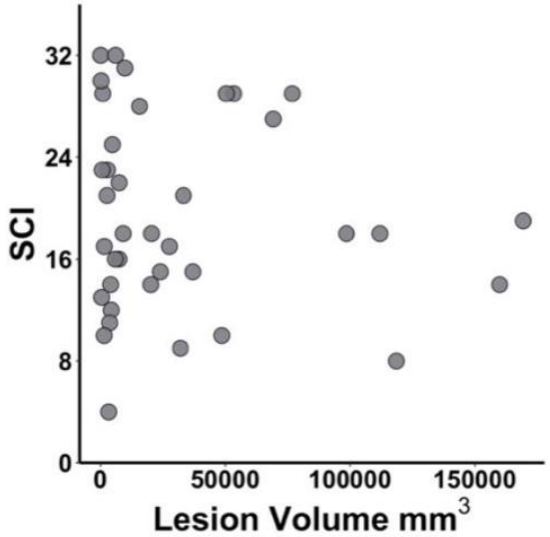


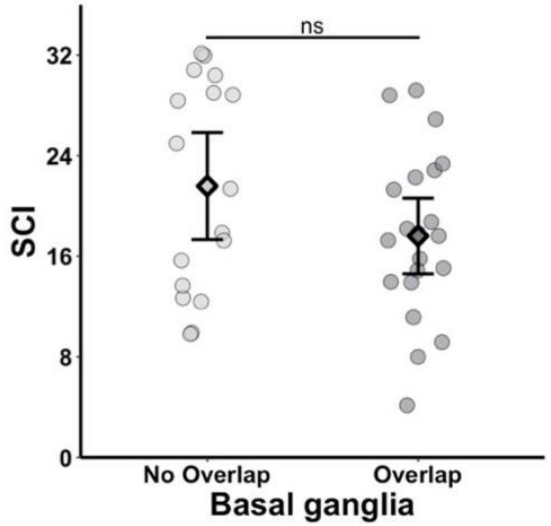


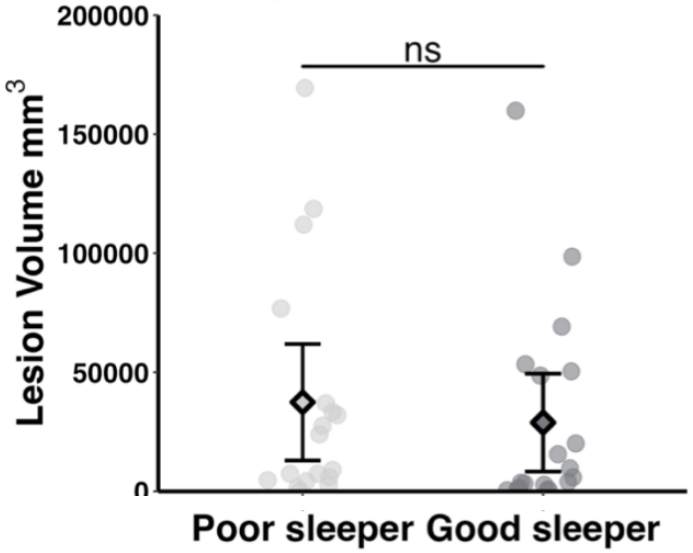
A) Lesion Overlap, B) Structural disconnectome map

Guttesen et al., MedRxiv, 2025

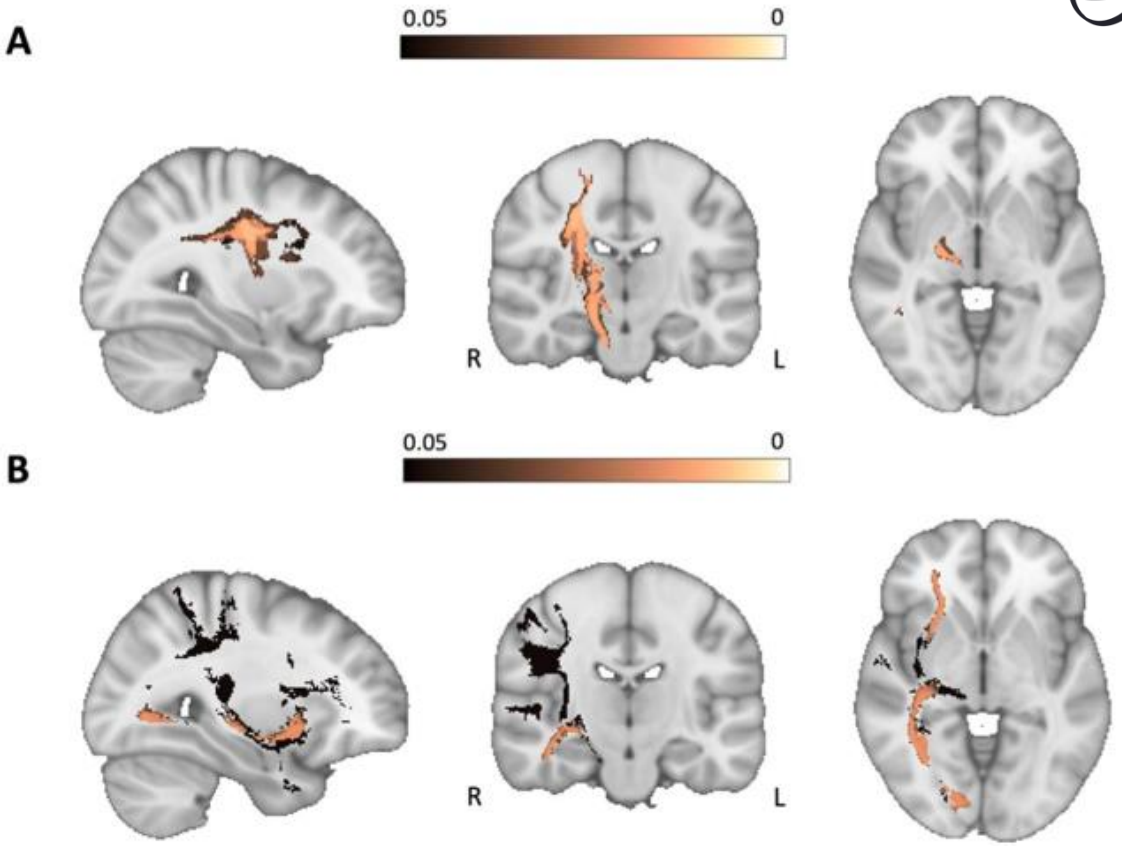
Stroke Lesions







Relationships between structural disconnectome and A) spindle density and B) spindle amplitude



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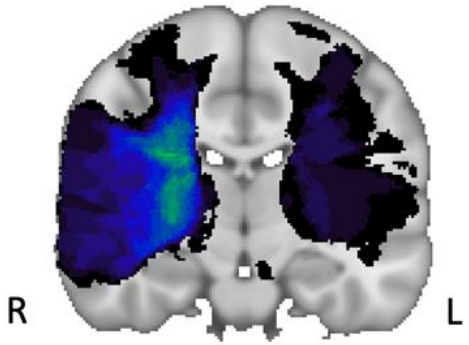
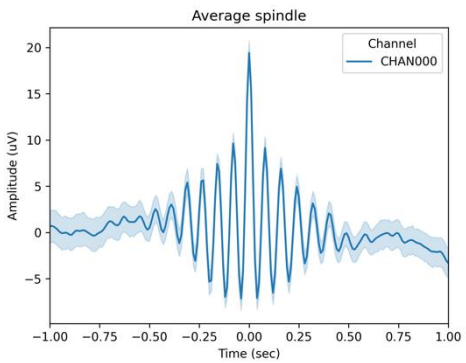
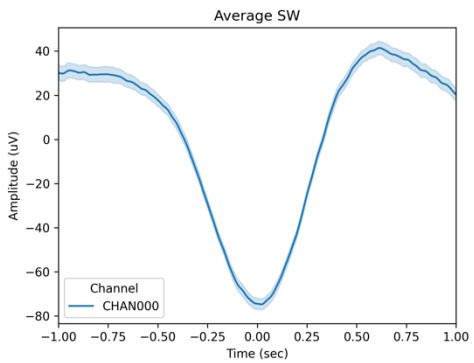


Memory Consolidation





Trial Design

≤ 7 days

Demographics

PREP2 Algorithm

- SAFE movement assessments, Age, TMS, NIHSS

~ 1 month

Sleep Assessments

- Sleep Condition Indicator (SCI), 7-night Actigraphy, Overnight EEG

Health Questionnaires

Motor Consolidation

- Serial Reaction Time Task (SRTT)

Train → Sleep → Test

~ 6 months

Sleep Assessments

- SCI, 7-night Actigraphy

Motor Outcomes

- ARAT, FMA, RMI, 9HPT

Health Questionnaires

Weightman et al., BMJ Open. 2024

Demographics

Consented

Ongoing

Complete

Withdrawn

Average age: **72.0** (43-91 yrs)

Sex: **Male** (45); **Female** (37)

NIHSS: **7.8** (1-24)

PREP2 scores: **Poor** (11); **Limited** (8); **Good** (26); **Excellent** (37)

Stroke type: **Ischemic** (68); **Haemorrhagic** (14)

Stroke laterality: **Right** (43); **Left** (37); **Unknown** (2)

Handedness: **Right** (74); **Left** (8)

mRS: **2.9** (1-5)

SCI: **22.0** (2-32)

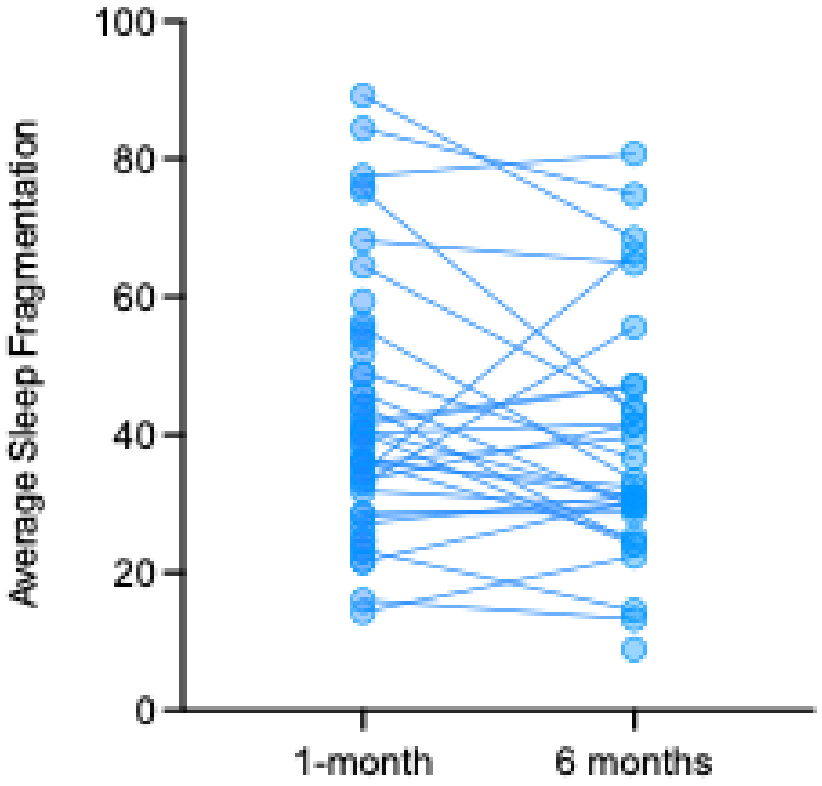
PHQ8: **5.6** (0-19)

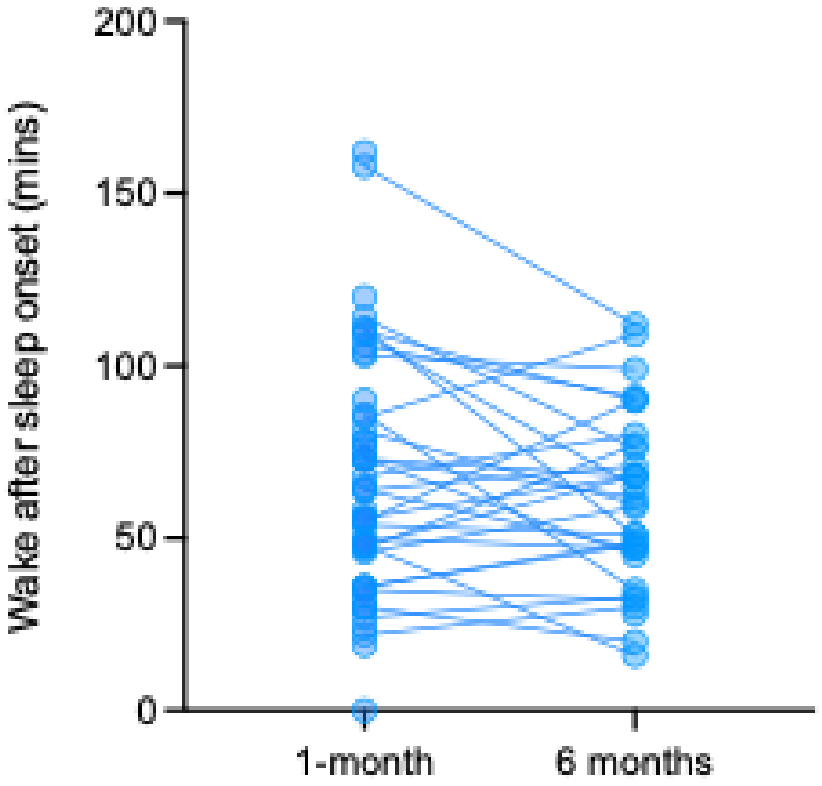
Stop-Bang: **3.7** (1-7)

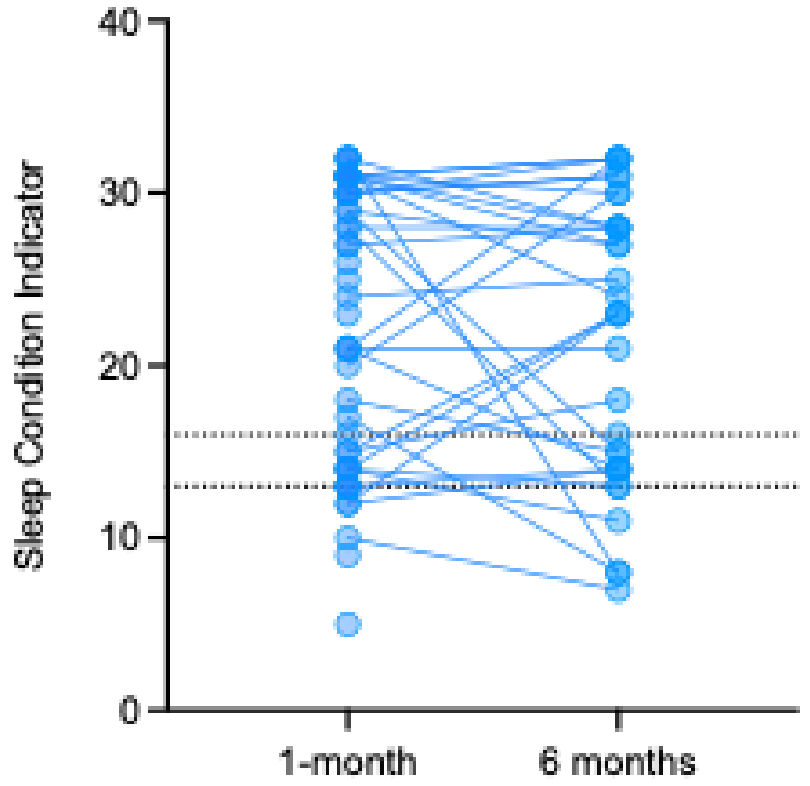
Sleep

⇩ = less disrupted sleep

⇧ = better self-reported sleep

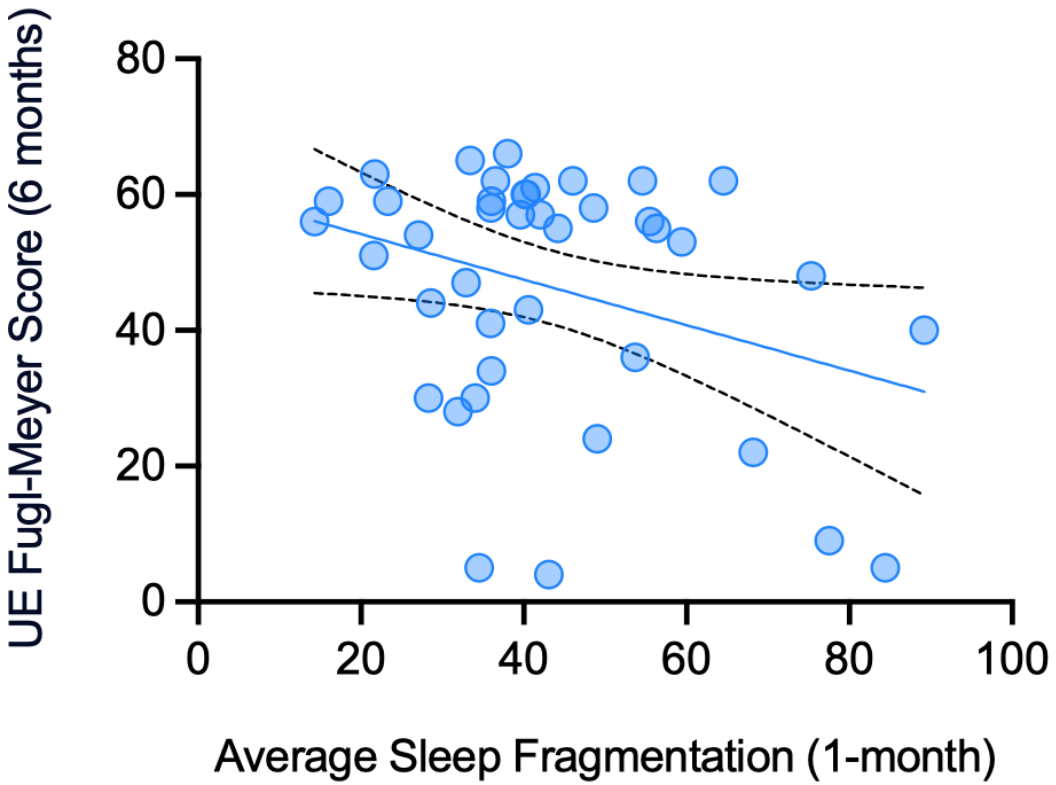


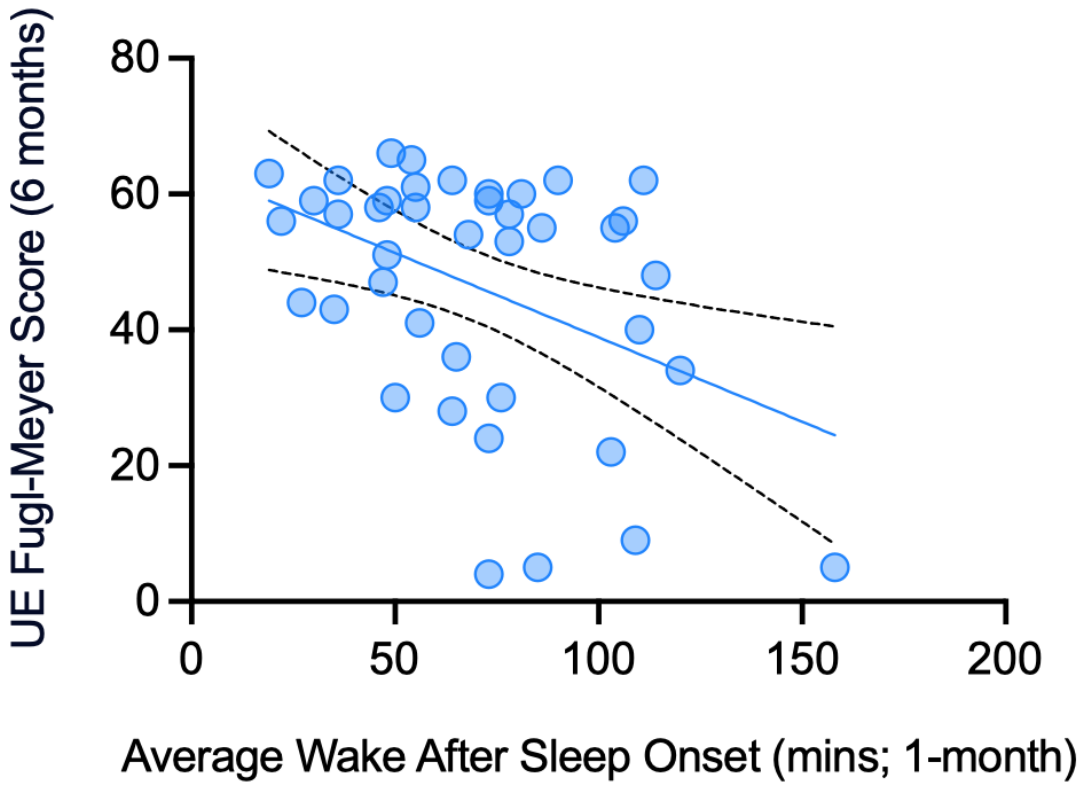




Sleep & Recovery

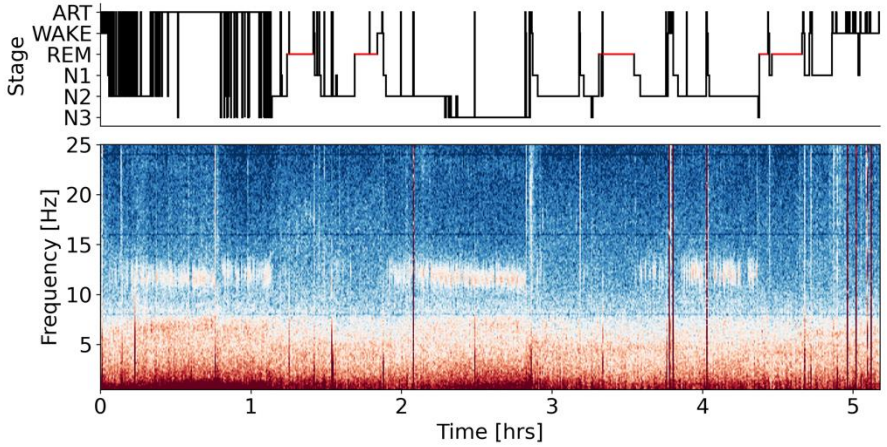
⇦ = less disrupted sleep, ⇧ = better function

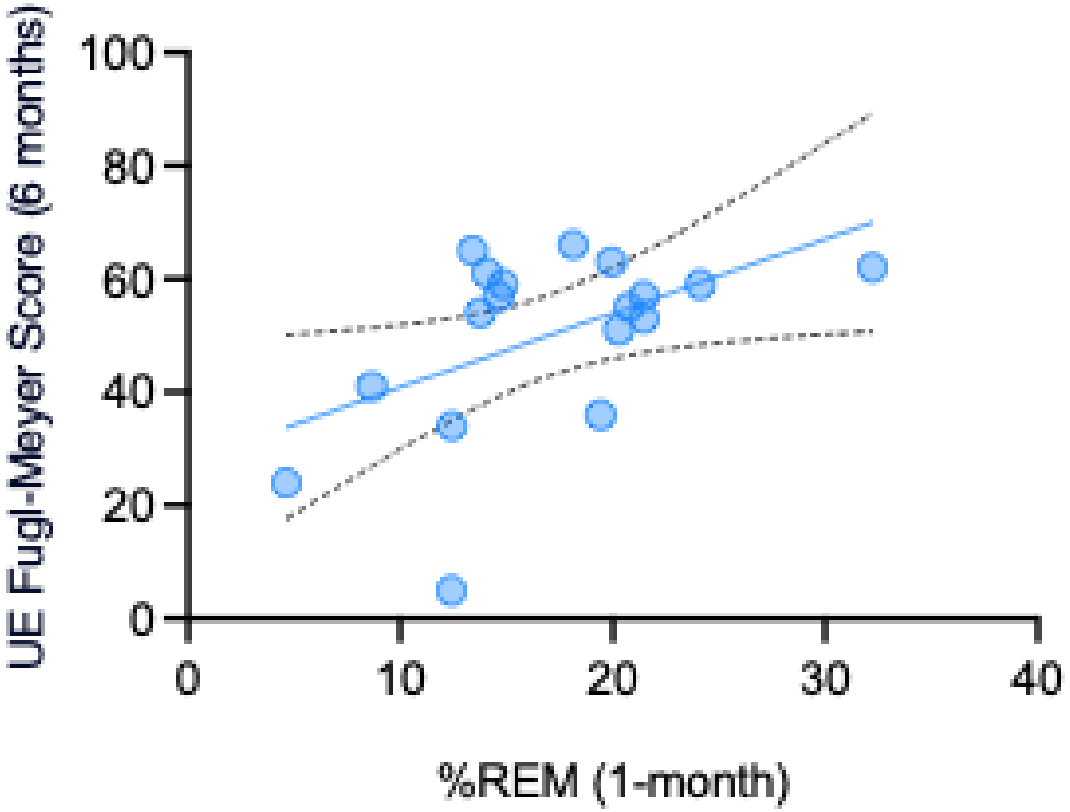




Sleep & Recovery

⇨ = more REM sleep, ⇧ = better function



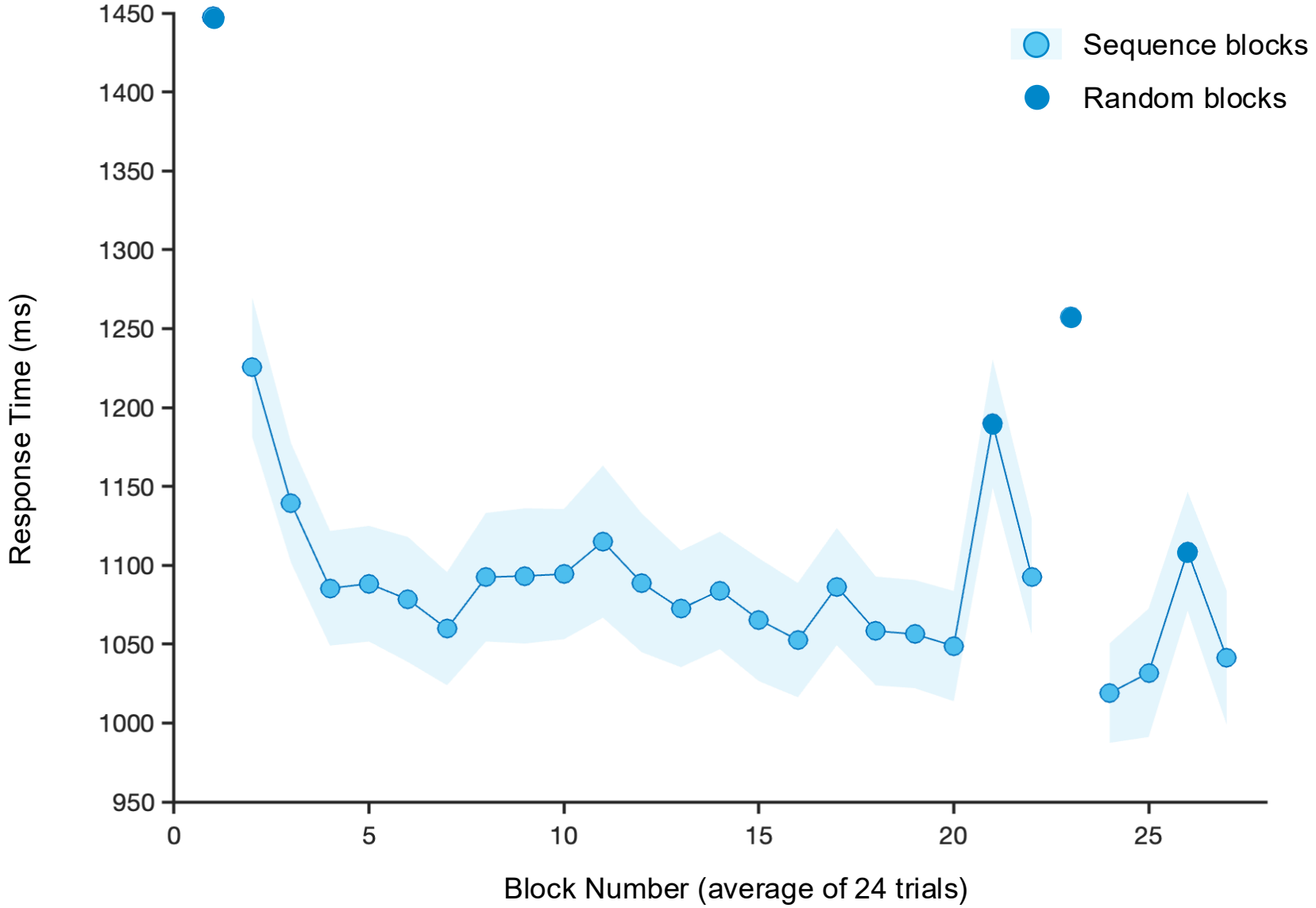


- Preliminary (& exploratory) indication that more REM sleep may be important to recovery processes.
- No current associations with any aspects of NREM sleep.

Motor Consolidation

n = 40 (~1-month post-stroke)

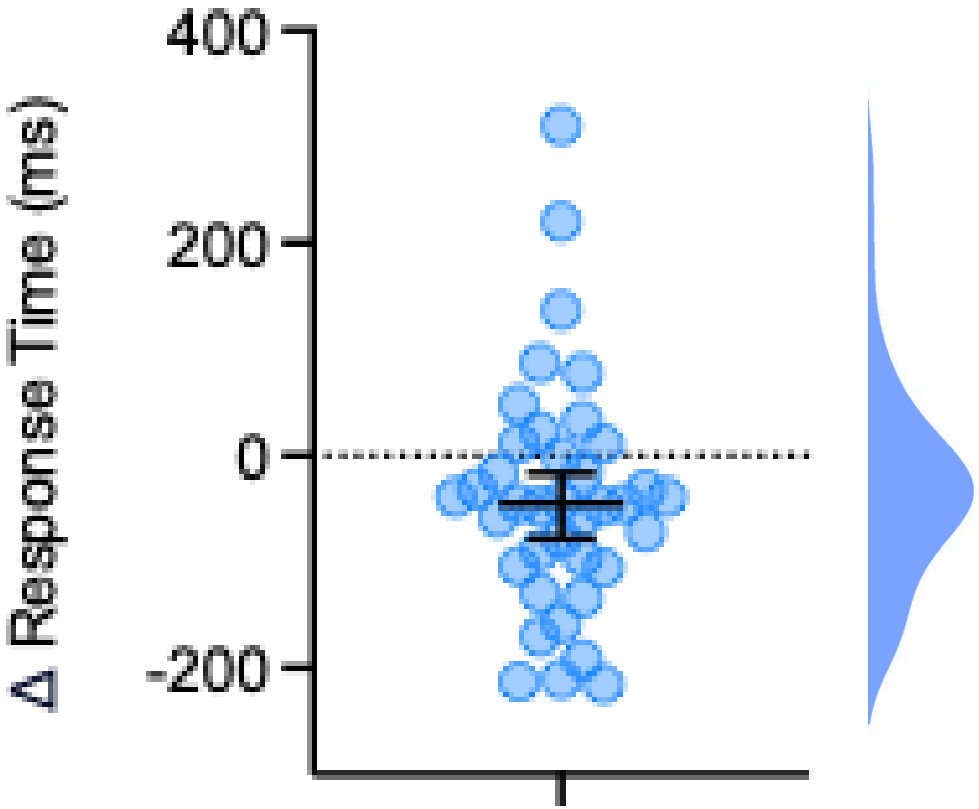
- 12-item sequence
- **Training:**
  2 Random Blocks;
  20 Sequence Blocks
- **Test:**
  2 Random Blocks;
  3 Sequence Blocks



Motor Consolidation

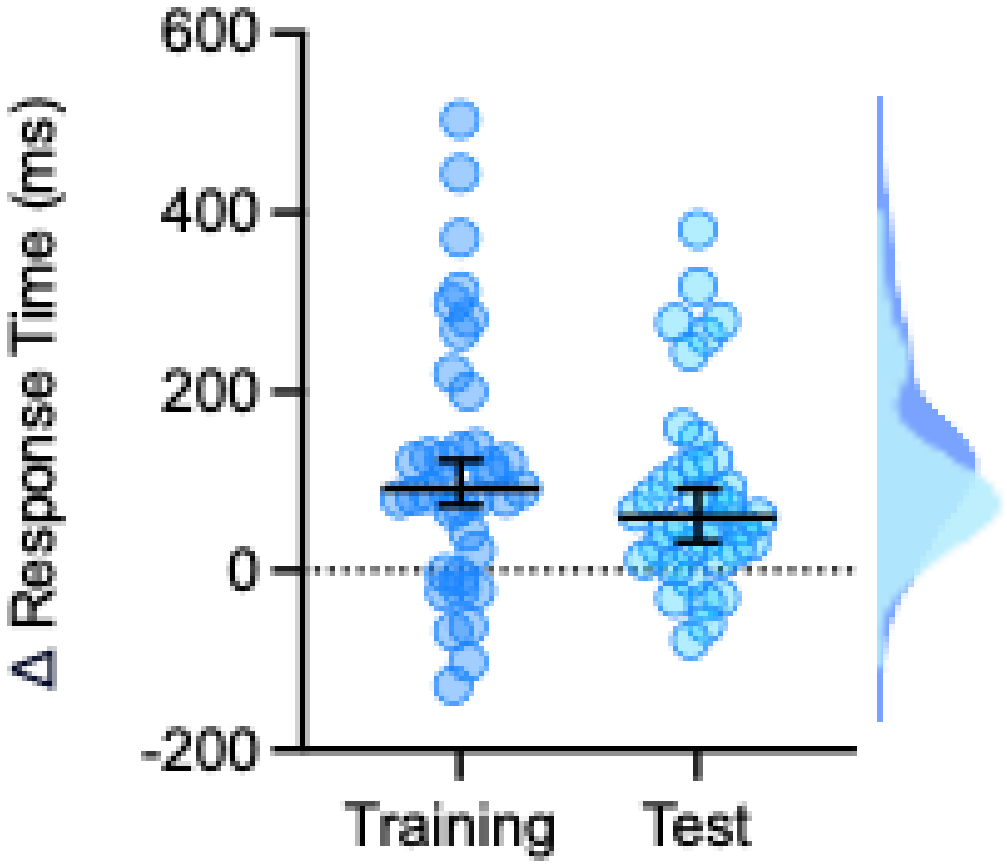
Overnight Consolidation

⇩ = more consolidation



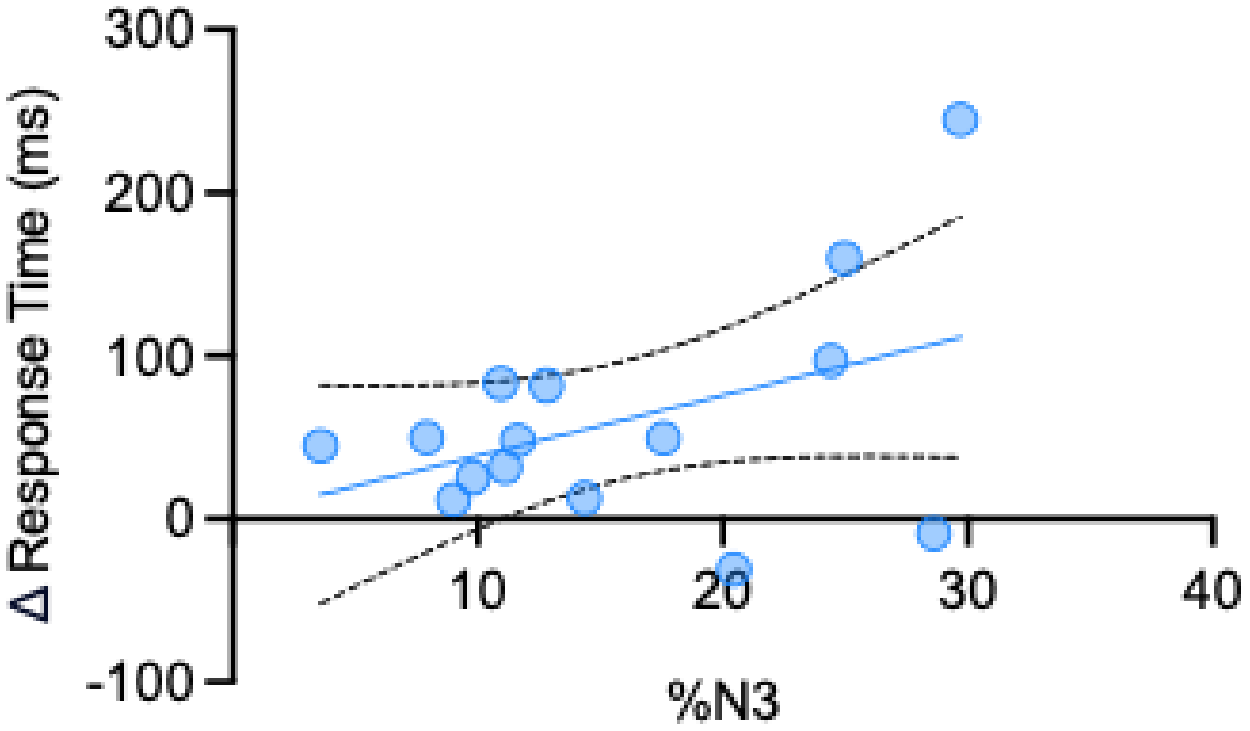
Sequence Specificity

⇧ = better sequence encoding



Motor Consolidation

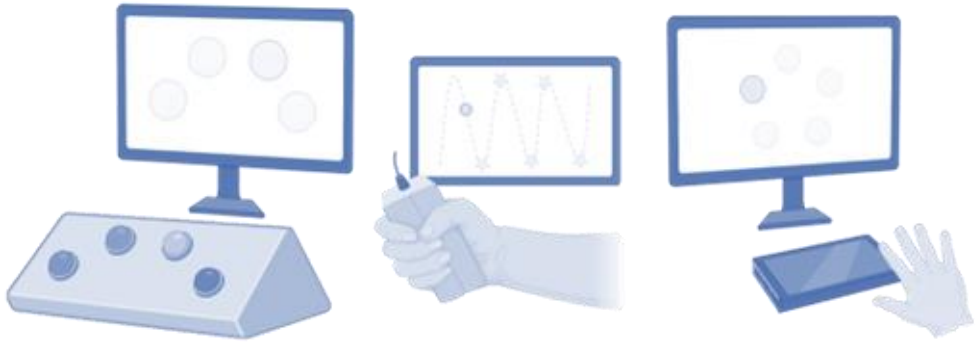
⇧ = better sleep-dependent sequence encoding



- Preliminary indication that more time spent in N3 (slow-wave sleep) may lead to better sequence encoding at re-test.
- Further analysis will endeavour to identify correlates with sleep microarchitecture e.g., sleep spindles, slow oscillations, and their temporal coupling

Conclusions

- Taken together, these multimodal findings suggest that sleep disruption is not merely a symptom of stroke but a potential mechanistic barrier to motor recovery.
- By linking behavioural consolidation, sleep physiology, and MRI, this work highlights sleep as an under recognised but modifiable target for potentially improving rehabilitation outcomes.
- Future interventions aimed at improving sleep quality and/or enhancing specific sleep physiology may offer a novel pathway to strengthen motor consolidation and promote functional gains after stroke.



Thank You!

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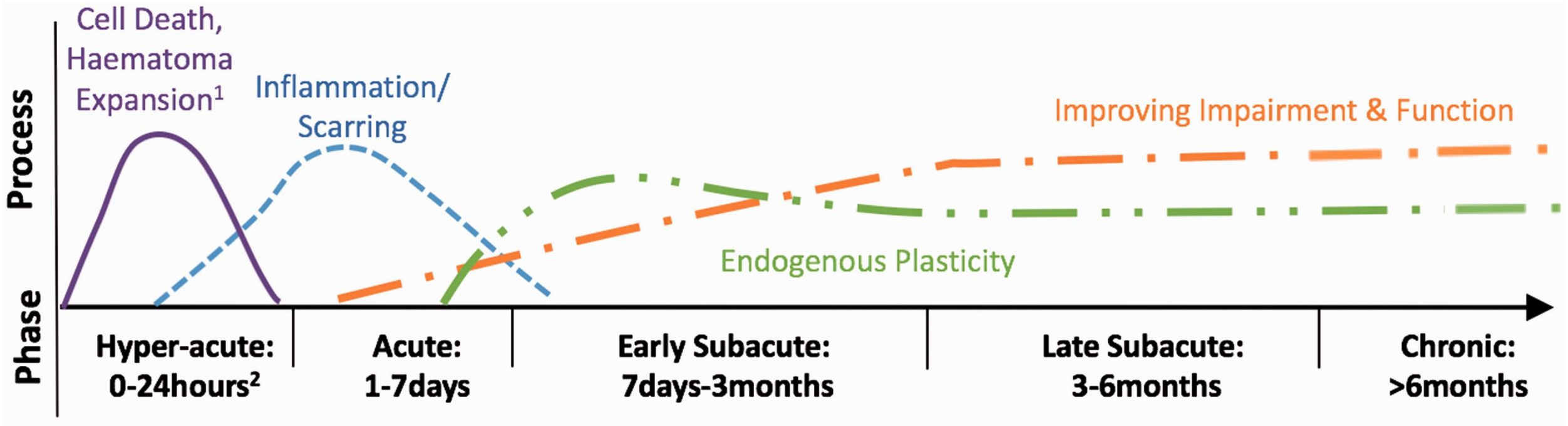
Process
Phase
Cell Death, Haematoma Expansion[1]
Inflammation/ Scarring
Improving Impairment & Function
Endogenous Plasticity
Hyper-acute: 0-24hours[2]
Acute: 1-7days
Early Subacute: 7days-3months
Late Subacute: 3-6months
Chronic: >6months