

Unobtrusive Longitudinal Monitoring Reveals Sleep and Heart Rate Alternations Associated with Amyloid Pathology



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Background

Sleep disturbances and changes in nocturnal heart rate dynamics are increasingly recognized as early features of neurodegenerative diseases. However, detecting these subtle, real-world changes in preclinical Alzheimer’s disease (AD) remains challenging. Unobtrusive, continuous sleep monitoring may afford an effective method for identifying early disease-related changes. This study examines longitudinal changes in multi-domain sleep physiological metrics among older adults stratified by amyloid PET-defined pathology.

Study design

- This study was conducted within the **Digital Evaluations and Technologies Enabling Clinical Translation for Alzheimer’s Disease (DETECT-AD) protocol** (ClinicalTrials.gov identifier: NCT05385913), an ongoing longitudinal observational cohort.
- Participants** were classified as amyloid positive (Aβ+) or amyloid negative (Aβ-) based on Aβ Florbetapir PET imaging.
- Sleep and nocturnal heart rate data** were collected via electronic bed mats (Emfit QS; Vaajakoski, Finland; [Figure 1](#)) in the homes of participants. Daily recordings of features were aggregated into weekly means.
- Linear mixed-effects models** examined the group*time interaction across metrics spanning sleep architecture, heart rate, heart rate variability, and sleep recovery (pre-awaking heart rate RMSSD/pre-sleep heart rate RMSSD). All models were adjusted for age, sex, education, APOE status, and clinical dementia rating (CDR) global score. Benjamini-Hochberg corrections were applied to adjust p-values for multiple testing.

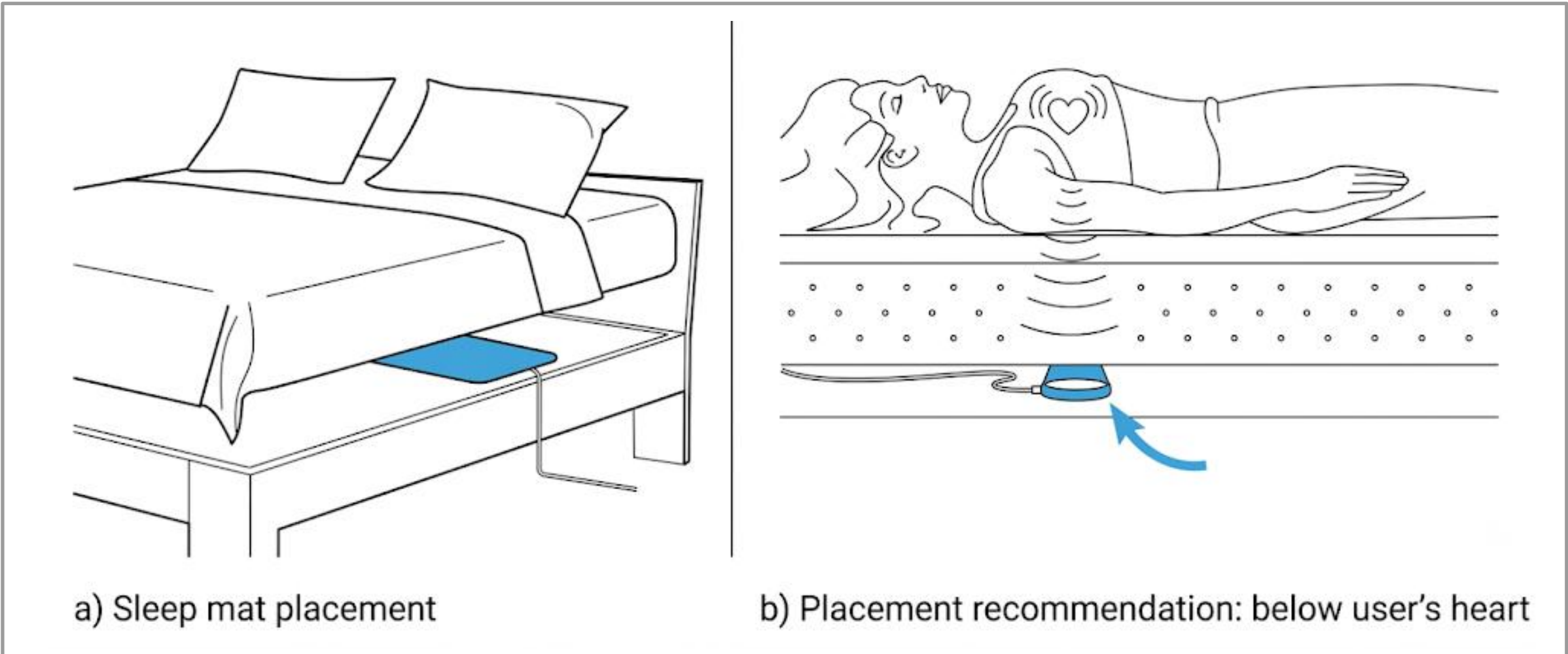


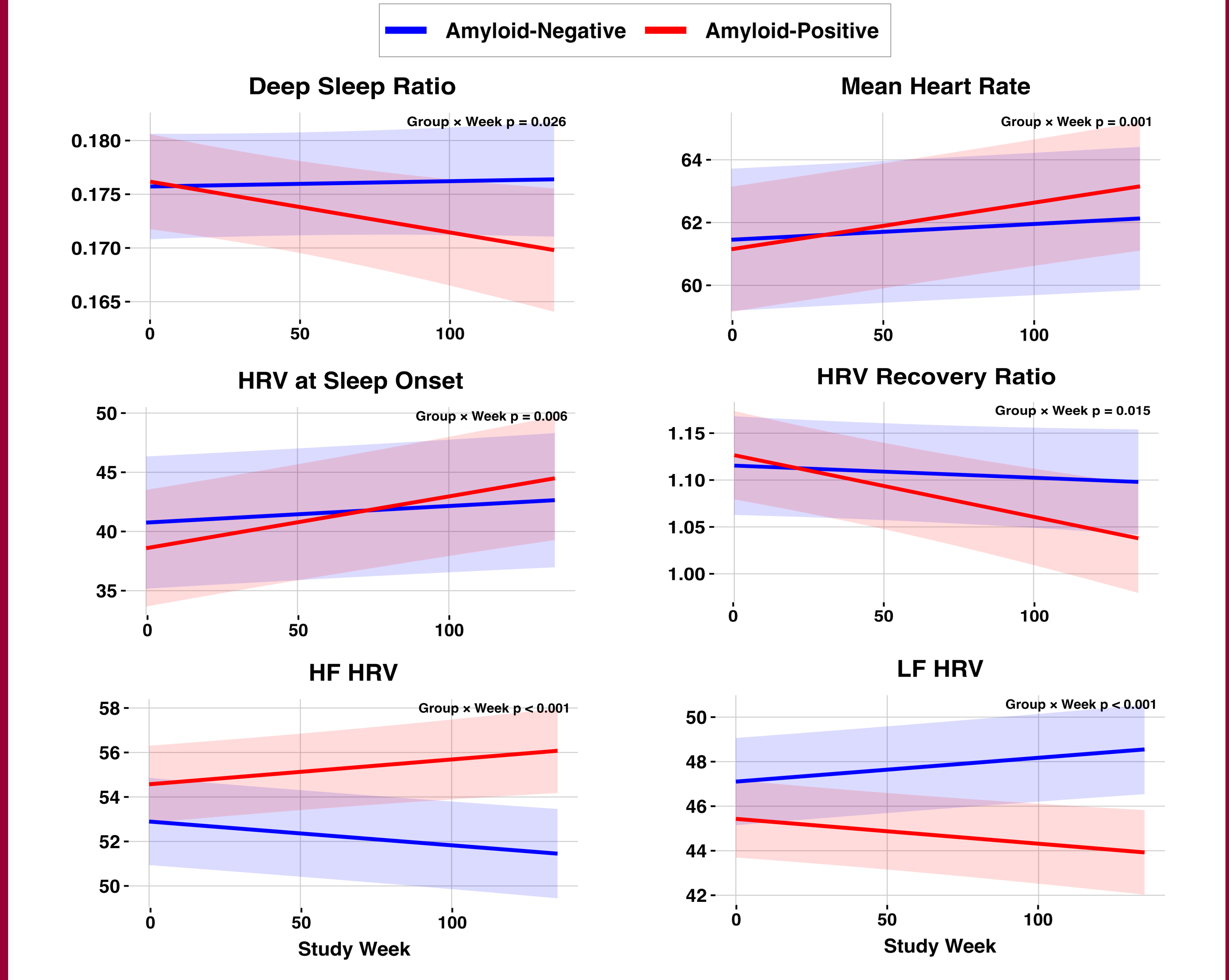
Figure 1. Electronic bed mat for sleep monitoring

Results

Participants were enrolled for an average of 89.2 weeks (range: 28-138 study weeks). Across the 89 participants, 4,828 total weeks of monitoring data were collected, corresponding to an average of 54.3 weeks of data per participant.

Table 1	Amyloid-Negative (N = 47)	Amyloid-Positive (N = 42)	p-value
Age at visit, years	77.62 (6.51)	75.67 (6.56)	0.19
Sex (male)	14 (30%)	13 (31%)	0.91
Education, years	17.06 (2.16)	17.48 (1.86)	0.56
CDR global score (0.5)	8 (17%)	16 (38%)	0.03
APOE ε4 carrier	6 (13%)	18 (43%)	<.01
Sleep Architecture			
Deep sleep ratio (deep sleep/total sleep duration)	0.18 (0.02)	0.18 (0.03)	0.83
Light sleep ratio (light sleep/total sleep duration)	0.58 (0.02)	0.58 (0.02)	0.35
REM sleep ratio (REM sleep/total sleep duration)	0.24 (0.03)	0.25 (0.03)	0.78
Heart Rate			
Mean heart rate, beats/min	62.33 (6.19)	61.51 (5.75)	0.61
Max heart rate, beats/min	99.43 (13.13)	95.87 (15.06)	0.23
Min heart rate, beats/min	46.49 (4.24)	46.41 (4.22)	0.99
Heart Rate Variability (HRV) Recovery			
HRV at sleep onset, ms	33.98 (20.49)	36.75 (23.20)	0.69
HRV before awakening, ms	31.83 (10.65)	36.67 (21.96)	0.63
Total overnight HRV recovery, ms	-2.16 (14.30)	-0.08 (6.91)	0.71
Integrated overnight HRV recovery, ms·h	235.06 (130.00)	223.64 (110.11)	0.75
HRV recovery rate, ms/h	-0.27 (2.16)	-0.12 (1.12)	0.48
HRV recovery ratio	1.07 (0.20)	1.10 (0.21)	0.82
HRV Activity			
Low frequency HRV, %	47.71 (6.50)	45.42 (6.44)	0.03
High frequency HRV, %	52.29 (6.50)	54.59 (6.45)	0.03
Morning RMSSD, ms	30.75 (9.67)	36.54 (23.01)	0.40
Evening RMSSD, ms	34.92 (25.49)	35.93 (22.37)	0.80

Figure 2. Slope difference by amyloid pathology groups



Amyloid pathology is associated with measurable alterations in sleep architecture and nocturnal cardiac physiology, detectable using continuous, unobtrusive in-home monitoring.